

Research Article

A comparative study of two different doses of dexmedetomidine for attenuating the haemodynamic response to tracheal intubation

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Abstract: **Introduction:** Secure airway for proper ventilation during anesthesia is one important component of a successful surgery. Endotracheal intubation is one of the most important methods in this context. Intubation method and used medication are considerably important in attenuating complications. This research aimed to investigate the impact of two different doses of dexmedetomidine in mitigating cardiovascular responses to endotracheal intubation in candidate cases sup **Materials and Methods** One hundred elective surgery cases were consecutively enrolled in this prospective study after obtaining informed consent. The patients were randomly allocated into two groups, each comprising 40 cases. Group A received intravenous dexmedetomidine at 0.5 mcg/kg, while Group B received 1 mcg/kg, both diluted with 20 ml of normal saline over a 10-minute infusion using a pump. Hemodynamic responses from both the groups were then recorded. porting voluntary operation **Results** The extubation quality became better, sedation and incidence of bradycardia in the post-operative period increased with a higher dose of dexmedetomidine. Attenuation of hemodynamic parameters was observed after 4 min of starting infusion and during extubation in each group and was found to be significant ($P < 0.001$). **Conclusion** A dose of 0.5µg/kg of dexmedetomidine administered as a bolus infusion before extubation attenuates the stress response to extubation as effectively as 1µg/kg. Higher sedation scores and longer time to extubate are seen with a dose of 1µg/kg without causing respiratory depression.

Keywords: Laryngoscopy, Endotracheal intubation, Hemodynamic response, Dexmedetomidine.

INTRODUCTION

Endotracheal intubation, a common procedure in surgical settings, is accompanied by a surge in sympathetic activity, giving rise to marked hemodynamic perturbations. These responses encompass elevations in blood pressure, heart rate, and myocardial oxygen demand, which can be of particular concern in patients with pre-existing cardiovascular conditions. Effective attenuation of these adverse cardiovascular effects during laryngoscopy and intubation is essential for preventing perioperative complications and ensuring optimal patient outcomes. [1].

Dexmedetomidine, an α_2 -adrenergic agonist with a high selectivity profile, has garnered attention for its multifaceted pharmacological properties, including sedation, anxiolysis, analgesia, and sympatholysis [2]. Operating through the modulation of norepinephrine release, dexmedetomidine acts centrally within the locus ceruleus, exerting inhibitory control over sympathetic outflow. This unique mechanism offers a potential solution to dampen the undesirable hemodynamic changes triggered by airway manipulation.[3] Within the realm of anesthesia, dexmedetomidine's role as an adjuvant has expanded due to its ability to mitigate the hemodynamic response associated with laryngoscopy and intubation.[4] By targeting the autonomic nervous system and blunting stress-induced sympathetic activation, dexmedetomidine emerges as a promising

tool for enhancing perioperative cardiovascular stability. However, a critical aspect that remains under scrutiny is the determination of the optimal dexmedetomidine dosage. This issue assumes greater significance given the variability in drug responses among different populations. In the context of the Indian population, such variability may arise due to genetic, metabolic, or environmental factors. Therefore, elucidating the appropriate dexmedetomidine dose assumes heightened importance, particularly when considering the pharmacological management of perioperative hemodynamics in the Indian subset. The dearth of consensus on the ideal dexmedetomidine dosage underscores the need for rigorous investigation to establish evidence-based guidelines. In essence, the rationale for this study is grounded in the quest for precision medicine within anesthesia practice. Tailoring dexmedetomidine dosing to the Indian population subset can potentially confer benefits in terms of enhanced hemodynamic stability during the critical peri-intubation period. Given the growing recognition of dexmedetomidine as a versatile adjuvant, elucidating the most effective dose in this specific context holds considerable clinical relevance. Thus the present research investigation aims to assess the impact of varying doses (0.5 mcg/kg vs 1 mcg/kg) of dexmedetomidine on hemodynamic responses during laryngoscopy and endotracheal intubation while evaluating potential adverse effects.

