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COMPARATIVE EVALUATION OF INTRAVENOUS DEXMEDETOMIDINE AND MAGNESIUM SULFATE FOR ATTENUATION OF PRESSOR RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION: A RANDOMIZED CONTROL STUDY

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ABSTRACT

Objectives: The present study compared the efficacy of intravenous dexmedetomidine versus intravenous magnesium sulfate for attenuation of pressor response during laryngoscopy and endotracheal intubation.

Methods: This prospective randomized study was conducted among 52 patients undergoing general anesthesia. Patients were divided into two groups – Group D and Group M of 26 patients each. Group D received Inj. Dexmedetomidine 1 mcg/kg intravenous (IV) and Group M received Inj. Magnesium sulfate 30 mg/kg IV. diluted in 100 mL normal saline over 30 min before induction of general anesthesia. Hemodynamic parameters such as heart rate (HR), systolic blood pressure, diastolic blood pressure, and mean arterial pressure were compared between both the groups till 7 min after intubation.

Results: Demographic parameters did not differ significantly between the groups (p>0.05). Group D showed significantly lower HR and blood pressure after induction, at intubation and at 1, 3, 5, and 7 min post-intubation compared to Group M (p<0.05).

Conclusion: Our study concluded that administering dexmedetomidine intravenously at a dose of 1 mcg/kg effectively reduced the sympathetic response to laryngoscopy and intubation as compared to magnesium sulfate 30 mg/kg intravenously.

Keywords: Dexmedetomidine, Magnesium sulfate, Laryngoscopy, Endotracheal intubation, Pressor response.

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INTRODUCTION

Laryngoscopy and endotracheal intubation are essential components of general anesthesia that allow for airway management and mechanical ventilation during surgical procedures. However, these maneuvers can trigger significant sympathetic activation and hemodynamic stress responses in patients. The insertion of a laryngoscope blade and endotracheal tube stimulates proprioceptors in the supraglottic region and trachea, leading to a surge in catecholamine release from the adrenal medulla and adrenergic nerve terminals [1]. This results in transient but potentially harmful increases in heart rate (HR), blood pressure, and myocardial oxygen demand [2].

While these hemodynamic changes may be well-tolerated by healthy individuals, they can pose serious risks for patients with pre-existing cardiovascular conditions, cerebrovascular disease, or increased intracranial pressure [3]. The abrupt rise in blood pressure and HR may precipitate myocardial ischemia, left ventricular failure, cerebral hemorrhage, or ruptured cerebral aneurysm in susceptible patients. Therefore, attenuation of the pressor response to laryngoscopy and intubation is an important goal in anesthetic management, particularly for high-risk patients [3].

Numerous pharmacological agents and techniques have been investigated to blunt this stress response, including opioids, betablockers, calcium channel blockers, vasodilators, and local anesthetic infiltration of the airway. However, many of these approaches have limitations related to side effects, delayed recovery, or inadequate efficacy. There is ongoing research to identify optimal strategies that can effectively attenuate the hemodynamic response while maintaining overall cardiovascular stability and allowing for rapid emergence from anesthesia [4].

In recent years, there has been growing interest in the use of alpha-2 adrenergic agonists and magnesium sulfate for this purpose. Dexmedetomidine, a highly selective alpha-2 agonist, has emerged as a promising option due to its sympatholytic, analgesic, and sedative properties without significant respiratory depression [5]. By activating presynaptic alpha-2 receptors, it inhibits norepinephrine release and attenuates central sympathetic outflow. This results in decreased catecholamine levels and blunting of the stress response to laryngoscopy and intubation [5].

Magnesium sulfate has also gained attention as a potential agent for attenuating the pressor response. It acts as a physiological calcium channel blocker and inhibits catecholamine release from adrenergic nerve terminals and the adrenal gland [6]. In addition, magnesium has vasodilatory effects and may provide some degree of analgesia through N-methyl-D-aspartate receptor antagonism. These properties make it an attractive option for modulating the cardiovascular changes associated with airway instrumentation [6].

