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Original Research Article

DEXMEDETOMIDINE DOSING FOR ATTENUATING HEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND INTUBATION: A COMPARATIVE STUDY OF 0.5 MCG/KG VS. 1 MCG/KG

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ABSTRACT

Background: Dexmedetomidine, renowned for its sedative, anxiolytic, and opioid-sparing properties, exerts its effects primarily within the locus ceruleus of the brainstem. By diminishing sympathetic outflow, it effectively attenuates stress responses. Despite its growing popularity, optimal dosing remains uncertain, particularly within the Indian population. This study aimed to compare the hemodynamic response attenuation achieved by two dexmedetomidine doses: 0.5 mcg/kg and 1 mcg/kg during laryngoscopy and intubation.

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 50 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.

Results: Baseline diastolic blood pressure was comparable between groups, and at 10 minutes post-drug administration, as well as during intubation and 1 minute post-intubation ($p>0.05$). However, post-intubation diastolic blood pressure was significantly lower in Group B compared to Group A at 3 minutes post-intubation and remained lower through subsequent readings until 10 minutes post-intubation. Similarly, mean arterial pressure was significantly lower in Group B compared to Group A for all readings up to 10 minutes post-intubation.

Conclusion: In the context of attenuating the hemodynamic response to laryngoscopy and intubation, this study establishes the superiority of intravenous dexmedetomidine at 1 mcg/kg over 0.5 mcg/kg dosing. The higher dose may be particularly beneficial for patients with a history of myocardial ischemia, hypertension, or cerebrovascular accidents, for whom excessive stress response during laryngoscopy and intubation is undesirable. Caution is advised when administering the 1 mcg/kg dose due to potential bradycardia during drug infusion.

Keywords: Anesthesia, Dexmedetomidine, Hemodynamic, Intubation, Laryngoscopy, Sympathetic outflow.

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 preexisting

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 α2-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.

MATERIAL AND METHODS

Study design

A prospective, randomized, and comparative study was conducted at a tertiary care center within the Department of Anaesthesiology.

Study population

The study included 100 adult patients aged 18 to 65 years, classified as ASA status I and II, scheduled for elective surgery under general anesthesia. Patients with Mallampatti airway grade 1 and 2 were considered eligible. The patients were divided in two groups as, 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.

Subject selection criteria

Patients considered for participation in the study must meet specific inclusion criteria, including being adult individuals aged 18 to 65 years, having ASA status I & II, and being scheduled for elective surgery under general anesthesia. Additionally, patients with Mallampatti airway grade 1 and 2 will be eligible. On the other hand, patients will be excluded if they refuse the procedure, have certain medical comorbidities such as ischemic heart diseases, arrhythmias, acute and chronic renal failure, severely deranged liver function, endocrine disorders, or are on long-term drug therapy with antipsychotics, antidepressants, or anxiolytics. Exclusions also apply to patients with basal heart rates less than 55 per minute, those expected to have a difficult airway, those with allergies to study drugs, pregnant or lactating women, hemodynamically compromised patients, and patients weighing more than 80 kg.

Study Protocol

A preoperative visit was conducted one day prior to surgery, during which detailed patient histories were obtained. Patients were monitored with electrocardiography (ECG), oxygen saturation (SpO2), and non-invasive blood pressure (NIBP). Intravenous access was established using a 20G cannula, and an infusion of Ringer's lactate solution was initiated. Baseline measurements of heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and SpO2 were recorded following pre-induction, and these served as baseline values for the study.

Anesthesia Administration

Standard general anesthesia was administered using the following sequential steps: Preoxygenation was initiated with 100% O2 for a duration of 3 minutes. Subsequently, intravenous administration of Inj. Glycopyrrolate was performed at a dosage of 4 mcg/kg, followed by the intravenous administration of Inj. Midazolam at a dosage of 1 mg/kg. The study drug was then administered. Parameters including HR, SBP, DBP, MAP and (SpO2 were recorded 10 minutes after the infusion of the study drug.

