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Research Article

Comparative Evaluation of Two Doses of Dexmedetomidine for Attenuation of Hemodynamic Responses to Laryngoscopy and Endotracheal Intubation: A Randomized Controlled Study

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation produce a transient sympathetic response characterized by increases in heart rate and blood pressure. Dexmedetomidine, a selective α2-adrenergic agonist, can attenuate this response, although the optimal dose remains uncertain. This study compared the efficacy of two doses of dexmedetomidine in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation.

Methods: This comparative study included 60 patients who were allocated into two equal groups of 30 patients each. Group L received dexmedetomidine 0.5 µg/kg, while Group H received dexmedetomidine 1 µg/kg. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were assessed at baseline and at 1, 3, 5, and 10 minutes following the intervention. Between-group comparisons were performed using independent-samples *t* test, while within-group changes were assessed using paired analysis with Bonferroni correction.

Results: The demographic characteristics were comparable between the groups. HR was significantly lower in the high-dose group at baseline and at 1, 3, 5, and 10 minutes ($p<0.001$). SBP, DBP, and MAP were also significantly lower in the high-dose group at 5 and 10 minutes ($p<0.05$), whereas differences at 1 and 3 minutes were not statistically significant. Within-group analysis showed no significant changes in SBP, DBP, or MAP from baseline in the low-dose group. In contrast, the high-dose group demonstrated significant reductions in SBP, DBP, and MAP at 1, 3, 5, and 10 minutes after administration.

Conclusion: Dexmedetomidine 1 µg/kg provided greater attenuation of peri-intubation hemodynamic responses than 0.5µg/kg. The higher dose produced a more sustained reduction in HR, SBP, DBP, and MAP, particularly from 5 minutes onward. Thus, 1 µg/kg may be more effective when stronger suppression of the cardiovascular response to laryngoscopy and intubation is desired.

Keywords: Dexmedetomidine, Laryngoscopy, Endotracheal Intubation, Hemodynamic Response, Heart Rate, Blood Pressure.

INTRODUCTION

Laryngoscopy and endotracheal intubation are essential components of general anaesthesia but constitute potent noxious stimuli capable of producing transient sympathetic activation. This response commonly manifests as increases in heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP). Although these changes are generally short-lived in healthy individuals, exaggerated cardiovascular responses may be clinically undesirable, particularly in patients with hypertension, coronary artery disease, or limited cardiovascular reserve [1, 2].

Several pharmacological agents have been investigated to attenuate the hemodynamic response to laryngoscopy and intubation. Dexmedetomidine, a highly selective α2-adrenergic receptor agonist, reduces central sympathetic outflow and produces sedation and analgesia with minimal respiratory depression. These properties make it useful as an adjunct during induction of general anaesthesia and airway manipulation [3].

Recent randomized studies have demonstrated that dexmedetomidine can effectively attenuate

the increases in HR and arterial pressure associated with laryngoscopy and endotracheal intubation. Intravenous dexmedetomidine at 0.5 µg/kg has been shown to provide hemodynamic control comparable with fentanyl, while a dose of 1 µg/kg has demonstrated significant attenuation of HR, SBP and DBP responses [4,5]. Studies using nebulized dexmedetomidine have also reported reduced cardiovascular responses following intubation, suggesting that its sympatholytic effect can be achieved through alternative routes [6,7].

The dose of dexmedetomidine is clinically important because increasing the dose may enhance sympatholysis but can also increase the risk of bradycardia and hypotension. A large meta-analysis involving 99 randomized studies and 6833 patients demonstrated significant reductions in HR, SBP and MAP during laryngoscopy with dexmedetomidine, but also identified clinically relevant risks of bradycardia and hypotension [8]. Recent clinical studies have further confirmed its efficacy in attenuating peri-intubation hemodynamic responses [9,10].

However, evidence regarding the optimal intravenous dose remains inconsistent. While higher doses may provide greater attenuation of sympathetic responses, lower doses may achieve adequate cardiovascular control with fewer adverse effects. Therefore, the present study was undertaken to compare intravenous dexmedetomidine 0.5 µg/kg and 1 µg/kg for attenuation of changes in HR, SBP, DBP and MAP during laryngoscopy and endotracheal intubation.