Both dexmedetomidine and magnesium sulfate have shown promise in various studies for attenuating the hemodynamic response to laryngoscopy and intubation.

The primary aim of this study is to compare the efficacy of intravenous (IV) dexmedetomidine versus IV magnesium sulfate in attenuating the pressor response to laryngoscopy and endotracheal intubation [7].

The secondary objectives evaluate any adverse effects or complications associated with their use. This information will help guide clinical decision-making and optimize perioperative management strategies,

particularly for patients at higher risk of complications from extreme hemodynamic fluctuations [8].

However, there is a lack of consensus regarding their comparative efficacy and optimal dosing regimens. Direct comparisons between these two agents are limited, and their relative benefits and drawbacks need further evaluation [9].

Hence by conducting a prospective, randomized study, we seek to provide valuable data on the relative effectiveness of these two agents in modulating HR, blood pressure, and overall hemodynamic stability during this critical phase of anesthesia induction [10].

METHODS

Study design and patient selection

This prospective randomized study was conducted at the Department of Anesthesiology, Dhiraj Hospital, S.B.K.S. Medical Institute and Research Center, Piparia, Vadodara, Gujarat after obtaining Institutional Ethics Committee approval (Research protocol no. SVIEC/BNPG21/SEP/22/42). Written informed consent was obtained from 52 patients of American Society of Anesthesiologists (ASA) physical status I and II, aged between 18 and 60 years, of either gender, scheduled for elective surgeries under general anesthesia.

Sample size calculation

A total 52 patients were included, with the sample size calculated based on a confidence level of 75% and margin of error 5%. The minimum sample size for each group was determined to be 26 patients using a sampling formula.

Inclusion criteria

Patients willing to sign written and informed consent. Male or female patients between ages of 18 and 60 years. Patients belonging to ASA physical status I and II. Patients posted for elective surgeries under general anesthesia.

Exclusion criteria

Patients who were unwilling to participate, ASA Grade III or IV as well as those with significant cardiovascular, cerebrovascular, hepatic, or renal diseases. Individuals with arrhythmias or heart block, psychiatric conditions, or those who were pregnant or as a according to thesis lactating were also excluded. Furthermore, patients on beta-blocker therapy or with a history of allergy to the study drugs were not eligible. Other exclusion factors comprised an anticipated difficult airway, a body mass index >30 kg/m², neuromuscular diseases, and a preoperative serum magnesium level exceeding 2.5 mEq/L.

Study procedure

Patients were randomly divided by computer generated random number in sealed envelope into two equal groups of 26 each.

- Group D: Received injection dexmedetomidine 1 mcg/kg intravenously, diluted in 100 mL normal saline, infused over 30 min before induction of general anesthesia.
- Group M: Received injection magnesium sulfate 30 mg/kg intravenously, diluted in 100 mL normal saline, infused over 30 min before induction of general anesthesia.

The consolidated standards of reporting trails flowchart are presented in Fig. 1.

Anesthesia technique

All patients underwent a thorough pre-anesthetic evaluation on the day before surgery. A through clinical examination and airway assessment was done. Patients were kept nil per orally 6 h and 4 h before surgery for solids and liquids, respectively, before surgery. Written and informed consent was taken. On the day of surgery IV, access was secured and standard monitors were attached, including electrocardiogram, non-invasive blood pressure, and Oxygen saturation (SpO $_2$).