Intravenous administration of Inj. Fentanyl was carried out at a dose of 1 mcg/kg, followed by induction using Inj. Thiopentone at a dosage of 4-5 mg/kg intravenously, continued until the obliteration of the eyelash reflex. Neuromuscular blockade was achieved with the administration of Inj. Atracurium at a dosage of 0.75 mg/kg. Anesthesia was maintained for a 3-minute period using a mixture of 50% O2 and 50% air, complemented by isoflurane in the range of 0.2 to 0.8%. Following a 3-minute interval from the administration of atracurium, laryngoscopy was performed using a standard Macintosh curved laryngoscope blade, followed by the successful execution of endotracheal intubation in a single attempt.

Management of adverse effects

The adverse effects associated with the administration of anesthesia, including bradycardia, tachycardia, hypotension, hypertension, and desaturation, were vigilantly monitored and addressed using the following specific interventions: In cases of bradycardia, defined as a HR below 55 beats per minute, an intravenous injection of Atropine was promptly administered to mitigate the condition. Similarly, instances of tachycardia, where the heart rate exceeded 30% above baseline, were managed through the controlled intravenous administration of Propofol in titrated doses.

For cases of hypertension, characterized by a SBP surpassing 140 mmHg, a stepwise approach was taken. Intravenous injection of Propofol in titrated doses was utilized, concomitantly with the escalation of isoflurane concentration up to a maximum of 1.2%, to effectively lower blood pressure. Conversely, in instances of hypotension, where the MAP fell below 60 mmHg, a combination of interventions was employed. Rapid intravenous fluid administration was initiated, in tandem with the reduction of isoflurane concentration. Additionally, intravenous administration of Ephedrine was carried out in titrated doses to counteract the drop in blood pressure.

Data Collection

Physiological parameters (HR, SBP, DBP, MAP, SpO2) were recorded at intubation, laryngoscopy, and 1-, 3-, 5-, and 10-minutes post-intubation. Intraoperative anesthesia included isoflurane (0.5 - 1%), 50% nitrous oxide, 50% O2, and intermittent intravenous Atracurium with closed circuit mechanical ventilation. Post-surgery, patients were reversed using glycopyrrolate (8 mcg/kg) and neostigmine (0.05 mg/kg), followed by extubation after oral suctioning. Vital parameters were monitored in the anesthesia recovery room.

Statistical Analysis

Quantitative data were depicted as mean ± standard deviation (SD), while categorical data were presented in percentage format. Quantitative data underwent analysis using the t-test, while categorical data were subjected to scrutiny via the chi-square test. The significance threshold for the p value was established at <0.05. These analyses were executed utilizing SPSS software version 21, ensuring rigorous statistical evaluation of the obtained data.

RESULTS

Comparative analysis of demographic and clinical characteristics between Group A and Group B revealed no significant differences in age ($p = 0.71$) or weight ($p = 0.26$). Gender distribution was comparable, with 44% females in Group A and 46% in Group B, as well as 56% males in Group A and 54% in Group B ($p = 1.00$). The distribution of patients by ASA Grade 1 and Grade 2 displayed similar percentages in both groups (Grade 1: 70% vs. 76%, Grade 2: 30% vs. 24%), without significant variation ($p = 0.65$). These findings demonstrate the well-matched nature of the study groups in terms of demographic and clinical characteristics, ensuring a valid basis for subsequent outcome comparisons. [Table 1]

The study examined changes in hemodynamic parameters over different time points among two study groups (Group A and Group B). HR, SBP, DBP were monitored and compared between the groups (Table 2). Notably, at 10 minutes after drug administration (T1) and during various post-intubation intervals, Group B exhibited statistically significant lower HR, SBP, and DBP compared to Group A. These findings suggest that the intervention influenced hemodynamic stability, with Group B consistently demonstrating more favourable outcomes. [Table 2]

