Section: Anaesthesia

Original Research Article

# COMPARATIVE EVALUATION OF DEXMEDETOMIDINE AND MAGNESIUM SULPHATE IN ATTENUATION OF PRESSOR RESPONSE TO VIDEO LARYNGOSCOPY AND INTUBATION UNDER GENERAL ANAESTHESIA IN ADULT PATIENTS

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***Received*** *: 05/06/2025*
***Received in revised form*** *: 20/07/2025*
***Accepted*** *: 11/08/2025*

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**DOI:** 10.70034/ijmedph.2025.4.29

**Source of Support:** Nil,
**Conflict of Interest:** None declared

**Int J Med Pub Health**
2025; 15 (4); 153-164

ABSTRACT

**Background:** The aim of this study was to compare intravenous Magnesium sulphate and Dexmedetomidine for attenuation of pressor response to video laryngoscopy and intubation under general anaesthesia.

**Materials and Methods:** The present study was a double blinded, prospective, randomized study conducted in Government general hospital, Kadapa during the period 2023-24. After obtaining institutional ethical committee approval and informed consent, 60 ASA I and II subjects in the age group of 20-60 years planned for elective surgeries were enrolled in this study. They were randomly allocated to one of the two study groups by using computer generated random numbers, Group D (Dexmedetomidine group) and Group M (Magnesium sulphate group).

**Results:** The study aimed to determine the effectiveness of dexmedetomidine (1 µg/kg) and magnesium sulphate (30 mg/kg) in reducing video laryngoscopy-induced hemodynamic responses when using the King Vision device for subsequent endotracheal intubation. In the present study, both groups showed equivalent demographic profiles during baseline measurements since they had no substantial differences in age, sex, body weight, Mallampati classification and ASA physical status. The agents proved effective in reducing the hemodynamic changes that occur when using video laryngoscopy with the King Vision device before endotracheal intubation. Dexmedetomidine proved to control heart rate and blood pressure better than magnesium sulphate did after endotracheal tube intubation. Heart rate decreased by 17% below the baseline after dexmedetomidine administration while magnesium sulphate maintained a decrease of only 5.5%. The research data demonstrated significant differences between groups during both the intubation process and next three-minute period ($p < 0.001$). Dexmedetomidine showed better blood pressure control of systolic and diastolic pressure and mean arterial pressure ($p < 0.05$ across all parameters) which confirmed its strong sympatholytic properties in this context. Treatment with both agents revealed good results regarding safety conditions. Among the group receiving dexmedetomidine, 2 patients (6.7%) developed bradycardia while no such occurrences were observed in the patients treated with magnesium sulphate ($p = 0.492$). This difference was not statistically significant. Dexmedetomidine produced significantly higher sedation (RSS 2.80 ± 0.48) than Magnesium Sulphate (RSS 2.07 ± 0.69; $p < 0.001$).

**Conclusion:** The randomized controlled trial evidence shows dexmedetomidine together with magnesium sulphate provides efficient stabilization of the hemodynamic response occurring during video laryngoscopy and endotracheal

intubation procedures. Dexmedetomidine controls heart rate and blood pressure better than magnesium sulphate does initially after intubation yet magnesium provides gentle stabilization benefits.
**Keywords:** Dexmedetomidine, Laryngoscopy, Endotracheal intubation, Magnesium sulphate, Hemodynamic parameters.

## INTRODUCTION

Laryngoscopy and endotracheal intubation is considered as standard of care in general anaesthesia and critical care since their introduction in 1921.[1,2] However laryngoscopy and intubation is associated with haemodynamic stress response in form of laryngo-sympathetic stimulation which can manifest as hypertension, tachycardia and arrhythmias.2 The intensity of this response can be influenced by factors such as depth of anaesthesia, duration, complexity of laryngoscopy and intubation, patient's characteristics like diabetes and cardiovascular disease.[3] Hence, the need to attenuate haemodynamic response to laryngoscopy and intubation is of paramount importance particularly in high risk American Society of Anaesthesiologists physical status III,IV patients undergoing general anaesthesia for various surgical procedures. The primary challenge in direct laryngoscopy with a Macintosh laryngoscope is the visual limitation inherent to the procedure, which requires a straight line of sight to view the glottis.3Haemodynamic response to tracheal intubation is effect of oropharyngeal stimulation produced by laryngoscopy and laryngotracheal stimulation secondary to tube insertion.[4,5] The magnitude of haemodynamic response increases with the force and duration of laryngoscopy. Tracheal intubation approaches that minimize oropharyngolaryngeal stimulation may attenuate this haemodynamic response. Video laryngoscopes do not require alignment of the oral, pharyngeal, and laryngeal axes for visualization of the glottis and tracheal intubation and cause minimal oropharyngolaryngeal stimulation and may hence potentially attenuate the pressor response.[6] The King vision video laryngoscope (KVVL), introduced in 2001, is one such device that utilizes digital technology to provide a clearer, indirect view of the airway.[6] Previous research indicates that video laryngoscopes generally improve intubation outcomes by enhancing visualization and reducing the force needed f or successful intubation, thereby minimizing tissue injury.[1,6] Pharmacological intervention for attenuation of pressor response to intubation includes administration of drugs such as alpha 2 adrenergic agonists (Clonidine and Dexmedetomidine), beta blockers (Metoprolol and Esmolol), calcium channel blockers, vasodilators such as Nitroglycerin (NTG) or Sodium nitroprusside (SNP).[5,7,8] Dexmedetomidine is widely used for attenuation of haemodynamic pressor response to intubation and it produces sedation, analgesia, anxiolysis and improved haemodynamic stability. However, it is not without side effects such as bradycardia and hypotension.[8] Magnesium sulphate, an NMDA receptor antagonist blocks release of catecholamines from adrenergic nerve terminals. Increased Magnesium levels can also inhibit the release of catecholamines. Magnesium also causes vasodilation by acting directly on blood vessels and in high doses, it attenuates vasopressin-mediated vasoconstriction.[8] Few studies have evaluated Magnesium sulphate for attenuation of stress response and the results are promising. The present study was undertaken to compare the effectiveness of intravenous Magnesium sulphate and Dexmedetomidine for attenuation of haemodynamic stress response to video laryngoscopy and endotracheal intubation.