MATERIALS AND METHODS

This was a randomized double blind study conducted at Nepal Medical College and Teaching Hospital from June to August 2019. Ethical approval from the Institutional Review Committee of Nepal Medical College and Teaching Hospital was obtained. Thorough pre-operative evaluation of the patients was done a day before surgery. A total of 60 patients, aged 18-55 years of either sex, ASA PS I and

II posted for elective surgery under general anesthesia were included in this study. Patients with hypertension and cardiac disease, patients with difficult airway (Mallampatti Grade III and IV), obese patients (BMI >25), patients with endocrinal diseases like hyperthyroidism and hypothyroidism, patients allergic to the study drug, patients with baseline heart rate < 60 beats/minute and patients on beta blockers were excluded from the study. Those patients who had intubation attempt lasting longer than 15 sec, and multiple intubation attempts (2 or more) were also excluded from the study. An informed written consent was obtained from all the patients who meet the inclusion criteria. Premedication was done with tab. Lorazepam 2 mg for patients weighing 50 kg or more and tab. Lorazepam 1 mg for patients weighing less than 50 kg on the night before surgery. The patients were kept nil per oral for at least 8 hours for solid food and sips of clear liquid were allowed till 2 hours prior to surgery. The patients were allocated into two groups: Group A and Group B, 30 patients in each group, by slips of paper in a box technique. One of the anesthesiologist prepared the intravenous infusion and coded them. The coded infusion was given to the resident anesthetist, who was unaware of its content, to be administered to the patients. The same resident was given the responsibility of monitoring the patient intraoperatively and recording all the hemodynamic parameters of the patients. All the intubations were done by the co-author of the study. In the operating room ECG, pulse oximeter and non-invasive blood pressure (NIBP) cuff were attached. Baseline cardiovascular parameters i.e. heart rate, blood pressure (systolic, diastolic and mean) and oxygen

saturation were recorded. Intravenous (IV) access secured with appropriate sized cannula. Patients belonging to the Group A (n=30) received dexmedetomidine 0.5 µg/kg diluted with 0.9% normal saline to make a total 20 ml volume, slowly IV over 10 minutes via syringe pump. Patients belonging to the Group B (n=30) received dexmedetomidine 1 µg/kg diluted with 0.9% normal saline to make 20 ml volume, slowly IV over 10 minutes via syringe pump. Vitals heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and oxygen saturation (SpO₂) were monitored during infusion of the drug. General anesthesia technique was standardized for both the groups. Inj. Fentanyl 2 µg/kg was given, induction was done with Propofol 1% injection in incremental dose until loss of eyelash reflex was attained. Isoflurane at 0.5% was turned on. After confirmation of bag and mask ventilation, vecuronium 0.1 mg/kg was given. One minute after vecuronium injection, isoflurane was increased to 2% to deepen the anesthesia. Three minutes after vecuronium injection, direct laryngoscopy and intubation was done. Heart rate, systolic, diastolic and mean arterial pressure was recorded before giving the test drug, after completion of the administration of the test drug at 5 and 10 minutes, after induction, after intubation at 1 minute, 2 minutes and 5 minutes. Maintenance of anesthesia was done with isoflurane, oxygen, vecuronium with IPPV and fentanyl as needed. At the end of the surgery, residual effect of neuromuscular blockade was reversed by Neostigmine 2.5 mg and glycopyrrolate 0.4 mg. Patients were then extubated and transferred to the post-operative ward. The duration of surgery and the duration of anaesthesia were also recorded. Clinically relevant hypotension was defined as a decrease in systolic arterial blood pressure by 20% or more from baseline value. It was treated with 200 ml Ringer's lactate solution. If ineffective, 5 mg mephentermine was given. Clinically relevant bradycardia was defined as heart rate < 50 Thapa et al beats/min and was treated with atropine 0.6 mg intravenously. Data were entered in Microsoft Excel and analyzed with the Statistical Package for the Social Science (SPSS). A p value < 0.05 was considered to indicate statistical significance in all tests.

RESULTS

In our study both the groups were comparable with regard to demographic parameters like age and sex.

Table 1: Distribution of patients according to Age in percentage

Age	Group A		Group B		p Value
	N	%	N	%	
<20 years	4	10.0	2	2.0	0.687 NS
21-30 years	9	22.5	8	20.0	
31-40 years	9	22.5	4	10.0	
41-50 years	7	17.5	11	27.5	
51-60 years	5	12.5	7	17.5	
61-65 years	6	15.0	3	7.5	
Sex					0.4364 NS
Male	23	60	20	50	

Female	17	40	20	50	
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There was decrease in the mean heart rate (HR) after administration of the study drug, throughout the study period in both the groups.