Table 3 displays the alterations in MAP and Spo2 across distinct time intervals for both Group A and Group B. The findings demonstrate a noteworthy reduction in MAP at multiple time points for both groups, indicating changes in blood pressure levels. In contrast, Spo2 remained relatively consistent across the observed periods, suggesting a consistent oxygen saturation trend. These findings underscore the dynamic nature of MAP and the stability of Spo2 during the study. [Table 3]

Table 4 summarizes the comparison of adverse events in Group A and Group B. Hypertension was observed in 2% of Group A and 0% of Group B, with no significant difference ($p=1.0$). No cases of hypotension were reported. Bradycardia occurred in 2% of Group B and was absent in Group A ($p=1.0$). Tachycardia was not observed in either group. Overall, the incidence of adverse events was low, with hypertension and bradycardia showing no significant differences between the groups. [Table 4]

Table 1: Comparative analysis of study groups: demographics and clinical characteristics

Variables	Group A Mean ± SD	Group B Mean ± SD	P value (Two-sample t-test)
Age (years)	29.4 ± 4.8	30.9 ± 5.6	0.71
Weight (kg)	61.3 ± 8.9	61.4 ± 11.1	0.26
Gender (Female)	22 (44%)	23 (46%)	1.00
Gender (Male)	28 (56%)	27 (54%)	
ASA Grade 1	35 (70%)	38 (76%)	0.65
ASA Grade 2	15 (30%)	12 (24%)	

Table 2: Changes in Hemodynamic Parameters Over Time Among Study Groups

Time Points	HR (beats/min)		P value	Systolic BP (mmHg)		P value	Diastolic BP (mmHg)		P value
	Group A	Group B		Group A	Group B		Group A	Group B	
Baseline (T0)	77.6 ± 9.6	75.9 ± 17.0	0.52	117.1 ± 5.9	119.7 ± 13.4	0.17	73.1 ± 4.7	74.4 ± 9.9	0.14
10 min after drug (T1)	71.5 ± 8.7 (-7.8%)	60.9 ± 14.9 (-19.7%)	<0.05	107.8 ± 7.5 (-8.4%)	109.7 ± 14.3 (-8.4%)	0.91	64.0 ± 3.3 (-12.4%)	65.6 ± 7.8 (-11.8%)	0.98
At time of intubation (T2)	85.8 ± 11.2 (10.6%)	79.2 ± 12.1 (4.3%)	0.19	123.4 ± 8.8 (4.8%)	114.5 ± 11.1 (-4.3%)	<0.01	79.3 ± 5.6 (8.5%)	73.2 ± 8.1 (-1.6%)	0.07
1 min post intubation (T3)	82.3 ± 6.2 (6.0%)	75.4 ± 13.6 (-0.7%)	0.1	117.2 ± 7.1 (-0.5%)	110.7 ± 8.3 (-7.5%)	<0.01	74.0 ± 5.6 (1.2%)	70.2 ± 6.0 (-5.6%)	0.12
3 min post intubation (T4)	78.7 ± 6.2 (1.4%)	72.4 ± 11.4 (-4.6%)	0.12	112.0 ± 8.5 (-4.8%)	106.5 ± 8.2 (-11.0%)	<0.05	69.4 ± 5.3 (-5.1%)	64.9 ± 11.0 (-12.8%)	<0.01
5 min post intubation (T5)	74.2 ± 6.1 (-4.4%)	69.9 ± 12.6 (-7.9%)	0.49	113.0 ± 7.8 (-3.9%)	103.6 ± 6.8 (-13.5%)	<0.01	67.8 ± 5.0 (-7.2%)	61.8 ± 9.6 (-16.9%)	<0.01
10 min post intubation (T6)	74.8 ± 8.3 (-3.6%)	67.7 ± 11.4 (-10.8%)	0.07	110.3 ± 7.7 (-6.3%)	99.2 ± 8.9 (-17.1%)	<0.01	65.4 ± 7.3 (-10.6%)	58.4 ± 10.5 (-21.5%)	<0.01