**Aims and Objectives of Study**

**Aim of the Study:** The aim of this study was to compare intravenous Magnesium sulphate and Dexmedetomidine for attenuation of pressor response to video laryngoscopy and intubation under general anaesthesia.

**OBJECTIVES OF THE STUDY**

**Primary Objective:** To compare haemodynamics (heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure) at specific time intervals during video laryngoscopy and orotracheal intubation between two groups.

**Secondary objective:**

- To compare sedation score between the two groups
- To compare adverse effects like bradycardia and hypotension between two groups.

## MATERIALS AND METHODS

The present study **"comparative evaluation of Dexmedetomidine and Magnesium sulphate in attenuation of pressor response to video laryngoscopy and intubation under general anaesthesia in adult patients."** was a double blinded, prospective, randomized study conducted in Government general hospital, Kadapa during the period 2023-24. After obtaining institutional ethical committee approval and informed consent, 60 ASA I and II subjects in the age group of 20-60 years planned for elective surgeries were enrolled in this study. They were randomly allocated to one of the two study groups by using computer generated random numbers, Group D (Dexmedetomidine group) and Group M (Magnesium sulphate group).

**Statistical test of significance:** Comparison of quantitative parameters was done using student's unpaired t test and categorical data was compared by using Chi-square test. P Value <0.05 was considered as statistically significant.

**Inclusion Criteria**

- Patients of 20-60 years age group of either sex
- ASA physical status I and II
- Patients with BMI <25kg/m2
- Mallampati grade I and II
- Patients undergoing elective surgical procedures under general anaesthesia.

**Exclusion Criteria**

- Patient refusal
- Patients <20 years and >60 years of age
- Heart rate<60/min
- Systolic blood pressure <100 mmofhg
- Mallampati Grading III and IV
- Total duration of video laryngoscopy more than 30 seconds
- ASA grade III or IV patients
- Patients with systemic disorders like left ventricular failure, any degree of heart block, ischemic heart disease, aortic stenosis and bronchial asthma.

**Pre-anaesthetic evaluation:** Preoperative evaluation was done a day prior to surgery. Patient's detailed history, general physical examination, and systemic examination were performed. Airway assessment was done using Modified Mallampati classification.

**Modified mallampati classification:** Grade I: The palatal arch, including the bilateral faucial pillars and bases of the pillars.

Grade II: The upper part of the pillars and the uvula are visible.

Grade III: Only the soft and hard palates are visible.

Grade IV: Only the hard palate is visible.

Routine investigations like haemoglobin, blood grouping and typing, bleeding time and clotting time, blood sugar, blood urea, serum creatinine, chest X-ray and ECG were done in all patients. Demographic characters like age, sex, height, and weight were recorded, and written informed consent was obtained.

**Premedication:** All the patients received Tab. Alprazolam 0.5 mg orally on the night before surgery. All patients were kept 6 hours nil per oral. Patients were premedicated with intravenous Ranitidine 1 mg/kg IV in preoperative room 60 minutes before surgery.

**In the Operating room:** The patients were shifted to the operative room after checking for informed consent and nil per oral status.

- The patients were connected to ASA standard monitors – Non-invasive blood pressure (NIBP), ECG, Pulse oximeter (SpO2). Baseline vital parameters - blood pressure, heart rate, and SpO2 were noted.
- 18G Intravenous line was secured.

**Group M**: Patients were administered 30mg/kg of 50% Magnesium sulphate in 100 ml of normal saline over a period of 15 min.

**Group D**: Patients received intravenous Dexmedetomidine 1mcg /Kg in 100 ml of normal saline over a period of 15 min.

After a period of 10 minutes vital parameters were recorded in both the groups and

- Patients were pre oxygenated with 100% Oxygen for 3 minutes.
- All patients were premedicated with Glycopyrrolate 4mcg/kg I.V, Midazolam 0.03mg/kg I.V, Ondansetron 0.08mg/kg I.V and Fentanyl 2mcg/kg I.V.
- Patients were induced with Propofol 1.5 - 2mg/kg I.V.
- After ensuring adequate mask ventilation, patients were paralyzed with Succinylcholine 1.5 mg/kg I.V.
- Patients were placed in sniffing position and laryngoscopy was performed with Kingvision video laryngoscope with channelled blade. Laryngoscopy was performed by an experienced anaesthesiologist (Minimum 25 intubations with Kingvision video laryngoscope were performed by the intubating anaesthesiologist before starting the study).
- Kingvision video laryngoscope was inserted from the middle of oral cavity, reaching up to glossoepiglottic fold, then, the blade was lifted gently for visualization of glottis. The preloaded appropriate sized ET tube was advanced into the glottis following which it was slided out of the channel. After passing the tube, cuff was inflated with air. Intubation was confirmed with capnography and bilateral equal air entry.
- The endotracheal tube was secured, anaesthesia was maintained with Oxygen, Nitrous oxide (40:60) and Sevoflurane.
- Heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were noted at baseline, 10 min after drug administration, after induction, during intubation and 1, 3, 5 and 10 minutes after intubation. At the end of the surgery patients were reversed with neostigmine 0.05mg/kg and glycopyrrolate 0.08 mg/kg.

**Assessment of parameters:** The parameters like heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were recorded at the following point of time**.**

- After shifting the patient to OT. (Baseline value)
- 10 minutes after completion of study drug administration
- during intubation (t0)
- 1 min after intubation (t1)
- 3 min after intubation (t3)
- 5 min after intubation (t5)
- 10 min after intubation (t10)

10 min after completion of infusion of study drug, sedation score was assessed using Modified Ramsay Sedation Score.

**Adverse effects**

Any adverse effects like bradycardia, hypotension, and delayed neuromuscular recovery were recorded in both the groups. Hypotension was said to have occurred if systolic blood pressure fell below 100 mm of hg or if diastolic blood pressure fell below 50 mm of hg or if the mean arterial blood pressure fell below 65 mm of hg. Patient was treated with 100% O2,

increasing the infusion rate of intravenous fluids and Inj. Ephedrine in incremental dose of 3mg given at interval of 2 minutes.

Bradycardia was defined if heart rate was less than 60/min and was treated with intravenous atropine 0.6mg.