Aim

To compare the effects of 0.5 µg/kg and 1 µg/kg intravenous dexmedetomidine in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation.

Objectives

To compare the effects of low-dose and high-dose dexmedetomidine on HR, SBP, DBP and MAP at baseline and at 1, 3, 5 and 10 minutes following endotracheal intubation.

MATERIALS AND METHODS

Study design and setting

This was a randomized, double-blind controlled study conducted among patients admitted for elective surgical procedures under general anaesthesia at Tertiary care Teaching Hospital, Tamilnadu.

A total of 60 patients were included and randomly allocated into two equal groups.

- **Group L:** Dexmedetomidine 0.5 µg/kg, n=30
- **Group H:** Dexmedetomidine 1 µg/kg, n=30

Study population

Patients aged 15–58 years scheduled for elective surgery requiring general anaesthesia and endotracheal intubation were recruited.

Data Collection

After obtaining institutional ethical approval and written informed consent, eligible patients scheduled for elective surgery under general anaesthesia were recruited. Demographic and baseline clinical data including age, sex, height, weight and ASA physical status were recorded for each participant. Patients were randomly allocated into two equal groups of 30 patients each. Group L received dexmedetomidine 0.5 µg/kg, whereas Group H received dexmedetomidine 1 µg/kg. The calculated dose was diluted in 100 mL of normal saline and administered intravenously over 10 minutes before induction of general anaesthesia.

Baseline haemodynamic parameters were recorded immediately before administration of the study drug. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) were documented using standard intraoperative monitoring. Following completion of the study-drug infusion, general anaesthesia was induced according to the standardized institutional anaesthetic protocol. Direct laryngoscopy followed by endotracheal intubation was performed by the anaesthesiologist. The duration of laryngoscopy and number of intubation attempts were recorded where applicable.

HR, SBP, DBP and MAP were subsequently recorded at 1, 3, 5 and 10 minutes after successful endotracheal intubation. The same monitoring technique and measurement procedure were maintained throughout the observation period to minimize measurement variability. The primary outcome was the difference in haemodynamic response between the low-dose and high-dose dexmedetomidine groups. Secondary outcomes included within-group changes in HR, SBP, DBP and MAP from baseline to 1, 3, 5 and 10 minutes following intubation.

All observations were entered into a structured data collection proforma and subsequently

transferred to a computerized database for statistical analysis. Particular attention was given to maintaining identical observation time points in both groups. The collected data were analysed using SPSS version 25.0.

Ethical considerations

The study was conducted after obtaining institutional ethical approval and written informed consent from all participants. The study followed the ethical principles of the Declaration of Helsinki.

Statistical analysis

Data were analysed using IBM SPSS Statistics version 19. Continuous variables were expressed as mean ± standard deviation. Categorical variables were expressed as frequencies and percentages. Baseline demographic comparisons were performed using the independent-samples *t* test for continuous variables and the chi-square test The effects of dose and time on HR, SBP, DBP and MAP were assessed using multifactorial ANOVA A *p* value <0.05 was considered statistically significant.

RESULTS

Demographic characteristics

A total of 60 patients were analysed, with 30 patients in each group. The mean age was 36.77 ± 13.98 years in Group L and 37.57 ± 10.44 years in Group H. Mean height and weight were also comparable between groups. The sex distribution did not differ significantly between groups [Table 1].

Table 1. Demographic characteristics of the study population

Variable	Low dose (n=30)	High dose (n=30)	Test statistic	p value
Age, years, mean ± SD	36.77 ± 13.98	37.57 ± 10.44	0.25[a]	0.80
Height, cm, mean ± SD	164.30 ± 7.41	166.73 ± 8.44	1.19[a]	0.24
Weight, kg, mean ± SD	60.13 ± 7.56	61.33 ± 6.29	0.67[a]	0.51
Male:Female	14:16	21:9	3.36[b]	0.067

p<0.05 considered statistically significant using [a]Independent-samples *t* test. [b]Chi-square test.

