

ORIGINAL ARTICLE

Double Blind RCT comparing Dexmedetomidine with Magnesium Versus Lignocaine for Pressor response attenuation to Laryngoscopy

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HOW TO CITE THIS ARTICLE:

Reena Nebu, Ekta Eknath Mohite et. al, Double Blind RCT comparing Dexmedetomidine with Magnesium versus Lignocaine for Pressor Response attenuation to Laryngoscopy. Ind J Anesth Analg. 2026; 13(1): 07-13.

ABSTRACT

Aims and Objectives: To compare hemodynamic responses to direct laryngoscopy and endotracheal intubation with intravenous dexmedetomidine combined with either magnesium sulphate or lignocaine.

Study Site and Design: Hospital-based, double-blinded randomized controlled study at a Municipal General Hospital.

Study Population and Sample Size: 128 adult patients (ASA I-II) scheduled for elective surgery under general anaesthesia, divided into two groups of 64 each.

Methodology: Patients received premedication with study drugs. Standardized anaesthesia induction was followed. Vital parameters like heart rate, blood pressures (SBP, DBP, MAP), SpO₂, and ECG were recorded at baseline, pre-induction, and at 1, 3, 5, 10, and 15 minutes post-induction. Postoperative analgesia and recovery times were monitored.

Statistical Analysis: Data were analysed using SPSS v22. Continuous variables were expressed as mean $\pm$ SD and compared using unpaired t-tests; categorical variables were analysed using Chi-square tests. Trends over time were evaluated; significance was set at $p < 0.05$.

Results: Demographics including age, gender, ASA grade, BMI, and comorbidities were comparable ($p > 0.05$). Both drug combinations maintained stable hemodynamics during and after intubation, with no significant differences in HR, BP, MAP, SpO₂, or ECG. Postoperative analgesia and induction-to-extubation times were similar ($p > 0.05$).

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➤ Received: 25-10-2025 ➤ Accepted: 20-12-2025



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Conclusion: Dexmedetomidine combined with either magnesium sulphate or lignocaine effectively attenuates the hemodynamic response to laryngoscopy and intubation, with stable perioperative parameters and comparable safety profiles. Both combinations are suitable for ASA I-II patients.

KEYWORDS

• Dexmedetomidine • Magnesium Sulphate • Lignocaine • Hemodynamic Response • Laryngoscopy

INTRODUCTION

Direct laryngoscopy and endotracheal intubation often trigger a sympathetic stress response, causing tachycardia and hypertension, which may lead to serious cardiovascular events in vulnerable patients.¹ This pressor response results from catecholamine release due to mechanical stimulation of the airway.^{2,3} Various pharmacological agents have been studied to attenuate this response, including dexmedetomidine, lignocaine, and magnesium sulphate.⁴⁻⁹ Combining these agents may provide synergistic effects, stabilizing hemodynamics during intubation. This study compares the efficacy of dexmedetomidine magnesium sulphate versus dexmedetomidine lignocaine combinations in controlling the hemodynamic stress response in ASA I and II patients.

MATERIALS AND METHODS

A hospital-based double-blinded randomized controlled study was conducted on in-patients at a Municipal General Hospital. Adult patients of ASA physical status I and II scheduled for elective surgery receiving general anaesthesia with direct laryngoscopy and intubation were included. Patients aged 18-60 years, of either gender, and without significant comorbidities were enrolled. Exclusion criteria included hypertension, bradycardia (<50 bpm), heart blocks, cardiovascular disease, pregnancy, drug allergies, BMI >30 kg/m², anticipated difficult intubation, hepatic or renal disorders, psychiatric illness, epilepsy, or refusal to participate.

A total of 128 participants were randomized into two groups (n=64 each). Group A received IV Dexmedetomidine 0.5 µg/kg and lignocaine 1.5 mg/kg, and Group B received IV Dexmedetomidine 0.5 µg/kg and magnesium sulphate 30 mg/kg. The

study drugs were infused in 100 ml normal saline over 15-20 minutes, 15 minutes before induction. All patients were premedicated with glycopyrrolate 0.2 mg and fentanyl 2 µg/kg IV. Pre-oxygenation was performed with 100% oxygen for three minutes. Induction was achieved with propofol 2 mg/kg IV, followed by atracurium 0.5 mg/kg IV. Direct laryngoscopy and intubation were performed, and vital parameters were recorded at baseline, 1, 3, 5, 10, and 15 minutes after induction.