HR = Heart Rate, Systolic BP = Systolic Blood Pressure, Diastolic BP = Diastolic Blood Pressure, Values in parentheses represent percentage change

Table 3: Changes in MAP and Spo2 Over Time Among Study Groups

Time Points	MAP		P value	Spo2		P value
	Group A	Group B		Group A	Group B	
Baseline (T0)	88.6 ± 5.0	89.3 ± 10.2	0.33	99.00 ± 0.83	98.87 ± 0.82	0.747
10 min after drug (T1)	78.7 ± 3.8 (-11.2%)	80.1 ± 6.6 (-10.3%)	0.88	98.93 ± 0.83 (-0.1%)	98.80 ± 0.81 (-0.1%)	0.8
At time of intubation (T2)	91.3 ± 4.9 (3.0%)	85.7 ± 5.3 (-4.0%)	<0.05	98.87 ± 0.91 (-0.1%)	99.01 ± 0.87 (0.1%)	0.88
1 min post intubation (T3)	88.3 ± 4.6 (-0.4%)	83.6 ± 5.5 (-6.4%)	<0.05	98.80 ± 0.71 (-0.2%)	98.80 ± 0.81 (-0.1%)	0.92
3 min post intubation (T4)	83.5 ± 5.6 (-5.7%)	78.5 ± 7.4 (-12.1%)	<0.01	98.80 ± 0.71 (-0.2%)	98.83 ± 0.79 (0.0%)	0.979
5 min post intubation (T5)	82.9 ± 4.8 (-6.5%)	75.6 ± 7.8 (-15.3%)	<0.01	98.70 ± 0.70 (-0.3%)	98.77 ± 0.77 (-0.1%)	0.667
10 min post intubation (T6)	80.4 ± 6.9 (-9.3%)	71.9 ± 9.8 (-19.5%)	<0.01	98.70 ± 0.70 (-0.3%)	98.77 ± 0.73 (-0.1%)	0.656

Table 4: Comparison of Adverse Events in the Study Groups

Adverse Events	Group A	%	Group B	%	Total	p-value
Hypertension	1	2%	0	0%	1	1.0
Hypotension	0	0%	0	0%	0	NA
Bradycardia	0	0%	1	2%	1	1.0
Tachycardia	0	0%	0	0%	0	NA

DISCUSSION

The introduction of general anesthesia revolutionized medical procedures by enabling controlled unconsciousness, rendering patients insensitive to pain and unaware of surgical events. However, patients under anesthesia often require artificial airway maintenance due to the inability to sustain their own airway. This is where techniques like laryngoscopy and endotracheal intubation play a crucial role. While intubation offers benefits like airway security and prevention of aspiration, it comes with its own set of complications. Laryngoscopy and endotracheal intubation can trigger various stress responses, such as tachycardia, hypertension, laryngospasm, bronchospasm, increased intracranial pressure, and elevated

intraocular pressure.[1] These physiological reactions highlight the intricate balance between the advantages of intubation and the potential adverse effects it may induce.

The initial insight into the hemodynamic shifts induced by laryngoscopy and intubation was provided by Reid and Brace (Reid and Brace,1940). Remarkably, this physiological response commences within mere seconds of direct laryngoscopy and becomes even more pronounced upon endotracheal tube insertion. The cascade is set in motion within just 5 seconds of laryngoscopy, reaching its zenith between 1 to 2 minutes, and subsequently subsiding to baseline levels by the 5-minute mark.[5,6] These transient alterations are generally well-tolerated by individuals without underlying health issues. However, for patients grappling with cardiovascular ailments, these shifts can potentially trigger detrimental consequences like myocardial ischemia, ventricular dysrhythmias, ventricular failure, and pulmonary edema. Additionally, individuals with cerebrovascular conditions are at risk of cerebrovascular accidents.[7,8] The dynamic interplay between these hemodynamic changes and various health conditions underscores the critical importance of this investigation.