Table 2. Between-group comparison of hemodynamic parameters at different time points

Parameter	Time	Low-dose (0.5 µg/kg), Mean ± SD	High-dose (1 µg/kg), Mean ± SD	*p* value	Significance
MAP (mmHg)	Baseline	90.49 ± 6.41	93.91 ± 3.98	0.016	Significant
	1 min	90.04 ± 6.02	90.20 ± 3.80	0.910	NS
	3 min	90.44 ± 6.22	88.04 ± 2.94	0.063	NS
	5 min	91.36 ± 6.70	86.89 ± 3.72	**0.003**	**Significant**
	10 min	91.56 ± 7.46	86.63 ± 3.73	**0.002**	**Significant**
HR (beats/min)	Baseline	79.93 ± 9.90	64.93 ± 5.27	**<0.001**	**Significant**
	1 min	79.93 ± 10.01	64.40 ± 1.16	**<0.001**	**Significant**
	3 min	80.67 ± 9.19	64.47 ± 6.36	**<0.001**	**Significant**
	5 min	80.53 ± 9.04	64.80 ± 6.18	**<0.001**	**Significant**
	10 min	80.67 ± 9.33	65.93 ± 6.42	**<0.001**	**Significant**
SBP (mmHg)	Baseline	120.00 ± 8.30	124.13 ± 4.70	0.022	Significant
	1 min	120.00 ± 7.74	119.67 ± 5.44	0.850	NS
	3 min	120.80 ± 7.80	117.60 ± 5.10	0.066	NS
	5 min	121.53 ± 8.23	116.13 ± 5.14	**0.004**	**Significant**
	10 min	121.60 ± 8.72	115.50 ± 5.15	**0.002**	**Significant**
DBP (mmHg)	Baseline	75.73 ± 6.03	78.80 ± 4.63	0.031	Significant
	1 min	75.07 ± 6.10	75.47 ± 3.96	0.760	NS
	3 min	75.27 ± 6.02	73.27 ± 3.04	0.110	NS
	5 min	76.27 ± 6.84	72.27 ± 3.74	**0.007**	**Significant**