## RESULTS

**Table 1: Comparison of Age between Group M (Magnesium Sulphate) and Group D (Dexmedetomidine)**

| Group | N | MeanAge (years) | Standard Deviation (SD) | p-value |
|---|---|---|---|---|
| GroupM (Magnesium Sulphate) | 30 | 37.43 | 11.64 | |
| GroupD (Dexmedetomidine) | 30 | 40.00 | 13.57 | 0.435(Not significant) |

An independent samples t-test was conducted to compare the age distribution between participants in Group M and Group D. The mean age of participants in Group M was 37.43 years (SD = 11.64), while the mean age in Group D was 40.00 years (SD = 13.57). The independent samples t-test revealed that the difference in mean age between the two groups was not statistically significant ($t = -0.786$, $df = 58$, $p = 0.435$). The mean difference was -2.57 years, with a 95% confidence interval ranging from -9.10 to 3.97, suggesting that any observed difference in age could reasonably be due to chance.

**Table 2: Comparison of Sex Distribution between Group M (Magnesium Sulfate) and Group D (Dexmedetomidine)**

| Sex | Group D (Dexmedetomidine) | Group M (Magnesium Sulfate) | Total |
|---|---|---|---|
| Female (F) | 18 | 19 | 37 |
| Male (M) | 12 | 11 | 23 |
| Total | 30 | 30 | 60 |
| Pearson Chi-square: p value= 0.791(Not significant) | | | |

A Chi-square test of independence was conducted to examine the association between sex distribution and treatment group. Group D (Dexmedetomidine): 18 females and 12 males

- Group M (Magnesium sulfate): 19 females and 11 males.

The Pearson Chi-square test revealed no statistically significant association between sex and study group, $\chi^2$ (1) = 0.071, p = 0.791.

**Table 3: Comparison of Body Weight between Group M (Magnesium Sulfate) and Group D (Dexmedetomidine)**

| Group | N | Mean Weight (kg) | Standard Deviation (SD) | p-value (independent samples t-test ) |
|---|---|---|---|---|
| GroupM (Magnesium Sulfate) | 30 | 53.10 | 7.89 | 0.849 (Not significant) |
| GroupD (Dexmedetomidine) | 30 | 52.73 | 6.90 | |

An independent samples t-test was performed to compare the mean body weight between patients in the two study groups.

The mean weight of participants in Group M was 53.10 kg (SD = 7.89), and in Group D it was 52.73 kg (SD = 6.90). The independent samples t-test showed that the difference in mean weight between the two groups was not statistically significant ($t = 0.192$, $df = 58$, $p = 0.849$)

**Table 4: Comparison of Modified Mallampati grading Distribution between Group M (Magnesium Sulfate) and Group D (Dexmedetomidine)**

| Mallampati Score | GroupD (Dexmedetomidine) | Group M (Magnesium Sulfate) | Total |
|---|---|---|---|
| Grade I | 15 | 19 | 34 |
| Grade II | 15 | 11 | 26 |
| Total | 30 | 30 | 60 |
| Pearson Chi-square: p value= 0.297 (Not significant) | | | |

A Chi-square test of independence was conducted to examine the distribution of Mallampati Scores between the two groups:

- Group D (Dexmedetomidine): 15 patients were classified as Mallampati Class I, and 15 as Class II.
- Group M (Magnesium Sulfate): 19 patients were Class I, and 11 were Class II.

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The Pearson Chi-square test showed no statistically significant difference in Mallampati classification between the two groups, $\chi^2$ (1) = 1.086, p = 0.297. This suggests that airway classification, as assessed by the Mallampati score, was comparable across groups, indicating similar baseline airway anatomical characteristics before intervention

**Table 5: Comparison of ASA Physical Status between Group M (Magnesium Sulfate) and Group D (Dexmedetomidine)**

| ASA Class | Group D (Dexmedetomidine) | Group M (Magnesium Sulfate) | Total |
|---|---|---|---|
| ASA I | 13 | 14 | 27 |
| ASA II | 17 | 16 | 33 |
| Total | 30 | 30 | 60 |
| Pearson Chi-square: p value= 0.795 (Not significant) | | | |

A Chi-square test of independence was used to evaluate the association between the ASA physical status classification and study group.

- Group D (Dexmedetomidine): 13 patients were ASA I and 17 were ASA II.
- Group M (Magnesium Sulfate): 14 patients were ASA I and 16 were ASA II.

The Pearson Chi-square test result was $\chi^2$ (1) = 0.067, with a p-value of 0.795, indicating no statistically significant difference in ASA classification between the groups.

This suggests that both groups were comparable in terms of baseline systemic health status, ensuring that ASA classification did not act as a confounding factor in the study.

**Table 6: Comparison of Heart Rate between Group M and Group D at Various Time Intervals**

| Time Point | Group M (Mean ± SD) | Group D (Mean ± SD) | 95% CI (Lower– Upper) | *p*- value |
|---|---|---|---|---|
| Baseline HR | 78.23 ± 4.48 | 78.00 ± 5.75 | -2.43 to 2.90 | 0.861 |
| 10 min After Drug infusion HR | 73.90 ± 3.28 | 64.90 ± 3.75 | 7.18 to 10.82 | <0.001 |
| 0 Min HR | 84.07 ± 3.55 | 76.63 ± 6.58 | 4.68 to 10.19 | <0.001 |
| 1 Min HR | 79.10 ± 4.02 | 74.70 ± 7.03 | 1.43 to 7.36 | 0.004 |
| 3 Min HR | 76.20 ± 4.14 | 72.53 ± 5.64 | 1.11 to 6.22 | 0.006 |
| 5 Min HR | 71.73 ± 5.95 | 69.50 ± 6.91 | -1.10 to 5.57 | 0.185 |
| 10 Min HR | 68.97 ± 5.87 | 66.17 ± 8.36 | -0.93 to 6.53 | 0.139 |
| Independent sample t test: p value ≤ 0.05 (Significant) | | | | |

This table summarizes the heart rate measurements between two groups (M and D) at different time intervals. Statistically significant differences ($p < 0.05$) were observed at all-time points from "10 min after Drug administration HR" to "3 Min HR", in Group D post-intervention. No significant difference was observed at baseline, 5 minutes, and 10 minutes after administration, suggesting convergence of heart rate responses between groups over time.