After recording baseline parameters, the study drug was infused over 30 min as per group allocation. Patients were shifted to OT. Patient was

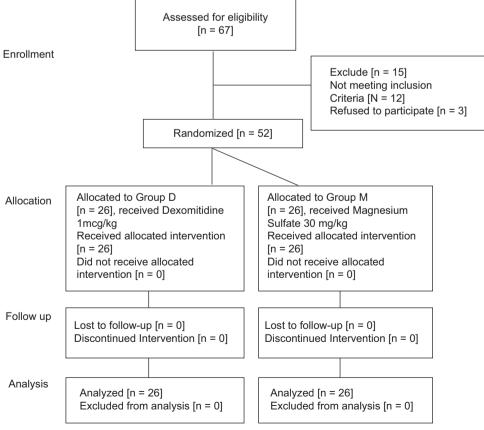


Figure 1: CONSORT Flowchart

pre-oxygenated with 100% oxygen for 3 min. Anesthesia was induced with injection propofol 2 mg/kg intravenously. After confirmation of mask ventilation, injection succinvlcholine 2 mg/kg was given to facilitate endotracheal intubation. Laryngoscopy and intubation were performed by an experienced anesthesiologist. HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at several time points throughout the study: At baseline, following the infusion of the study drug, after induction, during laryngoscopy and intubation, and at 1, 3, 5, and 7 min post-intubation. Anesthesia was maintained with oxygen, nitrous oxide, isoflurane, and intermittent boluses of injection atracurium 0.1 mg/kg IV. according to train of four count. Ventilation was adjusted to maintain end-tidal carbon dioxide between 35 and 40 mmHg. At the end of surgery, residual neuromuscular blockade was reversed with injection neostigmine 0.05 mg/kg and injection glycopyrrolate 0.01 mg/kg intravenously. Sedation was assessed using the Ramsay Sedation Scale (RSS) every 30 min post-extubation until a score of 2 was achieved. Any adverse events or complications were noted.

Statistical analysis

Sample size was calculated based on previous studies, considering a power of 80% and a significance level of 5%. Data were tabulated and analyzed using appropriate Excel and analyzed using Jamovi 2.4.14 software. Numerical variables were presented as mean and standard deviation, while categorical variables were expressed as frequency and percentage. The unpaired t-test and Chi-square used for comparing continuous variables, while the Chi-square test was applied for categorical variables. p<0.05 was considered statistically significant.

RESULTS

This prospective study compared the efficacy of IV dexmedetomidine versus IV magnesium sulfate for attenuation of pressor response during laryngoscopy and endotracheal intubation. A total of 52 patients undergoing elective surgeries under general anesthesia were enrolled and divided into two equal groups of 26 each: Group D and Group M.

Demographic characteristics

The demographic profiles of patients in both groups were comparable, with no statistically significant differences in age, gender distribution, ASA grade, or mean weight (p>0.05) (Table 1).

HR

Baseline HRs were similar between the two groups. Following administration of the study drugs and induction, both groups showed a slight decrease in HR, though this was not statistically significant (Table 2 and Fig. 2). During laryngoscopy and intubation, there was a transient increase in HR in both groups. However, the magnitude of increase was significantly higher in Group M compared to Group D (p=0.005). At 1, 3, 5, and 7 min post-intubation, Group D demonstrated a statistically significant lower HR compared to Group M (p=0.005, 0.01, 0.001, and 0.001, respectively).

SBF

Baseline SBP was comparable between groups (Table 2 and Fig. 3). Both groups experienced a decrease in SBP after study drug administration and induction, though this was not statistically significant. During laryngoscopy and at 1, 3, 5, and 7 min post-intubation, Group D showed significantly lower SBP compared to Group M (p=0.002, 0.001, 0.001, and 0.001, respectively).

DBP

The pattern observed with DBP mirrored that of SBP (Table 2 and Fig. 4). Group D exhibited significantly lower DBP during laryngoscopy and at 1, 3, 5, and 7 min post-intubation compared to Group M (p=0.001, 0.003, 0.03, 0.01, and 0.001, respectively).