	10 min	76.53 ± 7.43	72.20 ± 3.61	**0.006**	**Significant**

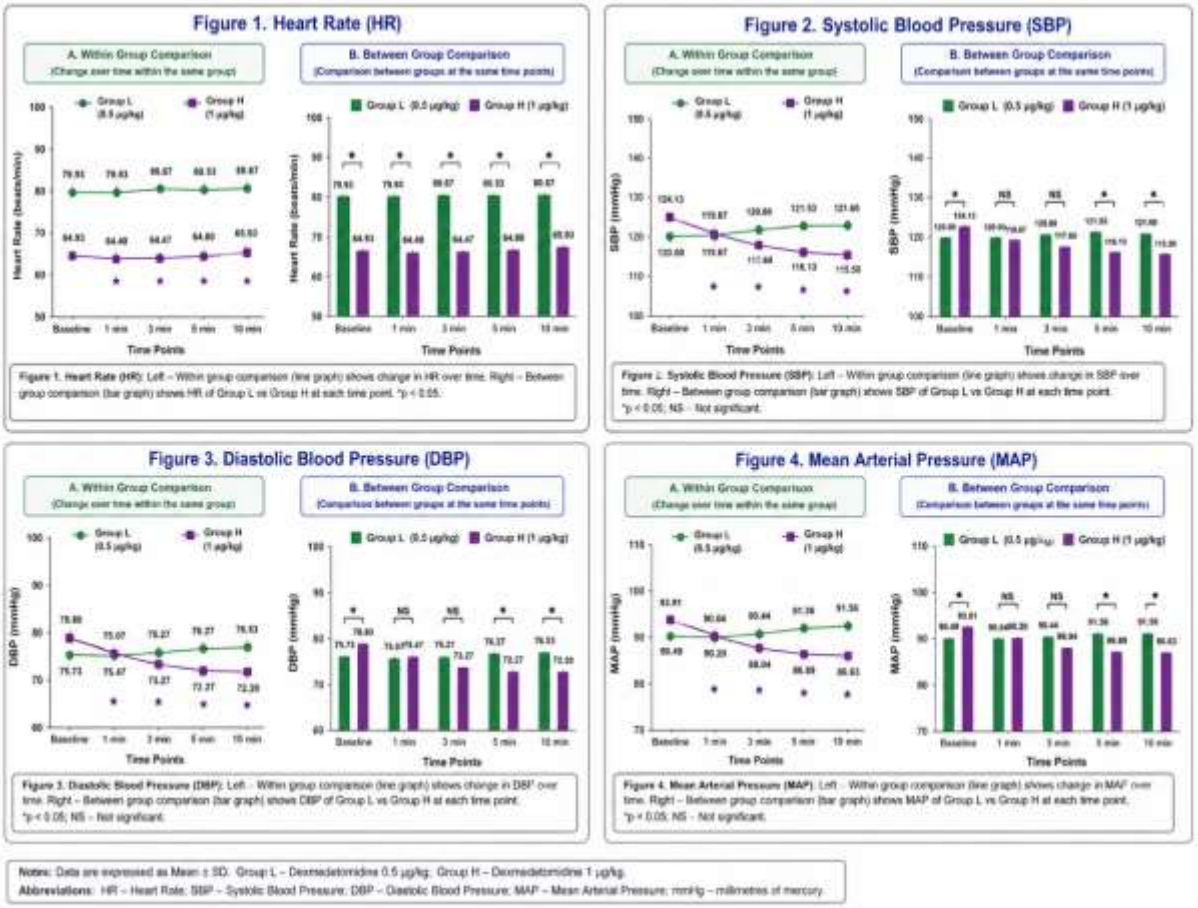
Note: Values are expressed as mean ± SD. Between-group comparisons were performed using an independent-samples *t*-test. NS = not significant. $p < 0.05$ was considered statistically significant.

The high-dose dexmedetomidine group (1 µg/kg) had significantly lower HR at all-time points compared with the low-dose group ($p < 0.001$). SBP, DBP and MAP were also significantly lower with the high dose at 5 and 10 minutes, while differences at 1 and 3 minutes were not significant. Overall, 1 µg/kg provided greater attenuation of the hemodynamic response, particularly after 5 minutes. (Table 2) [Fig 1-4]

Table 3. Within-group comparison of hemodynamic parameters following dexmedetomidine administration

Parameter	Time	Low-dose (0.5 µg/kg) Mean ± SD	Change from baseline	High-dose (1 µg/kg) Mean ± SD	Change from baseline
SBP (mmHg)	Baseline	120.00 ± 8.30	—	124.13 ± 4.70	—
	1 min	120.00 ± 7.74	0.00 (NS)	119.67 ± 5.44	**−4.46 (S)**
	3 min	120.80 ± 7.80	+0.80 (NS)	117.60 ± 5.10	**−6.53 (S)**
	5 min	121.53 ± 8.23	+1.53 (NS)	116.13 ± 5.14	**−8.00 (S)**
	10 min	121.60 ± 8.72	+1.60 (NS)	115.50 ± 5.15	**−8.63 (S)**
DBP (mmHg)	Baseline	75.73 ± 6.03	—	78.80 ± 4.63	—
	1 min	75.07 ± 6.10	−0.66 (NS)	75.47 ± 3.96	**−3.33 (S)**
	3 min	75.27 ± 6.02	−0.46 (NS)	73.27 ± 3.04	**−5.53 (S)**
	5 min	76.27 ± 6.84	+0.54 (NS)	72.27 ± 3.74	**−6.53 (S)**
	10 min	76.53 ± 7.43	+0.80 (NS)	72.20 ± 3.61	**−6.60 (S)**
MAP (mmHg)	Baseline	90.49 ± 6.41	—	93.91 ± 3.98	—
	1 min	90.04 ± 6.02	−0.45 (NS)	90.20 ± 3.80	**−3.71 (S)**
	3 min	90.44 ± 6.22	−0.05 (NS)	88.04 ± 2.94	**−5.87 (S)**
	5 min	91.36 ± 6.70	+0.87 (NS)	86.89 ± 3.72	**−7.02 (S)**
	10 min	91.56 ± 7.46	+1.07 (NS)	86.63 ± 3.73	**−7.28 (S)**