**Table 7: Correlation of DNI with Other Parameters of Pancreatitis in Total Population**

| Time Point | Group M (Mean ± SD) | Group D (Mean ± SD) | 95% CI (Lower– Upper) | *p*- value |
|---|---|---|---|---|
| Baseline SBP | 123.60 ± 8.18 | 125.73 ± 10.44 | -6.98 to 2.71 | 0.382 |
| After Drug SBP | 119.73 ± 7.42 | 115.90 ± 7.60 | -0.05 to 7.71 | 0.050 |
| 0 Min SBP | 132.73 ± 8.65 | 127.73 ± 8.22 | 0.64 to 9.36 | 0.025 |
| 1 Min SBP | 126.27 ± 9.52 | 121.83 ± 7.16 | 0.08 to 8.79 | 0.046 |
| 3 Min SBP | 122.43 ± 3.15 | 120.13 ± 3.82 | 0.49 to 4.11 | 0.014 |
| 5 Min SBP | 114.33 ± 8.76 | 113.27 ± 7.62 | -3.18 to 5.31 | 0.617 |
| 10 Min SBP | 112.80 ± 6.23 | 110.40 ± 5.37 | -0.60 to 5.41 | 0.115 |
| Independent sample t test: p value ≤ 0.05 (Significant) | | | | |

The independent sample t-test was used to compare systolic blood pressure (SBP) between Group M and Group D at various time intervals. At baseline, there was no statistically significant difference in SBP between the two groups ($p = 0.382$), indicating comparable initial values. After drug administration, SBP was lower in Group D compared to Group M at 0 min at 0 minutes ($p = 0.025$), 1 minute ($p = 0.046$), and 3 minutes ($p = 0.014$), suggesting a more pronounced and immediate blunting of pressor response to intubation. However, at 5 minutes ($p = 0.617$) and 10 minutes ($p = 0.115$), the differences were not statistically significant, indicating a diminishing effect over time. Overall, the findings demonstrate that the intervention in Group D resulted in a significantly greater reduction in systolic blood pressure during intubation compared to Group M

**Table 8: Comparison of Diastolic Blood Pressure (DBP) Between Group M and Group D**

| Time Point | Group M (Mean ± SD) | Group D (Mean ± SD) | 95% CI (Lower– Upper) | p- value |
|---|---|---|---|---|
| Baseline DBP | 75.80 ± 5.37 | 78.47 ± 6.38 | -5.71 to 0.38 | 0.085 |
| After Drug DBP | 74.60 ± 4.56 | 71.73 ± 5.22 | 0.33 to 5.40 | 0.027 |
| 0 Min DBP | 80.87 ± 1.87 | 78.97 ± 3.49 | 0.45 to 3.35 | 0.011 |
| 1 Min DBP | 79.10 ± 6.83 | 75.67 ± 6.13 | 0.08 to 6.79 | 0.045 |

| | | | | |
|---|---|---|---|---|
| 3 Min DBP | 75.57 ± 4.13 | 72.00 ± 6.06 | 0.89 to 6.25 | 0.010 |
| 5 Min DBP | 71.00 ± 5.17 | 69.00 ± 5.53 | -0.77 to 4.77 | 0.153 |
| 10 Min DBP | 68.80 ± 4.77 | 67.20 ± 5.67 | -1.11 to 4.31 | 0.242 |
| Independent sample t test: p value ≤ 0.05 (Significant) | | | | |

An independent sample t-test was used to compare diastolic blood pressure (DBP) between Group M and Group D across multiple time points. The baseline DBP was not significantly different between the groups (p = 0.085). After drug administration, Group D showed a significantly lower DBP compared to Group M (p = 0.027), indicating a notable immediate response. This difference remained significant at 0, 1, and 3 minutes post-intervention (p = 0.011, 0.045, and 0.010 respectively), suggesting that Group D experienced a greater and more sustained reduction in DBP. However, by 5 and 10 minutes, the difference between the groups was no longer statistically significant (p = 0.153 and 0.242), implying a diminishing effect over time. Overall, Group D demonstrated a faster and more pronounced reduction in diastolic pressure shortly after intervention

**Table 9: Mean Arterial Pressure (MAP) Comparison between Group M and Group D at Different Time Points**

| Time Point | Group M (Mean ± SD) | Group D (Mean ± SD) | 95% CI (Lower– Upper) | p- value |
|---|---|---|---|---|
| Baseline MAP | 90.87 ± 5.61 | 93.80 ± 7.23 | -6.28 to 0.41 | 0.084 |
| After Drug MAP | 89.57 ± 4.46 | 86.33 ± 4.54 | 0.91 to 5.56 | 0.007 |
| 0 Min MAP | 97.60 ± 1.94 | 95.67 ± 4.23 | 0.23 to 3.63 | 0.027 |
| 1 Min MAP | 93.40 ± 6.31 | 90.43 ± 4.57 | 0.12 to 5.81 | 0.041 |
| 3 Min MAP | 89.60 ± 4.95 | 87.00 ± 3.99 | 0.28 to 4.92 | 0.029 |
| 5 Min MAP | 85.47 ± 4.64 | 83.70 ± 4.71 | -0.65 to 4.18 | 0.149 |
| 10 Min MAP | 83.43 ± 3.55 | 81.53 ± 4.26 | -0.13 to 3.93 | 0.065 |
| Independent sample t test: p value ≤ 0.05 (Significant) | | | | |

The intergroup comparison of Mean Arterial Pressure (MAP) revealed no statistically significant difference at baseline (p = 0.084). However, following drug administration and during the early intraoperative period (0, 1, and 3 minutes), Group D demonstrated significantly lower MAP values compared to Group D (p = 0.007, 0.027, 0.041, and 0.029 respectively), suggesting better attenuation of pressor response to intubation. At 5 and 10 minutes, MAP was comparable in both the groups (p = 0.149 and 0.065 respectively).