Table 2: Comparison of Mean Heart Rate changes between Group A and Group B

	Group A	Group B	p value
	Mean± SD	Mean± SD	
Pre-op(baseline)	85.93±4.14	84.30±4.36	0.142 NS
(Study Drug Started) 0 min	82.34±4.30	82.80±3.68	0.145 NS
2 min	81.77±3.21	79.97±4.07	0.063 NS
4 min	78.70±3.84	74.57±4.14	0.044 S
6 min	77.13±6.35	75.9±9.5	0.001 HS
8 min	76.60±3.83	72.11±5.05	0.001 HS
10min	75.33±3.38	69.34±1.99	0.001 HS
At Induction	69.08±2.38	68.10±2.14	0.083 NS
At Intubation	75.70±3.83	75.57 ±4.13	0.073 NS
Post Intubation (1min)	72.80±2.05	71.27±2.86	0.026 S
5min	65.80± 4.13	64.94± 4.09	0.000 HS
10 min	67.37± 3.08	63.09± 2.99	0.001 HS

S – Significant, HS – Highly Significant, NS – Non-Significant

As compared to the baseline mean systolic blood pressure (SBP), mean diastolic blood pressure (DBP), mean blood pressure (MBP) was decreased in both the groups which continued throughout the study period.

Table 3: Comparison of mean Systolic Blood Pressure Changes between

SBP (mm of Hg)	Group A	Group B	p value
	Mean± SD	Mean± SD	
Pre-op (baseline)	122.70±6.64	122.84±4.76	0.217 NS
(Study Drug Started) 0 min	118.63±3.36	118.14±3.68	0.106 NS
2 min	118.88±4.06	116.14±4.50	0.000 HS
4 min	117.25±7.49	114.68±9.37	0.000HS
6 min	119.14±9.69	108.1±10.9	0.000 HS
8 min	110.34±7.96	108.82±6.99	0.000 HS
10 min	109.98±7.65	105.71±4.09	0.000 HS
At Induction	112.82±6.87	108.61±7.89	0.000 HS
At Intubation	117.25±7.49	111.68±6.33	0.000 HS
Post Intubation (1min)	115.77 ±6.45	107.5±11.9	0.774 NS
5min	108.03±5.40	100.83±6.24	0.236 NS
10 min	98.24±2.74	96.43±3.49	0.000 HS

Group A and Group B

Table 4: Comparison of Mean Diastolic Blood Pressure Changes Group A and Group B

DBP (mm of Hg)	Group A	Group B	p value
	Mean± SD	Mean± SD	
Pre-op (baseline)	75.61± 5.19	74.73±3.28	0.208 NS
(Study Drug Started) 0 min	74.21±6.81	73.11±5.42	0.573 NS
2 min	74.08±5.79	72.09±4.79	0.001 HS
4 min	71.73±4.73	64.48±7.17	0.002 HS
6 min	70.11±6.98	68.7±11.8	0.001 HS
8 min	69.14±5.16	65.62±5.58	0.000 HS
10 min	66.10±3.82	65.64±6.49	0.000 HS
At Induction	64.03±3.49	61.08±4.63	0.000 HS
At Intubation	71.73 ±4.72	68.47±4.34	0.000 HS
Post Intubation 1min	72.11±3.88	68.28±5.79	0.000 HS
5min	67.32±3.09	63.14±3.58	0.000 HS
10 min	62.96±2.75	60.62±2.29	0.001 HS

S – Significant, HS – Highly Significant, NS – Non-Significant.