S = Significant; NS = Not significant. Change from baseline = follow-up mean − baseline mean. Significance based on paired analysis with Bonferroni correction ($\boldsymbol{p} < 0.012$). Within the **low-dose group**, SBP, DBP and MAP remained relatively stable throughout the observation period, with no significant change from baseline. In contrast, the high-dose group showed significant reductions in SBP, DBP and MAP at 1, 3, 5 and 10 minutes compared with baseline. The magnitude of reduction became progressively greater over time, with the maximum reduction observed at 10 minutes. (Table 3); [Fig 1-4]



Notes: Data are expressed as Mean ± SD. Group L – Dexmedetomidine 0.5 µg/kg; Group H – Dexmedetomidine 1 µg/kg.
Abbreviations: HR – Heart Rate; SBP – Systolic Blood Pressure; DBP – Diastolic Blood Pressure; MAP – Mean Arterial Pressure; mmHg – millimetres of mercury.

DISCUSSION

Laryngoscopy and endotracheal intubation produce a transient sympathetic response characterized by increases in heart rate and arterial blood pressure. Although these changes are generally short-lived, they may be clinically important in patients with cardiovascular or cerebrovascular disease. Dexmedetomidine, a selective α2-adrenergic agonist, decreases central sympathetic outflow and has therefore been extensively investigated for attenuation of the hemodynamic response associated with airway manipulation. [11,12]

In the present study, 60 patients were equally allocated to receive dexmedetomidine 0.5 µg/kg or 1 µg/kg. The demographic characteristics, including age, height, weight and sex distribution, were comparable between the two groups, indicating satisfactory baseline comparability. This supports the interpretation that the subsequent differences in hemodynamic variables were predominantly related to the dexmedetomidine dose. [13]

The most prominent finding was the significantly lower heart rate in the high-dose group. HR was significantly lower with dexmedetomidine 1 µg/kg at baseline and at 1, 3, 5 and 10 minutes compared with the 0.5 µg/kg group (p<0.001). The mean HR remained approximately 65 beats/min in the high-dose group compared with approximately 80 beats/min in the low-dose group. This reduction is consistent with the sympatholytic action of dexmedetomidine and its ability to reduce catecholamine-mediated cardiovascular responses. [14,15]

The dose-dependent cardiovascular effect observed in our study is supported by previous comparative investigations. Studies evaluating different doses of dexmedetomidine have demonstrated more effective attenuation of tachycardia and blood pressure responses with higher doses. The pharmacological effects of dexmedetomidine are related to its central α2-adrenergic activity, which decreases sympathetic nervous system activity and modifies cardiovascular responses. [16,17]

In the present study, SBP, DBP and MAP were significantly lower in the high-dose group at 5 and 10 minutes, whereas differences at 1 and 3 minutes were not statistically significant. This suggests that the cardiovascular effect of the higher dose became more evident with increasing time following administration.

Previous studies have similarly demonstrated attenuation of the pressor response following dexmedetomidine administration. [18,19]

The within-group analysis provided additional evidence for the greater hemodynamic effect of the 1 µg/kg dose. In the low-dose group, SBP, DBP and MAP remained relatively stable and did not demonstrate significant changes from baseline. In contrast, the high-dose group showed significant reductions in all three parameters at 1, 3, 5 and 10 minutes. The magnitude of reduction increased progressively, with the greatest reduction occurring at 10 minutes. These findings suggest a more sustained sympatholytic effect with the higher dose. [20,21]

The present findings are also consistent with evidence from systematic reviews and meta-analyses. Dexmedetomidine has been shown to reduce the cardiovascular response to tracheal intubation, although the magnitude of attenuation may vary according to dose, route of administration and timing of administration. [22,23]