MAP

Baseline MAP was similar between groups (Table 2 and Fig. 5). During laryngoscopy and at subsequent time points (1, 3, 5, and 7 min

Table 1: Data presented as mean±SD or frequency (percentage)

Group D	Group M	p-value
12 (46.2)	10 (38.5)	0.57
14 (53.8)	16 (61.5)	
on		
21 (80.8)	16 (61.5)	0.12
5 (19.2)	10 (38.5)	
SD		
66.92±8.202	64.08±9.02	0.24
45.12	44.98	0.78
1.479	2.952	
	12 (46.2) 14 (53.8) on 21 (80.8) 5 (19.2) SD 66.92±8.202 45.12	12 (46.2) 10 (38.5) 14 (53.8) 16 (61.5) 500 21 (80.8) 16 (61.5) 5 (19.2) 10 (38.5) 50 66.92±8.202 64.08±9.02 45.12 44.98

n=26 per group. ASA: American society of anesthesiology, SD: Standard deviation

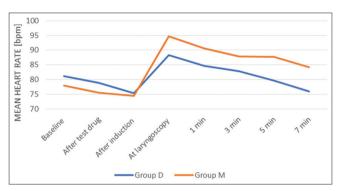


Fig. 2: Mean heart rate (beats per minute) in both the groups intraoperatively data presented

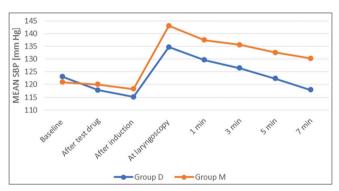


Fig. 3: Mean systolic blood pressure (in mm Hg) in both the groups intraoperatively data presented as mean±SD. n=26 per group. SD: Standard deviation

post-intubation), Group D maintained significantly lower MAP compared to Group M (p=0.001, 0.03, 0.03, 0.002, and 0.002, respectively).

Oxygen saturation

There were no significant differences in oxygen saturation (SpO_2) levels between Group D and Group M at any time point during the observation period (p>0.05) (Table 2 and Fig. 6).

Sedation scores

The RSS scores at extubation and thereafter were comparable between both groups, with a median value of 2 in both the groups. No statistically significant difference was noted (p>0.05) (Table 3).

Adverse effects

No notable side effects or complications were observed in either group during the study period.

DISCUSSION

This prospective observational study aimed to compare the efficacy of IV dexmedetomidine versus IV magnesium sulfate in attenuating the

Table 2: Hemodynamic parameters and oxygen saturation for both Group D (dexmedetomidine) and Group M (magnesium sulfate)

Time	Heart rate (HR) group D	Heart rate Heart rate HR) group D (HR) group M	Systolic blood pressure (SBP) group D	Systolic blood pressure (SBP) group M	Diastolic blood Diastolic blood pressure (DBP) group M	Diastolic blood pressure (DBP) group M	Mean arterial pressure (MAP) group D	Mean arterial pressure (MAP) group M	Oxygen saturation group D	Oxygen saturation group M
Baseline	81.15±7.428	77.92±7.515	123.04±7.164	120.92±7.359	83.54±6.935	82.15±6.117	92.88±5.982	93.92±6.286	100.00±0.000	100.00±0.000
After test drug	78.88±7.096	75.46±6.494	117.77±7.334	120.04±5.855	80.62±5.084	79.46±6.205	91.42 ± 4.536	93.00±5.012	99.88±0.326	99.88±0.326
After induction	75.35±7.304	74.38±6.432	115.12 ± 8.567	118.23 ± 6.713	79.42±6.712	77.04±6.277	91.50 ± 6.719	90.77±5.729	99.96 ± 0.196	100.00 ± 0.000
At laryngoscopy	88.27±7.035	94.69 ± 8.817	134.65 ± 7.802	143.08 ± 11.990	85.69±8.465	94.04±6.283	104.15 ± 6.044	111.23 ± 7.865	100.00 ± 0.000	100.00 ± 0.000
1 min	84.58±7.819	90.62±6.969	129.62 ± 7.726	137.46 ± 9.166	84.69 ± 9.452	91.65±6.449	102.46 ± 6.420	106.81 ± 7.611	100.00 ± 0.000	100.00 ± 0.000
3 min	82.85±7.708	87.88±6.731	126.42 ± 7.484	135.58 ± 8.603	85.35±7.899	89.69±6.260	101.04 ± 6.885	105.46 ± 7.793	99.96 ± 0.196	100.00 ± 0.000
5 min	79.65±7.526	87.65±6.627	122.38 ± 6.067	132.54 ± 6.707	81.81 ± 7.960	86.69±6.272	97.92±6.393	104.35 ± 7.990	100.00 ± 0.000	100.00 ± 0.000
7 min	75.92±8.265	84.23±5.609	117.92 ± 6.105	130.23 ± 6.458	78.04±9.569	86.62±7.955	93.50±7.050	100.12 ± 7.538	100.00 ± 0.000	99.88±0.326