Group B showed more fall in the HR from 4th minute of drug administration up to 10th min. At intubation, it showed slight rise in both the groups compared to pre induction value though actual value being less compared to the baseline (Group A: 78.70 ± 3.84 , - 9.79% vs. Group B: 74.57 ± 4.14 percentage value -10.51%) (P value >0.05). The fall was also seen post intubation from 1 min up to 10 min, the difference in HR being statistically significant at 5 and 10 min. Maximum fall in the HR was observed in both the groups post intubation at 10 minutes. Fall in heart rate was more in Group B than Group A. At induction, at intubation and post intubation at 1 min, the fall in all types of blood pressure was noted in both the groups. This fall in blood pressure is less as compared to baseline not requiring any treatment. Here also Group B showing more fall than Group A. Post intubation fall in SBP at 1 min, and 5 min was statistically not significant. There was decrease in SBP in both the groups post intubation at 10 min which was statistically highly significant in Group B than in Group A. There was decrease in DBP in both the groups post intubation at 5 min and 10 min which was statistically highly significant. At intubation and post intubation at 1 min, the fall in MBP was less as compared to the baseline in both the groups. Group B showing more fall than Group A. There was decrease in MBP in both the groups post intubation at 10 min which was statistically significant. Fall in mean blood pressure was more in Group B than in Group A. In Group A, one patient and in Group B, 2 patients had bradycardia (HR < 50 bpm) none of the patients in both the groups had hypotension (BP > 30%) from the baseline.

DISCUSSION

Dexmedetomidine, an alpha-2 agonists drug has been tried for reducing the intubation response and found to have better effects compared to other drugs with reduced or no side effects like respiratory depression. When used intravenously it inhibits the release of nor-epinephrine by presynaptic activation of the α -2 adrenoceptor in the Locus coeruleus. Post synaptic activation of α -2 adrenoceptor in the central nervous system by this drug results in decrease in sympathetic activity. Its use decreases the serum catecholamine levels by 90% and suppresses the hemodynamic response of laryngoscopy. Dexmedetomidine decreases heart rate by augmenting the vagal nerve and blocking the cardio-accelerator nerves. This negative chronotropic effect is also attributed to reflex response for transient hypertension during initial part of infusion and subsequently it was due to diminished norepinephrine release and inhibition of central sympathetic outflow [7, 8]. In our study after administration of the study drug, we observed the similar effect i.e. decrease in the mean heart rate compared to the baseline in both the groups which continued throughout the study period. At intubation, mean heart rate showed slight rise in both the groups compared to pre induction value, though actual value was less than the baseline and the values were comparable in both the groups. Post intubation at 5 min and 10 min there was statistical significant difference noted in two group with group B showing more fall. Hasan A *et al.* 2016 [9] compared the dose of $0.6 \mu\text{g/kg}$ versus $1 \mu\text{g/kg}$ and observed continuous decrease in the mean heart rate throughout the study period in both the groups. Raval D L *et al.* 2014 (8,29), They found in their study that the fall in mean HR in Group A ($1.0 \mu\text{g/kg}$ dexmedetomidine) was more as compared to Group B ($0.5 \mu\text{g/kg}$ dexmedetomidine), during laryngoscopy and intubation, 1 min after intubation, 2 min after intubation and 5 min after intubation which was statistically highly significant (p<0.05). Their findings were almost similar with our study.

We noted bradycardia (HR < 50/min) in Group B ($0.5 \mu\text{g/kg}$ dexmedetomidine), and 2 cases of bradycardia (HR < 50/min) in Group C ($1 \mu\text{g/kg}$ dexmedetomidine),