Data collection was double-blinded, with randomization via computer-generated sequences and concealment in sequentially numbered, opaque envelopes. Data were analysed using SPSS v24.0. Continuous variables were compared with independent t-tests; categorical variables with chi-square tests. Non-parametric tests were used when data were not normally distributed. A p-value <0.05 was considered statistically significant.

A written and informed consent was taken from all the enrolled patients in a language well understood by them. Approval from IEC (Institutional Ethics Committee) was obtained before starting the study and all information procured from the study was kept confidential and for academic purposes only.

RESULTS

Age distribution showed no significant difference (p=0.911), with mean ages 33.94 ± 10.04 years (Group A) and 34.58 ± 10.43 years (Group B). Gender distribution was similar (p=0.321), with 49 females (76.6%) in Group A and 44 (68.8%) in Group B. ASA grades were comparable (p=0.414). Comorbidities were minimal (p=0.171), and BMI distribution showed no significant difference (p=0.756).

Baseline heart rate¹ was 91.48 ± 14.08 bpm (Group A) and 89.94 ± 13.99 bpm (Group B), with no significant difference at any time

point. Both groups showed significant changes from baseline ($p < 0.001$). Baseline SBP^{II} was 129.66 ± 13.54 mmHg (Group A) and 127.78 ± 13.39 mmHg (Group B), with no intergroup differences; SBP changes were significant within groups ($p < 0.001$). Baseline DBP^{III} was 82.97 ± 8.98 mmHg (Group A) and 82.17 ± 9.41 mmHg (Group B), with significant intra-group changes ($p < 0.001$) but no intergroup differences. MAP^{IV} followed similar patterns. SPO2 remained 100% across all intervals in

both groups. ECG remained normal in all participants.

VAS scores showed no significant difference ($p = 0.576$), with most patients reporting scores between 3 and 4. Cholecystectomy was the most common surgery (50.0% Group A, 54.7% Group B). Time to induction (23.50 ± 4.26 min vs. 24.78 ± 4.04 min; $p = 0.087$) and induction-to-extubation duration (127.00 ± 44.37 min vs. 127.97 ± 47.14 min; $p = 0.905$) were similar.

Table 1: Comparison of Heart Rate between Study Groups

(N=128)

Heart Rate	Group		P Value
	Group A (n=64) Mean (SD)	Group B (n=64) Mean (SD)	
Baseline	91.48 (14.08)	89.94 (13.99)	0.534
Pre-induction	81.55 (13.04)	83.77 (12.51)	0.328
Mean Change	9.9	6.17	
P Value	<0.001**	0.009**	
1 min after Induction	81.84 (9.93)	81.98 (9.38)	0.935
Mean Change	9.61	7.96	
P Value	<0.001**	<0.001**	
3 min after induction	80.16 (9.62)	80.72 (9.77)	0.743
Mean Change	11.29	9.22	
P Value	<0.001**	<0.001**	
5 min after induction	80.14 (10.32)	79.44 (9.14)	0.684
Mean Change	11.31	10.50	
P Value	<0.001**	<0.001**	
10 min after induction	79.47 (10.51)	77.88 (8.97)	0.358
Mean Change	11.98	12.06	
P Value	<0.001**	<0.001**	
15 min after induction	79.09 (9.99)	77.28 (8.88)	0.280
Mean Change	12.36	12.66	
P Value	<0.001**	<0.001**	

Unpaired t Test, P Value Not Significant; Paired t test, P Value ** Significant

Table 2: Comparison of Systolic Blood Pressure between Study Groups

(N=128)

Systolic Blood Pressure	Group		P Value
	Group A (n=64) Mean (SD)	Group B (n=64) Mean (SD)	
Baseline	129.66 (13.54)	127.78 (13.39)	0.432
Pre-induction	118.09 (10.66)	113.70 (12.35)	0.133
Mean Change	11.59	14.08	
P Value	<0.001**	<0.001**	
1 min after Induction	106.77 (12.58)	105.34 (12.63)	0.525
Mean Change	22.89	22.44	
P Value	<0.001**	<0.001**	
3 min after induction	99.89 (11.01)	100.52 (10.52)	0.743
Mean Change	29.77	27.26	
P Value	<0.001**	<0.001**	
5 min after induction	99.11 (11.66)	95.53 (8.93)	0.054

Table Cont....