Data presented as mean±SD. n=26 per group. HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, SD: Standard deviation

Table 3: Data presented as median values

After extubation	Group D	Group M	p-value
0 min	2	2	
30 min	2	2	
60 min	0	0	
90 min	0	0	
120 min	0	0	
180 min	0	0	

n=26 per group. RSS: Ramsay Sedation Scale

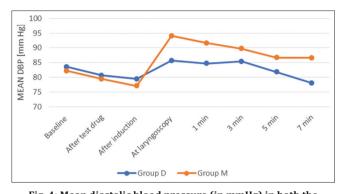
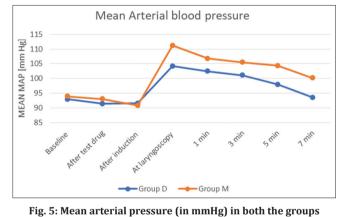


Fig. 4: Mean diastolic blood pressure (in mmHg) in both the groups intraoperatively data presented as mean±SD. n=26 per group. SD: Standard deviation



intraoperatively data presented as mean±SD. n=26 per group. SD:

Standard deviation

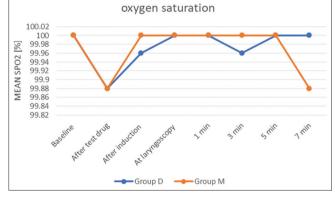


Fig. 6: Data presented as mean±SD. n=26 per group. SD: Standard deviation

pressor response during laryngoscopy and endotracheal intubation. The results demonstrate a superior effect of dexmedetomidine in

maintaining hemodynamic stability during this critical period of anesthesia induction.

The demographic profiles of both groups were comparable, ensuring that the observed differences in hemodynamic parameters could be attributed to the interventions rather than underlying patient characteristics. This strengthens the validity of our findings and allows for more robust conclusions.

In our study, baseline HR values were comparable between the two groups (Group D: 81.15 ± 7.428 bpm; Group M: 77.92 ± 7.515 bpm; p=0.12]. After administration of the test drugs and induction of anesthesia, there was a slight decrease in HR in both groups, but the difference was not statistically significant. (p>0.05) During laryngoscopy and intubation, HR increased in both groups, but the increase was significantly higher in Group M compared to Group D (p=0.005). Post-intubation, HR decreased gradually in both groups, with Group D showing a significantly lower HR at 1, 3, 5, and 7 min compared to Group M (p=0.005, 0.01, 0.001, and 0.001, respectively).

Our findings align with those of Zia $et\ al.$ (2023), who observed that dexmedetomidine at 1 µg/kg was more effective in attenuating HR response compared to magnesium sulfate at 30 mg/kg during laryngoscopy and intubation [7]. They reported a significant increase in HR in the magnesium sulfate group compared to the dexmedetomidine group immediately after intubation and at subsequent time points. Similarly, Choudhary $et\ al.$ found that dexmedetomidine provided superior control over HR compared to magnesium sulfate during the peri-intubation period. Their study demonstrated that dexmedetomidine effectively blunted the tachycardic response associated with laryngoscopy and intubation [8].