Various treatment approaches have been explored to address these challenges, including intravenous lignocaine, deeper levels of anesthesia through inhaled or intravenous agents, narcotics, calcium channel blockers like nifedipine, adrenergic blockers (beta blockers), and alpha2 agonists such as clonidine and dexmedetomidine. Despite their potential, each of these strategies carries its own set of limitations.

The α-2 adrenoreceptors play a pivotal role in regulating both the autonomic and cardiovascular systems. These receptors are situated on blood vessels, where they orchestrate vasoconstriction, and on sympathetic terminals, where they dampen the release of norepinephrine. Furthermore, they are distributed within the central nervous system (CNS), where their activation induces sedation, curbs sympathetic outflow, and enhances cardiac-vagal activity. This orchestration can lead to reductions in heart rate and cardiac output. Notably, the use of α-2 agonists in the perioperative setting has been linked to decreased anesthetic requirements and blunted heart rate and blood pressure reactions during stress-inducing events.

The multifaceted effects of dexmedetomidine, encompassing analgesia, sedation, anxiolysis, sympatholysis, and mitigation of exaggerated hemodynamic responses, are currently under intensive investigation. These effects predominantly stem from the activation of alpha-2 receptors nestled within post-synaptic terminals in the CNS. This activation culminates in diminished neuronal activity and a reinforcement of vagal activity. Clonidine, another α-2 agonist, has also been harnessed by various researchers to mitigate the hemodynamic surge triggered by laryngoscopy and intubation.[9, 10]

Dexmedetomidine emerges as a notably potent α-2 receptor agonist, exhibiting approximately eightfold greater potency than clonidine. Despite its robust effects, the action of dexmedetomidine is relatively brief, characterized by an elimination half-time of approximately 2 hours. An intriguing facet of dexmedetomidine's pharmacology is the availability of a reversal agent, atipamezole, designed to counteract its sedative impact. Atipamezole achieves its effects by augmenting the central turnover of noradrenaline. These distinct attributes collectively position dexmedetomidine as a superior option when compared to clonidine.[11]

While the effects of dexmedetomidine have been investigated by various researchers at doses of 0.5 and 1 µg/kg, few studies have delved into the comparative efficacy of these varying doses in attenuating the response elicited by laryngoscopy and endotracheal intubation. Recognizing this gap, we have undertaken a prospective comparative study aimed at assessing the effectiveness of two distinct dexmedetomidine doses (0.5 mcg/kg vs. 1 mcg/kg) in mitigating the hemodynamic response associated with these procedures.

In our study, after a 10-minute drug administration (T1), both groups saw decreased HRs from baseline. Group A's HR reduced to 71.5 (-7.8%), and Group B's HR dropped to 60.9 (-19.7%). The HR decrease was significantly more pronounced in Group B ($p < 0.5$). From intubation to 10 minutes post-intubation (T2-T6), HRs increased in both groups, but Group B consistently had a smaller increase compared to Group A. However, these differences were not statistically significant ($p > 0.05$). It is interesting to note that the HR remained below the baseline level in both groups starting from the 5th minute onwards. This finding resonates with those of other researchers in the field. Kato et al. documented a noteworthy reduction in HR at 5 and 10 minutes after drug infusion in Group A (1 mcg) compared to Group B (0.6 mcg).[12] Even after intubation, Group A consistently exhibited lower HR levels than Group B. A study reported a significant decrease in mean HR within the first two minutes following the infusion of dexmedetomidine at 1 mcg/kg over a 10-minute period.[13] Similarly, Maowei et al. noted transient bradycardia in response to infusion of dexmedetomidine nearly about 3%.[14] Shin et al., 2013 also observed a temporary HR reduction after the administration of 1 µg/kg dexmedetomidine. [15] Additionally, at 5 and 10 minutes post-intubation, both groups in our study displayed HR levels below baseline ($p < 0.05$), aligning with previous findings.[16,17] The consistent alignment of our findings with existing literature underscores the potential of dexmedetomidine to modulate HR responses during laryngoscopy and intubation.