**Table 10: Comparison of Incidence of Adverse effects Between Group D and Group M**

| Group | Bradycardia | NIL | Total |
|---|---|---|---|
| D | 2 | 28 | 30 |
| M | 0 | 30 | 30 |
| Total | 2 | 58 | 60 |
| Pearson Chi-Square: p-value=0.492(Not significant) | | | |

The Chi-Square test results indicate no significant association between the group (D vs. M) and adverse effects. The Pearson Chi-Square value of 2.069 with a p-value of0.150 suggests that the difference in the occurrence of Bradycardia between the two groups is not statistically significant.

**Table 11: Comparison of Ramsay Sedation Scores between Magnesium Sulphate and Dexmedetomidine Groups**

| Group | Mean RSS ± SD | 95% CI for Difference | p-value |
|---|---|---|---|
| Magnesium Sulphate (M) | 2.07 ± 0.69 | 0.425 to 1.042 | <0.001 |
| Dexmedetomidine (D) | 2.80 ± 0.48 | | |
| Independent sample t test: p value ≤ 0.05 (Significant) | | | |

The mean RSS in the Group M was 2.07 ± 0.69, whereas in the Group D, it was significantly higher at 2.80 ± 0.48. The mean difference in sedation scores between the groups was 0.733, with a 95% confidence interval ranging from 0.425 to 1.042.

An independent samples t-test revealed that this difference was statistically significant (p < 0.001), indicating that Group D produced significantly greater sedation compared to Group M. The assumption of equal variances was confirmed using Levene's test (p= 0.129), and thus, equal variance was assumed for the t-test.

These findings suggest that Group D is more effective in achieving higher sedation levels, as measured by RSS, in the studied population

## DISCUSSION

Laryngoscopy and endotracheal intubation cause sympathoadrenal activation.[1,2] and this often results in marked cardiovascular instability—manifesting as elevated heart rate, arterial hypertension, and potential arrhythmias. Such hemodynamic responses can be especially perilous in individuals with underlying cardiovascular comorbidities, as they may precipitate acute ischemic events or cerebrovascular accidents.[3] In this prospective, randomized investigation, we sought to assess and compare the effectiveness of two agents—dexmedetomidine and magnesium sulphate—in attenuating the hemodynamic surges linked with video laryngoscopy and intubation. The outcomes contribute meaningful evidence regarding the cardiovascular modulation offered by these drugs during the peri-intubation window, carrying important clinical relevance for anaesthetic practice and patient safety.

**Demographic and Baseline Characteristics Age Distribution (Table 1):** The mean age was 37.43 ± 11.64 years in Group M and 40.00 ± 13.57 years in Group D, showing no statistically significant difference (p = 0.435), which ensures comparability for age-dependent cardiovascular reactivity. These findings are consistent with similar studies by Panda et al.[7] and Sharma and Mehta,[8] while Kumaret al.[9] studied an older population that demonstrated heightened hemodynamic variability.

**Gender Distribution (Table 2):** he study included 37 females (61.7%) and 23 males (38.3%), with even distribution between groups: Group D had 18 females and 12 males, while Group M consisted of 19 females and 11 males (p = 0.791). This balanced gender representation differs from studies by Singh ET al.[10] and Mahendru et al.[11] who reported male predominance, and helps mitigate sex-based confounding given that Li et al.[13] demonstrated sex-related differences in hemodynamic responses during laryngoscopy.

**Body Weight (Table 3):** Mean body weight was comparable between groups at 53.10 ± 7.89 kg in Group M and 52.73 ± 6.90 kg in Group D (p = 0.849). This uniformity is important for weight- dependent pharmacological agents and facilitates accurate dosing. The weights were notably lower than Western populations studied by Pipanmekaporn et al.[14] (64.8-66.3 kg) but consistent with South Asian studies like Gupta et al.[15] (54.2-55.7 kg), with narrow standard deviations minimizing weight-related confounders.

**Mallampati Score Distribution (Table 4):** Mallampati classification, an established predictor of airway difficulty, showed a balanced distribution across the two groups in our study, with no statistically significant difference noted (p = 0.297). MMPG I was most prevalent comprising 50% of Group D and 63.3% of Group M. This uniformity in airway anatomy is crucial when assessing cardiovascular perturbations during laryngoscopy, as more complex airway management typically elicits a heightened sympathoadrenal response due to prolonged or forceful instrumentation.[16] In contrast, Rajput et al. reported a higher representation of Class II airways (58% and 62%) in their patient groups, suggesting a shift in airway profile potentially influenced by geographic, anatomical, or sampling differences.[17] These inter-study variations underscore the importance of accounting for regional and methodological diversity when comparing hemodynamic outcomes associated with airway interventions. The predominance of favourable Mallampati grades (Class I and II) in our cohort likely contributed to a standardized intubation environment, minimizing procedural variability and thereby enhancing the reliability of the cardiovascular data obtained. By restricting the study population to patients with anticipated easy airways, we effectively controlled for a major confounding variable—airway difficulty—which is well-documented to intensify cardiovascular responses during intubation. However, while this methodological rigor strengthens the internal validity of our hemodynamic comparisons, it concurrently narrows the scope of generalizability. Patients with Mallampati Class III and IV, who may exhibit exaggerated or prolonged pressor responses, were not represented in our sample. Future research should aim to explore these dynamics in more heterogeneous airway populations to broaden clinical applicability.

**ASA Physical Status (Table 5):** In the present study, 27 participants (45%) were categorized as ASA Physical Status Class I, while the remaining 33 (55%) were classified as ASA Class II. The distribution between the two intervention groups was statistically comparable (p = 0.795), indicating successful randomization and balanced baseline health status. This mix of healthy individuals and those with mild systemic conditions enhances the clinical generalizability of the observed hemodynamic outcomes.

Our findings closely mirror those of Panda et al., who reported a similar distribution of ASA I (48%) and ASA II (52%) patients in their cohort.[7] Sharma et al. also documented nearly identical proportions—47% ASA I and 53% ASA II.[8] Such alignment across studies supports the external validity of our results and allows for more meaningful inter-study comparisons in the context of airway manipulation and anaesthetic pharmacodynamics. In contrast, Mahendru et al. reported a cohort more heavily weighted toward ASA I classification (67%), potentially contributing to the more stable hemodynamic trends observed in their study population.[11] Their reduced inclusion of patients with systemic comorbidities may account for diminished cardiovascular reactivity under procedural stress. By incorporating a substantial proportion of ASA II patients, our study reflects a broader spectrum of real-world clinical scenarios. This subgroup often encompasses individuals with controlled hypertension, type 2 diabetes, or early-

stage cardiovascular pathology—conditions known to intensify the pressor response to laryngoscopy and endotracheal intubation. Thus, the near-equal representation of ASA classifications between groups not only supports the robustness of randomization but also minimizes confounding effects related to pre-existing systemic health status.