Systolic Blood Pressure	Group		P Value
	Group A (n=64) Mean (SD)	Group B (n=64) Mean (SD)	
Mean Change	30.55	32.25	
P Value	<0.001**	<0.001**	
10 min after induction	96.64 (8.82)	95.95 (9.25)	0.668
Mean Change	33.02	31.83	
P Value	<0.001**	<0.001**	
15 min after induction	97.46 (9.30)	97.42 (10.08)	0.982
Mean Change	32.20	30.36	
P Value	<0.001**	<0.001**	

Unpaired t Test, P Value * Significant; Paired t test, P Value ** Significant

Table 3: Comparison of Diastolic Blood Pressure between Study Groups (N=128)

Diastolic Blood Pressure	Group		P Value
	Group A (n=64) Mean (SD)	Group B (n=64) Mean (SD)	
Baseline	82.97 (8.98)	82.17 (9.41)	0.625
Pre-induction	77.61 (8.15)	73.91 (10.31)	0.126
Mean Change	5.36	8.26	
P Value	<0.001**	<0.001**	
1 min after Induction	68.48 (10.63)	68.09 (11.70)	0.844
Mean Change	14.49	14.08	
P Value	<0.001**	<0.001**	
3 min after induction	62.41 (10.59)	62.67 (9.87)	0.884
Mean Change	20.56	19.50	
P Value	<0.001**	<0.001**	
5 min after induction	59.73 (9.17)	58.48 (8.51)	0.426
Mean Change	23.24	23.69	
P Value	<0.001**	<0.001**	
10 min after induction	59.77 (8.30)	58.27 (9.58)	0.346
Mean Change	23.20	23.90	
P Value	<0.001**	<0.001**	
15 min after induction	60.80 (9.21)	59.84 (10.44)	0.585
Mean Change	22.17	22.33	
P Value	<0.001**	<0.001**	

Unpaired t Test, P Value *Significant; Paired t test, P Value ** Significant

Table 4: Comparison of Mean Arterial Pressure between Study Groups (N=128)

Mean Arterial Pressure	Group		P Value
	Group A (n=64) Mean (SD)	Group B (n=64) Mean (SD)	
Baseline	97.80 (10.34)	95.80 (10.94)	0.290
Pre-induction	89.51 (8.60)	84.52 (10.46)	0.114
Mean Change	8.29	11.28	
P Value	<0.001**	<0.001**	
1 min after Induction	79.68 (10.61)	78.80 (10.82)	0.646
Mean Change	18.12	17.00	
P Value	<0.001**	<0.001**	
3 min after induction	73.56 (9.78)	73.58 (9.76)	0.993
Mean Change	24.24	22.22	

Table Cont....

Mean Arterial Pressure	Group		P Value
	Group A (n=64) Mean (SD)	Group B (n=64) Mean (SD)	
P Value	<0.001**	0.009**	
5 min after induction	71.31 (9.35)	69.28 (7.84)	0.186
Mean Change	26.49	26.52	
P Value	<0.001**	<0.001**	
10 min after induction	69.95 (7.64)	69.08 (8.46)	0.540
Mean Change	27.85	26.72	
P Value	<0.001**	<0.001**	
15 min after induction	71.17 (8.67)	70.42 (9.37)	0.639
Mean Change	26.63	25.38	
P Value	<0.001**	<0.001**	

Unpaired t Test, P Value Not Significant

DISCUSSION AND CONCLUSION

Endotracheal intubation elicits reflex sympathetic activation due to mechanical stimulation of the larynx and trachea, mediated by increased catecholamine release.^{1,2,4} This manifests as tachycardia and hypertension, potentially compromising myocardial oxygen balance in at-risk patients.¹⁷ Several agents including opioids, β -blockers, lignocaine, and α_2 -agonists have been employed to blunt this response.^{6,10,11}