The study compared BP changes between Group A and Group B at various time points. SBP at T0 was 117.1 mmHg in Group A and 119.7 mmHg in Group

B. After 10 minutes (T1), SBP decreased by approximately 8.4% in both groups. At intubation (T2), Group B experienced a significant decrease (4.3%), while Group A had a slight rise (4.8%) compared to baseline. This trend continued till 10 mins post-intubation. For DBP, baseline (T0) was 73.1 mmHg in Group A and 74.4 mmHg in Group B. After infusion (T1), DBP decreased by 12.4% and 11.8% in Group A and B respectively. At intubation (T2), Group A increased by 8.5%, while Group B decreased by 1.6%. Similar trends persisted throughout, with Group B showing more significant reductions. MAP baseline (T0) was 88.6 mmHg in Group A and 89.3 mmHg in Group B. After infusion (T1), MAP decreased by around 11.2% in Group A and 10.3% in Group B. At intubation (T2), Group A increased by 3.0%, whereas Group B decreased by 4.0%. Group B consistently showed greater reduction in MAP from intubation till 10 mins post-intubation. Thus, Group B exhibited more significant reductions in SBP, DBP, and MAP compared to Group A after intubation, sustaining this trend throughout the subsequent readings till 10 mins post-intubation.

Our investigation aligns seamlessly with the existing body of research, providing valuable insights into the use of dexmedetomidine for attenuating hemodynamic responses during laryngoscopy and intubation. One noteworthy study demonstrated that nebulized dexmedetomidine at 1 µg/kg effectively blunts the stress response to laryngoscopy and intubation without adverse effects, which aligns with our observations of reduced SBP, DBP, and MAP post-intubation. [18] Similarly, the findings of Misra et al. suggested that nebulized dexmedetomidine at the same dosage not only reduces heart rate increases post-laryngoscopy but also decreases intraoperative anesthetic consumption without affecting early postoperative nausea and vomiting, indicating its potential as a favorable alternative to intravenous administration in short-duration surgeries.[19] Additionally, Chappa and associates reported that nebulized dexmedetomidine effectively blunts the pressor response to laryngoscopy and intubation, with statistically significant reductions in MAP, SBP, and DBP values, and a lower mean dose of propofol required for induction.[20] Our study's results are further supported by a team who demonstrated that combining intravenous dexmedetomidine with nebulized lidocaine provides superior control of post-intubation heart rate and blood pressure compared to either treatment alone. This combination offers enhanced hemodynamic stability. [21] Furthermore, research by Mahajan et al. highlighted the efficacy of dexmedetomidine (1 µg/kg) and magnesium sulphate (30 mg/kg) in significantly reducing heart rate and blood pressure during laryngoscopy and intubation, whereas normal saline showed no such pressor response.[22] This supports our findings on dexmedetomidine's effectiveness in mitigating hemodynamic stress. Another study on 80 patient cohort emphasized dexmedetomidine's (1 µg/kg) superior performance over nalbuphine (0.2 mg/kg) in attenuating hemodynamic responses, reinforcing its role as a potent agent in managing these physiological changes. [23] Comparative studies, such as the one conducted by Bhaskar and Khan, found that dexmedetomidine (0.5 µg/kg) was more effective than clonidine (1 µg/kg) in reducing mean heart rate, SBP, and DBP post-intubation, highlighting its robustness in attenuating pressor responses. [24] Additionally, other work noted a significant reduction in hemodynamic parameters with a lower dose of dexmedetomidine (0.6 µg/kg body weight), suggesting a dose-dependent relationship.[25] Hou et al. observed that the effects of loading dose of dexmedetomidine (1 µg/kg over 10 min) vary with anesthesia type, increasing arterial blood pressure when combined with total intravenous anesthesia and decreasing it with inhaled anesthesia in pediatric patients with obstructive sleep apnea-hypopnea syndrome, indicating the need for context-specific dosing considerations. [26]

Lee and Kim also demonstrated that a single preanesthetic dose of 0.5 µg/kg dexmedetomidine effectively suppresses hemodynamic responses in elderly patients with hypertension, further reinforcing its clinical utility across diverse patient populations. [27]These studies collectively underscore the consistent efficacy of dexmedetomidine in mitigating hemodynamic responses during laryngoscopy and intubation, aligning with our findings and highlighting its potential for broader clinical applications.