Baseline SBP was comparable between the groups (Group D: 123.04 ± 7.164 mmHg; Group M: 120.92 ± 7.359 mmHg; p=0.29). Postadministration of test drugs and induction, there was a reduction in SBP in both groups, but the difference was not statistically significant. During laryngoscopy and intubation, SBP increased in both groups, with Group M exhibiting a significantly higher increase compared to Group D (p=0.004). Post-intubation, SBP decreased gradually, with Group D showing significantly lower SBP at 1, 3, 5, and 7 min compared to Group M (p=0.002, 0.001, 0.001, and 0.001, respectively).

Our results are consistent with those of Ahmad $\it{et~al.}$, (2023) who found that dexmedetomidine was more effective than magnesium sulfate in controlling SBP during laryngoscopy and intubation. Their study demonstrated that patients in the dexmedetomidine group had significantly lower SBP compared to the magnesium sulfate group at all measured time points [9]. El-Wakeel $\it{et~al.}$ also reported that dexmedetomidine at 1 $\it{\mu}g/kg$ provided better attenuation of SBP response compared to magnesium sulfate at 30 $\it{mg/kg.}$ The study highlighted the superior efficacy of dexmedetomidine in maintaining hemodynamic stability during airway manipulation [10].

Baseline DBP values were similar between the groups (Group D: 83.54 ± 6.935 mmHg; Group M: 82.15 ± 6.117 mmHg; p=0.44). After administration of test drugs and induction, DBP decreased in both groups without significant differences. During laryngoscopy and intubation, DBP increased in both groups, but the increase was significantly higher in Group M compared to Group D (p=0.001). Post-intubation, DBP decreased gradually, with Group D showing significantly lower DBP at 1, 3, 5, and 7 min compared to Group M (p=0.003, 0.03, 0.01, and 0.001, respectively).

Our findings are supported by the study of Mahajan *et al.*, (2018) who demonstrated that dexmedetomidine significantly attenuated the increase in DBP during laryngoscopy compared to magnesium sulfate. The study indicated that dexmedetomidine effectively controls both systolic and diastolic components of blood pressure [11]. Similarly, Swaro *et al.* (2016) found that dexmedetomidine at doses of 0.5 and

 $1 \mu g/kg$ reduced DBP during intubation more effectively than placebo, with the $1 \mu g/kg$ dose showing superior efficacy [12].

Baseline MAP was comparable between the groups (Group D: 92.88±5.982 mmHg; Group M: 93.92±6.286 mmHg; p=0.54). After administration of test drugs and induction, MAP decreased slightly in both groups without significant differences. During laryngoscopy and intubation, MAP increased in both groups, with a significantly higher increase in Group M compared to Group D (p=0.001). Post-intubation, MAP decreased gradually, with Group D showing significantly lower MAP at 1, 3, 5, and 7 min compared to Group M (p=0.03, 0.03, 0.002, and 0.002, respectively).

Our results are in agreement with those of Sebastian et~al., who found that dexmedetomidine at 0.75 µg/kg effectively attenuated the increase in MAP during laryngoscopy and intubation compared to lower doses and placebo. The study highlighted the dose-dependent efficacy of dexmedetomidine in controlling MAP [13]. Thapa and Gauchan (2019) also reported that dexmedetomidine at both 0.5 and 1 µg/kg doses attenuated the MAP response, with no significant difference between the two doses, suggesting that even lower doses may be effective [14].

Dexmedetomidine's superior efficacy in attenuating the stress response can be attributed to its unique pharmacological profile. As a highly selective $\alpha 2$ -adrenergic agonist, dexmedetomidine reduces sympathetic outflow and catecholamine release [11]. This mechanism explains the more stable HR and blood pressure observed in Group D during laryngoscopy and post-intubation periods.