**Heart Rate Variations (Table 6):** Analysis of heart rate (HR) dynamics at multiple peri-intubation time points revealed statistically significant intergroup differences between the dexmedetomidine (Group D) and magnesium sulphate (Group M) cohorts. At baseline, both groups exhibited similar mean HR values—78.23 ± 4.48 bpm for Group M and 78.00 ± 5.75 bpm for Group D ($p = 0.861$)—indicating well-balanced pre-intervention cardiovascular status.

Following administration of the respective agents, a marked decline in heart rate was observed in the dexmedetomidine group, with a mean HR of 64.90 ± 3.75 bpm. In contrast, Group M demonstrated a more modest reduction to 73.90 ± 3.28 bpm. The intergroup difference at this juncture was highly significant ($p < 0.001$). This pronounced negative chronotropic effect of dexmedetomidine is in line with its pharmacodynamic action as a potent central α2-adrenergic agonist, which inhibits sympathetic tone and enhances parasympathetic modulation via vagal stimulation.[18] The approximate 17% HR reduction from baseline observed in our dexmedetomidine cohort is congruent with prior findings by Reddy et al. (19% reduction),[19] and exceeds the 14% reduction documented by Singh et al.[10] Magnesium sulphate, by contrast, produced a milder HR decline—approximately 5.5% from baseline—consistent with its mechanism of action. Rather than direct chronotropic suppression, its cardiovascular effects are primarily mediated through calcium antagonism and peripheral vasodilation.[20] These differing hemodynamic profiles reflect the distinct pharmacological pathways of the agents under study and bear clinical significance when tailoring anaesthesia to individual cardiovascular profiles. During laryngoscopy and intubation (0 minutes), both groups demonstrated increase in HR, consistent with the expected surge in catecholamine release following airway stimulation. However, the rise was significantly attenuated in Group D (76.63 ± 6.58 bpm) compared to Group M (84.07 ± 3.55 bpm), with a p-value of < 0.001. This attenuation highlights dexmedetomidine's superior efficacy in suppressing the acute tachycardic response, corroborating the findings of Srivastava et al., who documented a 17% HR reduction during laryngoscopy in the dexmedetomidine cohort relative to controls.[21] Sustained intergroup differences in HR persisted at 1 minute ($p = 0.004$) and 3 minutes ($p = 0.006$) post-intubation, reflecting continued sympatholytic activity of dexmedetomidine during the early post-laryngoscopy window. By 5-minute ($p = 0.185$) and 10-minute ($p = 0.139$) marks, however, heart rate values between groups had largely converged, indicating the waning of initial pharmacological divergence and suggesting eventual haemodynamic equilibration. This temporal trajectory parallels the observations of Soliman et al., who also noted that dexmedetomidine's differential impact on HR persisted only up to 5 minutes following intubation.[22] It is worth noting that our results diverge from those reported by Kumar et al., who found no significant intergroup HR differences in the immediate post-intubation phase.[9] This discrepancy may be attributable to methodological variation—specifically, their use of conventional laryngoscopy versus our utilization of the King Vision video laryngoscope. Video-assisted techniques generally require less direct manipulation of supraglottic tissues, resulting in diminished adrenergic stimulation and more controlled cardiovascular responses.[23] The reduced mechanical stress inherent in video laryngoscopy may amplify the observable hemodynamic distinctions between agents such as dexmedetomidine and magnesium sulphate. Ultimately, while dexmedetomidine exhibited a superior capacity to blunt tachycardic responses in the immediate post-intubation period, both agents facilitated eventual cardiovascular stabilization. This observation is especially pertinent in the context of short-duration surgeries, wherein the critical window of hemodynamic vulnerability is brief yet clinically significant.

**Systolic Blood Pressure Dynamics (Table 7):** Initial assessment of systolic blood pressure (SBP) revealed no significant difference between the two cohorts, with baseline values recorded at 123.60 ± 8.18 mmHg for Group M and 125.73 ± 10.44 mmHg for Group D ($p = 0.382$), indicating comparable hemodynamic status prior to drug administration. Upon intervention, a decline in SBP was observed across both groups; however, the reduction was more notable in Group D, which reached 115.90 ± 7.60 mmHg, as opposed to 119.73 ± 7.42 mmHg in Group M. Although this difference approached the threshold of statistical significance ($p = 0.050$), it underscores a trend toward greater efficacy of dexmedetomidine in reducing SBP. This observation corroborates the findings of Panda et al., who reported a more substantial pre-intubation SBP reduction with dexmedetomidine (18.5%) relative to magnesium sulphate (12.7%).[7] The mechanical basis for this disparity likely stems from dexmedetomidine's capacity to engage both central and peripheral α2-adrenoreceptors, eliciting sympatholysis and direct vasomotor modulation. In contrast, magnesium sulphate exerts its hypotensive effect predominantly via calcium channel inhibition and resultant peripheral vasodilation.[24] At the point of laryngoscopy and intubation (0 minutes), a physiological SBP surge— reflecting the acute sympathetic response to airway manipulation—was evident in both groups. However, this pressor response was significantly blunted in Group D (127.73± 8.22 mmHg) when compared to Group M (132.73 ± 8.65 mmHg), achieving statistical significance ($p = 0.025$). These results are consistent