Dexmedetomidine, a highly selective α_2 -adrenoceptor agonist, attenuates sympathetic outflow and reduces circulating norepinephrine levels.^{9,18} Magnesium sulphate acts as a calcium channel antagonist, inhibiting catecholamine release and providing vasodilation.^{6-8,17} Lignocaine stabilizes neuronal membranes by blocking sodium channels and dampening the hemodynamic response.^{12,19} When dexmedetomidine is combined with either magnesium or lignocaine, their complementary mechanisms may provide additive hemodynamic stability.

In this study, both combinations achieved effective control of peri-intubation hemodynamics without significant intergroup differences. Similar findings were observed by Muchhal *et al.*²⁰ and Srivastava *et al.*²¹, who reported that dexmedetomidine and magnesium each effectively attenuated cardiovascular responses to laryngoscopy. Gunalan *et al.*²² also demonstrated dexmedetomidine's superior efficacy over fentanyl in controlling stress responses.

Lignocaine remains a reliable adjunct due to its rapid onset and membrane-

stabilizing effect.^{12,13,19} Our results align with Tariq and Yaqoob,²⁴ who found comparable attenuation of HR and BP with lignocaine and dexmedetomidine. Collinsworth *et al.*²³ and Benowitz and Meister¹⁴ described its favourable pharmacokinetic profile, supporting its intraoperative use.

Magnesium sulphate, when administered intravenously, has been shown to blunt catecholamine release and stabilize BP.^{7,17,25} Studies by Mohan and Saha,²⁵ Dube and Gosavi,²⁶ and Rishardhan and Chavan²⁷ corroborate our findings, reporting significant suppression of hemodynamic variability without adverse effects.

Comparative analyses further support our data: Eniya *et al.*²⁸ found dexmedetomidine lignocaine combinations equally effective as dexmedetomidine alone, while Mahajan and Kaur²⁹ observed that dexmedetomidine and magnesium both achieved better pressor control than placebo. Balata and Abdel Latif³⁰ confirmed comparable outcomes among dexmedetomidine, magnesium, and lignocaine in abdominal surgeries.

Our findings also align with those of Gupta and Vyas,³¹ who reported dexmedetomidine's efficacy in minimizing HR and BP surges compared to β -blockers, and Honarmand and Safavi,³² who demonstrated magnesium's dose-dependent blunting of intubation-induced cardiovascular changes.

The absence of significant differences between dexmedetomidine magnesium and dexmedetomidine lignocaine groups indicates that both combinations effectively modulate autonomic response during laryngoscopy.

These results highlight the clinical flexibility available to anaesthesiologists based on drug availability, cost, and patient profile.

Minor intra-group variations likely reflect pharmacodynamic differences: lignocaine's shorter duration of action versus magnesium's sustained vasodilatory effect.⁶⁻⁸ However, both agents, when used with dexmedetomidine, ensured hemodynamic stability without bradycardia, hypotension, or desaturation.

The study employed a robust randomized, double-blind design that minimized potential bias. The sample size was adequate and well-distributed between groups. Comprehensive monitoring of multiple hemodynamic parameters provided detailed data for analysis. The study groups were well-matched in terms of demographic and clinical characteristics. The research protocol was standardized and consistently followed throughout the study period.

The study was conducted at a single centre, which may limit its external validity. The patient population was predominantly ASA I, potentially restricting the applicability of the findings to high-risk patients. The variety of surgical procedures, while enhancing the scope of the study, may have introduced some variability in the results. The study did not include a control group receiving no intervention.

Future research should include multi-centre trials to improve external validity and broader applicability. Studies should be conducted on high-risk patients (ASA III and IV) to evaluate the safety and efficacy of these combinations in more complex cases. Research focusing on specific surgical populations could help optimize drug combinations for particular procedures. Developing standardized protocols for different patient populations and surgical settings would also be beneficial.

Conflict of Interest: None declared.

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