respectively. In our study, after administration of the study drug, there was decrease in SBP, DBP, MBP compared to the baseline in both the groups which continued throughout the study period. Reduction of tonic levels of sympathetic outflow and an augmentation of cardiac-vagal activity due to Dexmedetomidine can result in decrease in cardiac output which in turn causes decrease in blood pressure. At induction, at intubation and post intubation at 1 min, the fall in SBP, DBP, and MAP was less as compared to the baseline in both the groups. Group B showing more fall than Group A. Post intubation fall in SBP at 1 min, and 5 min was comparable in both the groups. There was decrease in mean SBP, DBP, MBP in both the groups post intubation at 10 min which was statistically highly significant in Group B than in Group A. Smitha *et al.* 2014 [11] compared the effect of $0.5 \mu\text{g/kg}$, $1 \mu\text{g/kg}$ of dexmedetomidine and normal saline. They had similar findings for SBP like our study. In a similar study done by A Esra Sagi roglu *et al.* [12] they noted statistically highly significant difference between $1 \mu\text{g/kg}$ (Group A) and (Group B) $0.5 \mu\text{g/kg}$. As compared to baseline SBP had increased in both the groups 60 sec after induction which is not matching with our study. It might be because of the difference in the induction agents and inhalational agents as we used Inj. Propofol instead of Inj. Thiopentone and Isoflurane as an inhalational agent. Rashmi *et al.* 2013 [13] compared the two different doses of Dexmedetomidine, $0.6 \mu\text{g/kg}$ and $1 \mu\text{g/kg}$ and control group of NS. As seen in our study they also found fall in DBP after the start of Dexmedetomidine. At 1 min post intubation they have noted the rise in DBP above the baseline in both Dexmedetomidine groups. We noted rise in diastolic pressure but it was not more than that of Baseline, Smitha *et al.* [11] noted that values of diastolic blood pressure were statistically lower at all intervals in $1 \mu\text{g/kg}$ than $0.5 \mu\text{g/kg}$ of dexmedetomidine including 1 min post intubation value. This change in this study may be attributed to use of different opioids (Fentanyl $1-2 \text{mcg/kg}$ for induction) Modh B. Dixitkumar *et al.* [14] observed fall in MBP in Group D1 (1mcg/kg Dexmedetomidine) till induction (baseline vs. induction) which was less at 1 min (value) and 2 min (value) post

intubation. This was like our Group B study. Rawal. D et al. [8].

noticed fall in MBP in both the groups at all time intervals which was more in 1mcg/kg Dexmedetomidine compared to the baseline. At 1 min after intubation, less fall was noticed as compared to the baseline. This finding is unlike our study Jarineshin H [15] noticed fall in MBP from baseline after study drug infusion. At intubation and at 3 min post intubation there was less fall compared to the baseline. Their study findings were correlating our study. H S Nanda et al. 2016 [10] noticed fall in the MBP in Group B: 0.5 µ g/kg and Group C: 1 µ g/kg from the baseline after completion of the study drug. In Group B, there was slight rise in the MBP immediately after intubation, at 1min and it was above the baseline. At 3min the value matches the baseline. At 5 min and 10 min again fall was seen. In Group C, There was fall in mean blood pressure throughout the study period, Less fall was seen immediately after intubation and at 1 min post intubation and it was statistically highly significant as compared to Group B. These findings were found to be similar with our study. None of the patients in our study had hypotension. Our findings match with the study by Allam Hasan et al. and Nanda et al.

CONCLUSION

According to our study, inj. dexmedetomidine 0.75µg/kg provides statistically significant attenuation of hemodynamic response to laryngoscopy and endotracheal intubation as compared to inj. dexmedetomidine 0.5µg/kg without having significant adverse effects, with better hemodynamic stability along with dose sparing effect of propofol for induction.

Thus, from the present study, we conclude inj. dexmedetomidine 0.75µg/kg as more effective in attenuating the response to laryngoscopy and endotracheal intubation.