with the meta-analysis by Li et al., which highlighted the superior efficacy of dexmedetomidine in suppressing pressor responses to intubation over other agents, including magnesium sulphate.[25] The intergroup disparity in SBP persisted at 1 minute (p = 0.046) and 3 minutes (p = 0.014) following intubation, signifying a sustained modulatory effect of dexmedetomidine on hemodynamic responses during this critical period. This temporal pattern mirrors that reported by Sharma and Mehta, who demonstrated continued SBP divergence between the two agents for up to five minutes post- intubation.[8] By 5 minutes (p = 0.617) and 10 minutes (p = 0.115) after intubation, the SBP levels in both groups began to converge, implying a resolution of the acute adrenergic surge irrespective of the prophylactic agent employed. This convergence aligns with the known timeline for catecholamine metabolism and clearance, typically stabilizing within 5–10 minutes in the absence of further stimuli.[26] When juxtaposed with literature involving conventional laryngoscopy, the magnitude of SBP elevation observed in our study appears attenuated. For example, Rajput et al. documented SBP peaks reaching 15–20% above baseline despite prophylactic pharmacologic measures,[17] whereas the increases in our population were limited to approximately 7–9%. This difference likely reflects the hemodynamic advantages conferred by video laryngoscopy—specifically, the King Vision device—which enhances glottic exposure while minimizing mechanical stress on upper airway structures.[27] Our results are consistent with Abdelgawad et al., who also reported diminished hemodynamic perturbations during video-assisted intubation relative to direct laryngoscopy across diverse prophylactic protocols.[28] The superior SBP modulation offered by dexmedetomidine carries important clinical implications, particularly in patients with heightened cardiovascular vulnerability. In individuals with underlying hypertension, cerebrovascular pathologies, or aorticaneurysms—conditions where transient hypertensive spikes may precipitate adverse outcomes—the enhanced efficacy of dexmedetomidine may justify its selection, notwithstanding its higher cost and potential for distinct adverse effects.

**Diastolic Blood Pressure Patterns (Table 8):** Group D participants had slightly higher baseline diastolic blood pressure at 78.47 ±6.38 mmHg in comparison to group M (75.80 ± 5.37 mmHg) but this difference was not considered significant statistically (p = 0.085). A significant statistical difference appeared between Groups D and M after drug administration since Group D showed lower DBP values at 71.73 ± 5.22 mmHg when compared to Group M at 74.60 ± 4.56 mmHg with p = 0.027. The decline in DBP matches the SBP results, which demonstrates that dexmedetomidine provides stronger vasodilatory and sympatholytic effects. The stimulation of α2-adrenoceptors on vascular smooth muscle postsynaptically most likely explains dexmedetomidine's ability to promote vasodilation for reducing peripheral vascular resistance.[29]The modest DBP lowering action of magnesium sulphate follows its main mechanism of calcium channel antagonism since it lacks direct sympatholytic properties.[30] The anticipated stress-induced blood pressure increase following laryngoscopy and intubation procedure (0 minutes) occurred in both treatment groups. During intubation, the dexmedetomidine group demonstrated attenuation of the expected arterial pressure reaction while the participants who received magnesium sulphate maintained their hemodynamic response to intubation. IV dexmedetomidine produced a sustained modification of stress reaction to intubation procedures as demonstrated by significantlower blood pressure findings at 1 and 3 minutes post-intubation (p = 0.045 and p = 0.010 respectively). The results of this study match Panda et al.'s findings about lower DBP readings following intubation among dexmedetomidine users during the first five minutes.[7] Soliman et al. concluded that dexmedetomidine showed better DBP reducing effects than magnesium sulphate and lidocaine when they analyzed data.[22] By 5 minutes (p = 0.153) and 10 minutes (p = 0.242) following intubation, DBP levels in both groups converged, reflecting a shared trajectory toward hemodynamic stabilization. This trend parallels observations in SBP and heart rate (HR) profiles, suggesting that while dexmedetomidine offers a more robust early-phase modulation of sympathetic activity, both agents ultimately yield comparable post-intubation equilibrium. Notably, the magnitude of DBP elevation during laryngoscopy was modest in both groups—approximately 6.7% for Group M and only 0.6% for Group D—substantially lower than the values typically reported with conventional laryngoscope techniques. For instance, Vora et al. described DBP increases ranging from 12% to 15% despite the use of prophylactic measures during traditional laryngoscopy.[31] This discrepancy underscores the potential benefit of employing video laryngoscopy, such as the King Vision device, in reducing the mechanical and sympathetic stimuli associated with airway instrumentation. Beyond general hemodynamic stability, effective control of DBP bears substantial clinical relevance. Elevated diastolic pressure, particularly during the perioperative period, can contribute to increased myocardial oxygen demand, ventricular wall stress, and heightened intracranial pressure—all of which may exacerbate existingcardiovascular or neurologic conditions.[32] Therefore, the superior control of DBP observed with dexmedetomidine may be of particular importance in high-risk populations, including those with coronary artery disease, left ventricular hypertrophy, or elevated intracranial pressure.

**Mean Arterial Pressure Evaluation (Table 9):** Mean arterial pressure functions as the main indicator for monitoring global tissue perfusion together with

cardiovascular stability. The baseline MAP readings showed minimal variation as Group D reported 93.80 ± 7.23 mmHg while Group M recorded90.87 ± 5.61 mmHg and the difference remained statistically insignificant (p = 0.084). After infusion of the study drug, MAP decreased in both groups but Group D showed a profound reduction compared to Group M as their MAP readings were 86.33 ± 4.54 mmHg and 89.57 ± 4.46 mmHg respectively with p = 0.007. Dexmedetomidine administration led to a significant decrease in mean arterial pressure which supports previous reports regarding its sympatholytic characteristics as described by Sharma and Mehta.[8] A MAP elevation indicative of pressor response occurred for both groups at the moment of laryngoscopy and intubation (0 minutes). Group D participants experienced a modest in blood pressure increase after intubation with a mean arterial pressure measured 95.67 ± 4.23 mmHg Vs Group M participants who demonstrated 97.60 ±1.94 mmHg (p = 0.027). The stabilizing effect of dexmedetomidine maintained its influence on MAP during the first (p = 0.041) and third (p = 0.029) minutes after intubation due to its prolonged ability to suppress adrenergic responses during stressful procedures. The research by Zhang et al. through their meta-analysis demonstrated that dexmedetomidine provided superior management of MAP elevation following intubation compared to other pharmacological agents such as magnesium sulphate with an average 10.7 mmHg peak MAP reduction.[33] Research by Soliman et al. indicated dexmedetomidine minimized MAP elevations to a greater extent compared to the use of magnesium sulphate as an intervention during airway instrumentation.[22] The MAP levels of both groups converged at 5- and 10-minutes post-intubation (p = 0.149 and p = 0.065 respectively) but Group M showed consistently higher pressure values. Both experimental groups showed moderate MAP elevation during intubation which amounted to 7.4% in Group M versus 2.0% in Group D. Video laryngoscopy procedures produce lesser increases than what conventional direct laryngoscopy protocols do. When direct laryngoscopy was used, Kumar et al. noted an elevation of MAP up to 15–18% despite using prophylactic medications.[9] King Vision video laryngoscopy together with its specific design shows promise because it reduces sympathetic activation through minimal stimulation of oropharyngeal structures.[34] The benefits from controlling MAP effectively create essential clinical results particularly for surgical patients with elevated risks. Medical research has established that MAP fluctuations greater than 20% above baseline perioperatively correspond to major adverse postoperative health risks such as myocardial damage and acute kidney problems and cerebrovascular complications.[35]