Comparative evidence further indicates that dexmedetomidine can provide effective control of post-intubation hemodynamic changes when compared with other pharmacological agents used to attenuate the sympathetic response. These findings support its role as an effective sympatholytic agent during airway manipulation. [24]

Nevertheless, the greater hemodynamic suppression produced by the 1 µg/kg dose should be considered alongside the potential for bradycardia and hypotension. The same α2-adrenergic mechanism responsible for suppression of sympathetic activity can produce excessive cardiovascular depression in susceptible patients. Therefore, although the higher dose demonstrated superior efficacy in the present study, careful patient selection and continuous hemodynamic monitoring remain important. [1]

Overall, the present study demonstrates that dexmedetomidine 1 µg/kg provides greater attenuation of heart rate and blood pressure responses than 0.5 µg/kg**.** The difference was particularly evident for SBP, DBP and MAP at 5 and 10 minutes, while HR remained significantly lower throughout the observation period. These findings support the use of 1 µg/kg when stronger suppression of the hemodynamic response to laryngoscopy and endotracheal intubation is clinically desirable, provided appropriate cardiovascular monitoring is maintained.

CONCLUSION

In this study, dexmedetomidine 1 µg/kg was more effective than 0.5 µg/kg in attenuating hemodynamic changes associated with airway manipulation. The high-dose group demonstrated significantly lower heart rate at all assessed time points and significantly lower SBP, DBP, and MAP at 5 and 10 minutes compared with the low-dose group. Within-group analysis showed progressive and significant reductions in SBP, DBP, and MAP following administration of 1 µg/kg, whereas the 0.5 µg/kg group maintained relatively stable values. Thus, dexmedetomidine 1 µg/kg provides superior hemodynamic attenuation compared with 0.5 µg/kg and may be preferred when effective suppression of the cardiovascular response to laryngoscopy and endotracheal intubation is required. However, because higher doses may increase the risk of bradycardia and hypotension, dose selection should be individualized according to the patient's cardiovascular status and clinical requirements.

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REFERENCES

1. Sahay N, Khurana P, Sethi S. Attenuation of pressor response to laryngoscopy and intubation with intravenous dexmedetomidine: a randomized controlled study. J Anaesthesiol Clin Pharmacol. 2018;34(3):328-33.
2. Ismail EA, Mostafa AA, Abdelatif MM. Attenuation of hemodynamic response to laryngoscopy and endotracheal intubation with single dose dexmedetomidine in controlled hypertensive patients: prospective randomized double-blind study. Ain-Shams J Anesthesiol. 2022;14:57.
3. 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):11-17. doi:10.4103/joacp.JOACP_93_21.