Our vigilant monitoring of adverse events yielded significant insights in both groups. In Group A, a single patient (2%) experienced hypertension (SBP > 140 mmHg) during laryngoscopy, while none in Group B did. In Group B, one case (2%) of bradycardia (HR < 55/min) emerged during the 10-minute drug infusion, unlike Group A. Importantly, hypotension (SBP < 30% from baseline) and tachycardia (HR > 30% of baseline) were absent in both cohorts, with no statistically significant differences noted ($p > 0.05$). These findings are consistent with previous work that demonstrated that a preanesthetic single infusion of dexmedetomidine at 1 µg/kg maintains stable hemodynamics and decreases anesthetic consumption without affecting recovery profiles. [15] A similarly work identified 0.75 µg/kg intravenous dexmedetomidine as the optimal dose to attenuate the stress response during laryngoscopy and intubation without adverse effects. [28] Moreover, a recent work showed that combining 0.5 µg/kg dexmedetomidine with 4% sevoflurane is more effective in attenuating the pressor response, albeit with a minor risk of bradycardia and hypotension. [29] This aligns with our finding that higher doses can be associated with manageable adverse events. Sharma and co-worker corroborated that dexmedetomidine at both 0.5 µg/kg and 1 µg/kg is

effective in reducing the induction dose of propofol with a lesser incidence of adverse effects at the lower dose.[30] Furthermore, a study done in year 2020 also confirms that 1 µg/kg dexmedetomidine significantly attenuates hemodynamic responses without significant postoperative adverse events, supporting the efficacy and safety profile we observed. [31] A simultaneously conducted research also noted that 1 µg/kg is more effective than 0.5 µg/kg in attenuating hemodynamic stress responses during cardiac surgery, without causing hypotension or bradycardia, emphasizing the higher dose's enhanced efficacy.[32] However, Kakkar et al. reported that while both 0.5 µg/kg and 1 µg/kg dexmedetomidine doses are effective in attenuating responses, clonidine at 1 µg/kg is associated with fewer side effects, particularly bradycardia.[10]

Therefore, our rigorous analysis supports the efficacy of a 1 mcg/kg dexmedetomidine loading dose in attenuating hemodynamic responses during laryngoscopy and endotracheal intubation, compared to a 0.5 mcg/kg dose. Additionally, the occurrence of bradycardia during drug infusion suggests the need for careful monitoring in patients receiving the higher dose.

Limitations of our study include its small sample size, involving only 100 patients, which may restrict the generalizability of the findings. Additionally, we did not measure cardiac output or serum catecholamine levels, potentially limiting a comprehensive understanding of the hemodynamic responses.

CONCLUSION

In summary, our investigation into the comparative efficacy of two distinct doses of Dexmedetomidine (0.5 mcg/kg and 1 mcg/kg) in mitigating the hemodynamic response to laryngoscopy and intubation yielded compelling results. Notably, the administration of IV Dexmedetomidine at 1 mcg/kg exhibited pronounced superiority in attenuating the pressure response, signifying its potential clinical utility. This heightened dose holds promise for patients with a medical history encompassing myocardial ischemia, hypertension, and cerebrovascular accidents, as it effectively curbed the undesirable stress response elicited during laryngoscopy and intubation. It is prudent to exercise circumspection, however, when administering the 1 mcg/kg dosage, as evidenced by the documented instances of bradycardia during drug infusion. Collectively, our findings indicates the prospect of tailoring dexmedetomidine dosing to the distinctive requisites of specific patient cohorts, while concurrently advocating for meticulous monitoring to ensure patient well-being.

Conflict of Interest: Authors declares no conflict of interest

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