The results for magnesium sulfate, while showing some attenuation of the pressor response, were less pronounced compared to dexmedetomidine. Magnesium's effects are likely mediated through its calcium channel blocking properties and inhibition of catecholamine release [4]. The relatively lower efficacy observed in our study might be due to the dose used (30 mg/kg) or the complex interplay of magnesium's various physiological actions. This finding is consistent with the study by Choudhary *et al.* [8] which also found dexmedetomidine to be more effective than magnesium sulfate in attenuating the hemodynamic response.

Interestingly, both groups showed a slight decrease in hemodynamic parameters after drug administration and induction, suggesting some pre-emptive effect. However, the significant differences emerged during and after laryngoscopy, highlighting the critical nature of this period and the importance of effective pharmacological intervention.

In our study, there were no significant differences in oxygen saturation (\mbox{SpO}_2) levels between the two groups at all-time intervals (p>0.05), indicating that neither drug adversely affected oxygenation. In addition, the Ramsay Sedation Scores were similar in both groups post-extubation, with no patients experiencing excessive sedation or delayed recovery.

Chattopadhyay *et al.* (2016) reported that dexmedetomidine provided adequate sedation without respiratory depression. Similarly, Zhang *et al.* (2023) found that nebulized dexmedetomidine did not compromise SpO₂ levels while providing effective sedation [15,16].

The lack of significant differences in oxygen saturation between the groups is reassuring, indicating that the hemodynamic effects of both drugs were achieved without compromising oxygenation. This is particularly important for dexmedetomidine, which has been associated with respiratory depression at higher doses [17].

The comparable RSS scores between groups suggest that the superior hemodynamic control achieved with dexmedetomidine was not at the cost of excessive sedation. This is a crucial finding, as it implies that dexmedetomidine can effectively attenuate the stress response while still allowing for timely emergence from anesthesia.

The absence of notable side effects in both groups is encouraging and suggests that the doses used in this study (1 mcg/kg for dexmedetomidine and 30 mg/kg for magnesium sulfate) strike a good balance between efficacy and safety. This aligns with the findings of El-Wakeel *et al.* [18] who reported no adverse events or hemodynamic instability with similar doses.

Limitations

The study had a small sample size from a single center and lacked blinding, which could limit the generalizability of the results. In addition, the study focused on surrogate outcomes over a short followup period in a specific demographic, without a standardized placebo group.

Clinical implications

For patients undergoing general anesthesia, particularly those at higher risk of complications from hemodynamic fluctuations, dexmedetomidine appears to be a superior choice for attenuating the stress response to laryngoscopy and intubation. Its ability to maintain more stable hemodynamics without compromising oxygenation or causing excessive sedation makes it an attractive option for a wide range of surgical procedures.

CONCLUSION

Our observational study suggests that dexmedetomidine may be more effective than magnesium sulfate in attenuating the pressor response at 1 mcg/kg is more effective than IV magnesium sulfate at 30 mg/kg in attenuating the pressor response to laryngoscopy and endotracheal intubation. A larger, multi-center trial could provide more definitive evidence. In addition, we focused on the immediate perioperative period; future studies could investigate longer-term outcomes or effects on specific high-risk patient populations.

Our findings contribute to the growing body of evidence supporting the use of dexmedetomidine in perioperative hemodynamic management. Future research should focus on optimizing dosing regimens, investigating potential synergistic combinations, and exploring the benefits in specific patient populations.

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Sara Mary Thomas Professor and Head , Sujay Ghetiya Senior resident , Arpit shah assistant professor, Jigisha Mehta : Associate professor

AUTHORS' CONTRIBUTIONS

Dr. Sara Mary Thomas: Conceptualization, methodology, supervision, manuscript review, and editing. Dr. Sujay Ghetiya: Data collection, analysis, manuscript writing, and revision. Dr. Arpit Shah: Data collection, statistical analysis, and manuscript review. Dr. Jigisha Mehta: Methodology, data interpretation, and manuscript review.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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