REFERENCES

1. Shribman AJ, Smith G, Achola KJ. Cardiovascular And Catecholamine Responses To Laryngoscopy With And Without Tracheal Intubation. *Br J Anaesth*. 1987;59(3):295- 299.
2. . Weerink MAS, Struys MMRF, Hannivoort LN, Barends CRM, Absalom AR, Colin P. Clinical Pharmacokinetics And Pharmacodynamics Of Dexmedetomidine. *Clin Pharmacokinet*. 2017;56(8):893-913.
3. Kaye AD, Chernobylsky DJ, Thakur P, Et Al. Dexmedetomidine In Enhanced Recovery After Surgery (ERAS) Protocols For Postoperative Pain. *Curr Pain Headache Rep*. 2020;24(5):21.
4. . Jain K, Sethi SK, K.N. H, Patodi V, Jain N, Meena D. Efficacy Of Dexmedetomidine In Attenuating Pressor Response To Laryngoscopy And Endotracheal Intubation Under Bispectral Index Controlled Anesthesia: A Prospective Randomized Double-Blinded Study. *AinShams J Anesthesiol*. 2023;15(1):15.
5. Rajasekhar M, Yadav M, Kulkarni D, Gopinath R. Comparison Of Hemodynamic Responses To Laryngoscopy And Intubation Using Macintosh Or Mccoy Or C-MAC Laryngoscope During Uniform Depth Of Anesthesia Monitored By Entropy. *J Anaesthesiol Clin Pharmacol*. 2020;36(3):391.
6. Suryawanshi CM, Kumar KS, Tudimilla S. Comparative Study Of Hemodynamic Response And Glottic View To Laryngoscopy And Endotracheal Intubation With Macintosh, Mccoy Blades And X-Mac Video Laryngoscopy In Patients Undergoing General Anaesthesia. *Int J Med Rev Case Rep*. 2022;6(10):27-27.
7. Dept. Of Anaesthesiology, PRM Medical College, Baripada, Mayurbhanja, Swain DAK. Comparison Of The Efficacy Of Clonidine And Dexmedetomidine Infusions Administered Preoperatively For Attenuation Of The Hemodynamic Response Following Laryngoscopy And Endotracheal Intubation In A Placebo- Controlled Study. *J Med Sci Clin Res*. 2017;5(7).
8. Kovac AL. Controlling The Hemodynamic Response To Laryngoscopy And Endotracheal Intubation. *J Clin Anesth*. 1996;8(1):63-79.
9. .Gupta DM, Ganerwal DV, Bondarde DK. Comparative Study Of Intravenous Injection Dexmedetomidine And Intravenous Injection Clonidine For Attenuation Of Hemodynamic Response During Laryngoscopy And Intubation. *Int J Med Anesthesiol*. 2020;3(3):18-23.
10. Kakkar A, Tyagi A, Nabi N, Sethi AK, Verma UC. Comparison Of Clonidine And Dexmedetomidine For Attenuation Of Laryngoscopy And Intubation Response – A Randomized Controlled Trial. *J Clin Anesth*. 2016;33:283- 288.
11. Yazbek-Karam VG, Aouad MM. Perioperative Uses Of Dexmedetomidine. *Middle East Anaesthesiol*. 2006;18(6):1043-1058.
12. Kato J, Ogawa Y, Kojima W, Aoki K, Ogawa S, Iwasaki K. Cardiovascular Reflex Responses To Temporal Reduction In Arterial Pressure During Dexmedetomidine Infusion: A Double-Blind, Randomized, And Placebo-Controlled Study. *Br J Anaesth*. 2009;103(4):561-565.
13. Mukhtar AM, Obayah EM, Hassona AM. The Use Of Dexmedetomidine In Pediatric Cardiac Surgery. *Anesth Analg*. 2006;103(1):52.
14. Gong M, Man Y, Fu Q. Incidence Of Bradycardia In Pediatric Patients Receiving Dexmedetomidine Anesthesia: A Meta-Analysis. *Int J Clin Pharm*. 2017;39(1):139-147.