**Adverse Effects Analysis (Table 10):** The safety profile was well maintained throughout the study in both treatment groups indicating dexmedetomidine and magnesium sulphate are safe when used in this situation. Bradycardia manifesting as heart rate falling below 50 beats per minute occurred in 2 dexmedetomidine-treated patients (6.7% of Group D) without any recorded cases in the magnesium sulphate-treated patients (Group M). The safety profile between dexmedetomidine and magnesium sulphate showed non-substantial differences since the statistical test yielded a p-value of 0.492. Two patients in Group D (6.7%) experienced bradycardia due to the pharmacological mechanism of dexmedetomidine which activates central α2-adrenergic receptors to enhance vagal tone and restrict sympathetic activity.[36] Similar such observations of bradycardia were noted by Reddy et al. reporting 8%.[19] and Singh et al. with 5%[10] and Sharma and Mehta showing 10%.[8] The bradycardia incidence we observed is lower than figures Panda et al. reported (16%).[7] along with those found in Mahendru et al. (12%).[11] The variations in drug administration protocols and infusions rates alongside different patient characteristics likely explain these discrepancies in bradycardia occurrence. The infusion duration for dexmedetomidine at 1 µg/kg differed between studies, Panda et al. using a 5-minute administration while our study used a 15-minute period which could explain the contrasting negative chronotropic effects. None of the patients in the study groups had hypotension. It is probable that our preoperative fluid loading with crystalloid at a rate of 10 ml/kg in combination with a slower infusion rate might have mitigated hypotensive effect of the study drugs. All patients in our cohort failed to report any adverse magnesium sulphate effects which normally manifest as facial flushing and warmth sensation as well as nausea and muscle weakness. The combination of low dose and controlled administration rate in this study led to beneficial results in mgso4 group. According to the study's findings, neither of the treatment groups experienced respiratory depression and delayed neuromuscular blockade. Respiratory safety of both drugs remains strong when healthcare providers use therapeutic dose levels. Tests conducted by Soliman et al.[22] and Kumar et al.[9] demonstrated similar conclusions about normal respiratory function at similar conditions. The lack of adverse events between both groups demonstrates that dexmedetomidine and magnesium sulphate can safely be used as prophylactic drugs to manage the hemodynamic response to laryngoscopy. The limited detection of delayed or uncommon complications is possible due to the study's small participant count of n = 60. Wider trials that monitor drug safety outcomes should follow in order to fully understand the benefits and risks associated with these pharmacologic treatment approaches.

**Ramsay Sedation Score Analysis (Table 11):** In the present study, Dexmedetomidine demonstrated significantly higher sedation levels compared to Magnesium Sulphate, as evidenced by the Ramsay SedationScore (RSS). The Dexmedetomidine group achieved a mean RSS of 2.80 ± 0.48, while the

Magnesium Sulphate group attained a lower mean RSS of 2.07 ± 0.69, with a statistically significant mean difference of 0.733 ($p < 0.001$) (Table 11). These findings are consistent with several previous studies that assessed the sedative efficacy of both agents in perioperative settings. In a comparative study by Abd El-Aal et al.[37] (2021) on patients undergoing awake fiberoptic intubation, a higher median RSS (3 [IQR: 2–4]) was recorded in the Dexmedetomidine group compared to 1 [IQR: 1–2] in the Magnesium Sulphate group, with the difference being highly significant ($p < 0.001$). These results support our observation that Dexmedetomidine provides deeper sedation, which is crucial for procedures requiring patient cooperation without airway compromise. Taken together, these findings affirm that Dexmedetomidine provides significantly superior sedation than Magnesium Sulphate, aligning with the pharmacological profile of Dexmedetomidine as a selective α2-adrenergic agonist capable of inducing sedation that mimics natural sleep. This is particularly advantageous in procedures requiring patient calmness without respiratory depression.

**Strengths and Limitations:** The study has multiple sound methodological attributes. Both methodological features of randomized control methods enhance internal validity together with the detailed measurements of hemodynamic parameters across different time points for understanding pharmacodynamics behaviour. Standardizing the anaesthesia protocol works to eliminate confusion affecting study results which makes the findings more repeatable. The implementation of video laryngoscopy with King Vision technology serves as a present-day advanced tool for airway management because it mirrors modern approaches in clinical anaesthetic work. The results about peri-intubation hemodynamic response do not reveal information about downstream morbidity or mortality consequences due to lacking long-term clinical outcome evaluations.

## CONCLUSION

The randomized controlled trial evidence shows dexmedetomidine together with magnesium sulphate provides efficient stabilization of the hemodynamic response occurring during video laryngoscopy and endotracheal intubation procedures. Dexmedetomidine controls heart rate and blood pressure better than magnesium sulphate does initially after intubation yet magnesium provides gentle stabilization benefits. The two drugs showed tolerability but dexmedetomidine demonstrated a tendency to slow heart rate which requires cautious choice of patients. Agent selection should be personalized to account for patient-specific heart conditions combined with anaesthetic targets and healthcare centre operational parameters. Further studies requiring bigger participant groups including larger patient populations and longer follow-ups need to validate these results while developing better drug treatment methods for high-risk surgical patients.

**Conflict of Interest:** None

**Funding Support:** Nil

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