4. Mahiswar AP, Dubey PK, Ranjan A. Comparison between dexmedetomidine and fentanyl bolus in attenuating the stress response to laryngoscopy and tracheal intubation: a randomized double-blind trial. Braz J Anesthesiol. 2022;72(1):103-9. doi:10.1016/j.bjane.2021.02.060. PMID:34000325.
5. 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 placebo-controlled prospective randomised trial. Indian J Anaesth. 2018;62(5):337-43. doi:10.4103/ija.IJA_1_18. PMID:29910490.
6. 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. 2021;74(2):150-7. doi:10.4097/kja.20153. PMID:32434291.
7. Shrivastava P, Kumar M, Verma S, Sharma R, Kumar R, Ranjan R, Prakash J. Evaluation of nebulised dexmedetomidine given pre-operatively to attenuate hemodynamic response to laryngoscopy and endotracheal intubation: a randomised control trial. Cureus. 2022;14(5):e25223. doi:10.7759/cureus.25223.
8. De Cassai A, Boscolo A, Geraldini F, Zarantonello F, Pettenuzzo T, Pasin L, et al. Effect of dexmedetomidine on hemodynamic responses to tracheal intubation: a meta-analysis with meta-regression and trial sequential analysis. J Clin Anesth. 2021;72:110287. doi:10.1016/j.jclinane.2021.110287. PMID:33873003.
9. Rani A, Ahlawat G, Kumar A, Mangal K, Bala R. Nebulised dexmedetomidine versus nebulised lignocaine in blunting the haemodynamic response to laryngoscopy and endotracheal intubation: a randomised control study. J Clin Diagn Res. 2024;18(6):UC05-09. doi:10.7860/JCDR/2024/70032.19510.
10. Singh S, Gupta P, Dhar M, et al. Effect of preoperative intravenous and inhalational dexmedetomidine on the hemodynamic response of laryngoscopy: a prospective double-blinded randomized study. J Res Pharm Pract. 2024;12(4):123-9. doi:10.4103/jrpp.jrpp_12_24.
11. 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):48-54. doi:10.4103/0019-5049.198404. PMID:28216704.
12. Gulabani M, Gurha P, Dass P, Kulshreshtha N. Comparative analysis of efficacy of lignocaine 1.5 mg/kg and two different doses of dexmedetomidine (0.5 μg/kg and 1 μg/kg) in attenuating the hemodynamic pressure response to laryngoscopy and intubation. J Anaesthesiol Clin Pharmacol. 2015;31(1):5-14. doi:10.4103/0259-1162.150167. PMID:25886414.
13. Li A, Yuen VMY, Goulay-Dufay S, Kwok PCL. Pharmacokinetics and pharmacodynamics of dexmedetomidine. Drug Dev Ind Pharm. 2016;42(12):1917-27. doi:10.1080/03639045.2016.1232727. PMID:27595299.
14. 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. doi:10.1007/s40262-017-0507-7. PMID:28105598.
15. Lee S. Dexmedetomidine: present and future directions. Korean J Anesthesiol. 2019;72(4):323-30. doi:10.4097/kja.19259. PMID:31220910.
16. Zhao L, Xu L, Zheng J, Wang Q. Comparison of intravenous dexmedetomidine versus esmolol for attenuation of hemodynamic response to tracheal intubation after rapid sequence induction: a systematic review and meta-analysis. Biomed Res Int. 2019;2019:6791971. doi:10.1155/2019/6791971. PMID:31139645.
17. Gupta M, Rohilla R, Gupta P, Tamilchelvan H, Joshi U, Kanwat J. Nebulized dexmedetomidine for attenuating hemodynamic response to laryngoscopy and endotracheal intubation in adult patients undergoing

surgeries under general anaesthesia: a systematic review and meta-analysis of randomized controlled trials. BMC Anesthesiol. 2023;23(1):406. doi:10.1186/s12871-023-02366-9. PMID:38082217.

18. 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;33:283-8. doi:10.1016/j.jclinane.2016.04.026.
19. Kunisawa T, Nagata O, Nagashima M, Mitamura S, Ueno M, Suzuki A, 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):194-9. doi:10.1016/j.jclinane.2008.08.015. PMID:19464613.
20. Saleh A, Kan S, Singhal S, Ouanounou A, Wong M. Effects of dexmedetomidine vs esmolol on postintubation hemodynamics: a meta-analysis. Anesth Prog. 2025;72(1):9-23. doi:10.2344/23-0059. PMID:40657827.
21. Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: a review of clinical applications. Curr Opin Anaesthesiol. 2008;21(4):457-61. doi:10.1097/ACO.0b013e328305e3ef. PMID:18660652.
22. Khan ZP, Ferguson CN, Jones RM. Alpha-2 and imidazoline receptor agonists: their pharmacology and therapeutic role. Anaesthesia. 1999;54(2):146-65. doi:10.1046/j.1365-2044.1999.00659.x. PMID:10215710.
23. Li Y, He R, Ying X, Xie G, Xu J, Liu X, et al. Effects of adding dexmedetomidine to nebulized lidocaine on control of hemodynamic responses to laryngoscopy and intubation: a randomized clinical trial. BMC Anesthesiol. 2023;23:168.
24. Sethi N, Pawar R, Garg R, Mishra S, Mehta V, Sharma S. Comparison of clonidine and dexmedetomidine for attenuation of laryngoscopy and intubation response: a randomized controlled trial. J Anaesthesiol Clin Pharmacol. 2016;32(2):212-7.