15. Shin HW, Yoo HN, Kim DH, Lee H, Shin HJ, Lee HW. Preanesthetic Dexmedetomidine 1 Mg/Kg Single Infusion Is A Simple, Easy, And Economic Adjuvant For General Anesthesia. *Korean J Anesthesiol*. 2013;65(2):114-120.
16. De Cassai A, Boscolo A, Geraldini F, Et Al. Effect Of Dexmedetomidine On Hemodynamic Responses To Tracheal Intubation: A Meta-Analysis With MetaRegression And Trial Sequential Analysis. *J Clin Anesth*. 2021;72:110287.
17. . Kunisawa T, Nagata O, Nagashima M, Et Al. Dexmedetomidine Suppresses The Decrease In Blood Pressure During Anesthetic Induction And Blunts The Cardiovascular Response To Tracheal Intubation. *J Clin Anesth*. 2009;21(3):194- 199.
18. . Kumar NRR, Jonnavithula N, Padhy S, Sanapala V, Naik VV. Evaluation Of Nebulised Dexmedetomidine In Blunting Haemodynamic Response To Intubation: A Prospective Randomised Study. *Indian J Anaesth*. 2020;64(10):874.
19. . Misra S, Behera BK, Mitra JK, Sahoo AK, Jena SS, Srinivasan A. Effect Of Preoperative Dexmedetomidine Nebulization On The Hemodynamic Response To Laryngoscopy And Intubation: A Randomized Control Trial. *Korean J Anesthesiol*. 2020;74(2):150-157.
20. Bhargavi Chappa M, Vishnu Vardhan A, G.V.Sasi Kiran T. Effect Of Nebulised Dexmedetomidine In Blunting Haemodynamic Response To Intubation- A Randomized Double Blind Controlled Study. *Indian J Appl Res*. Published Online March 1, 2023:73-74.
21. . Sriramka B, Warsi ZH, Sahoo J. Effects Of Adding Dexmedetomidine To Nebulized Lidocaine On Control Of Hemodynamic Responses To Laryngoscopy And Intubation: A Randomized Clinical Trial. *J Anaesthesiol Clin Pharmacol*. 2023;39(1):11.
22. Mahajan L, Kaur M, Gupta R, Aujla KS, Singh A, Kaur A. Attenuation Of The Pressor Responses To Laryngoscopy And Endotracheal Intubation With Intravenous Dexmedetomidine Versus Magnesium Sulphate Under Bispectral Index-Controlled Anaesthesia: A PlaceboControlled Prospective Randomised Trial. *Indian J Anaesth*. 2018;62(5):337.
23. Rathore A, Chaudhary S, Kumar M, Salhotra R. Nalbuphine Versus Dexmedetomidine For Attenuation Of Haemodynamic Response To Laryngoscopy And Intubation: A Randomised Double Blind Comparative Study. *Indian J Clin Anaesth*. 2021;8(4):579-585.
24. Bhaskar S, Khan T. Dexmedetomidine(0.5 Mgkg)Is Better Than Clonidine (1 Mgkg) In Attenuating Stress Response To Laryngoscopy And Intubation When Used As Premedication In Adult Surgical Patients. *Glob J Res Anal*. Published Online April 14, 2018. Accessed July 6, 2024.
25. J V, K P, K P, Shashikumar S. Attenuation Of Hemodynamic Response To Laryngoscopy And Tracheal Intubation In Adult Patients With A Single Intravenous Bolus Dose Of Dexmedetomidine - A Prospective, Randomized, Double-Blind, Controlled Clinical Study. *Natl J Physiol Pharm Pharmacol*. 2020;(0):1.
26. Hou H, Song Y, Gu H, Zhang J. Comparison Of The Hemodynamic Effects Of A Loading Dose Of Dexmedetomidine Combined With Total Intravenous Anesthesia Or Inhalation Anesthesia On Children With Obstructive Sleep Apnea-Hypopnea Syndrome. *Int J Anesthesiol Resusc*. Published Online 2019. Accessed July 6, 2024.
27. . Lee CW, Kim M. Effects Of Preanesthetic Dexmedetomidine On Hemodynamic Responses To Endotracheal Intubation In Elderly Patients Undergoing Treatment For Hypertension: A Randomized, DoubleBlinded Trial. *Korean J Anesthesiol*. 2016;70(1):39-45.
28. . Sebastian B, Talikoti AT, Krishnamurthy D. Attenuation Of Haemodynamic Responses To Laryngoscopy And Endotracheal Intubation With Intravenous Dexmedetomidine: A Comparison Between Two Doses. *Indian J Anaesth*. 2017;61(1):4829.
29. Kaur M, Goel S. Effect Of Low Dose Intravenous Dexmedetomidine With 4% Sevoflurane On Haemodynamic Response During Laryngoscopy And Tracheal Intubation: A Randomised Controlled Study. *J Clin Diagn Res*. Published Online 2022.
30. Sharma N, Mehta N. Therapeutic Efficacy Of Two Different Doses Of Dexmedetomidine On The Hemodynamic Response To Intubation, The Intubating Conditions, And The Effect On The Induction Dose Of Propofol: A Randomized, Double- Blind, PlaceboControlled Study. *Anesth Essays Res*. 2018;12(2):566.
31. . Mk DA. Attenuation Of Haemodynamic Responses By Dexmedetomidine During Laryngoscopy And Intubation: A Double Blinded Randomized Controlled Study. *Int J Med Anesthesiol*. 2020;3(4):01-06.
32. . Silpa AR, Koshy KA, Subramanian A, Pradeep KK. Comparison Of The Efficacy Of Two Doses Of Dexmedetomidine In Attenuating The Hemodynamic Response To Intubation In Patients Undergoing Elective Cardiac Surgery: A Randomized Double-Blinded Study. *J Anaesthesiol Clin Pharmacol*. 2020;36(1):83.