DOI: 10.7860/JCDR/2026/82694.22244

Original Article

Anaesthesia Section

Comparison of the Two Doses (30 mg/kg versus 50 mg/kg) of Magnesium Sulphate in Attenuation of Haemodynamic Stress Response during Laryngoscopy and Tracheal Intubation: A Randomised Clinical Study

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ABSTRACT

Introduction: Laryngoscopy and endotracheal intubation are known to elicit significant sympathoadrenal responses, which can lead to haemodynamic instability. Magnesium sulphate has been shown to attenuate this stress response through various mechanisms.

Aim: To compare the efficacy of two different doses of magnesium sulphate (30 mg/kg vs 50 mg/kg) in attenuating the haemodynamic changes during laryngoscopy and tracheal intubation.

Materials and Methods: This randomised clinical study was conducted at the Department of Anaesthesiology, Shrimati Bhikhiben Kanjibhai Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed To Be University, Piparia, Vadodara, Gujarat, India, from January 2023 to June 2024. It included 60 American Society of Anaesthesiologists (ASA) physical status grade I and II patients aged 18-65 years scheduled for elective surgeries under general anaesthesia. Patients were randomly divided using computerised randomisation into two groups of 30 each: Group I received magnesium sulphate 30 mg/kg intravenoulsy (i.v.), and Group II received magnesium sulphate 50 mg/kg i.v. diluted in 50 mL of normal saline, administered over 10 minutes before induction. Haemodynamic parameters (heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure) were recorded at baseline, after drug administration, after induction, during laryngoscopy and intubation, and at 1, 3, 5, 7, and 10 minutes post-intubation. Statistical analysis was performed using unpaired Student's t-test and Chi-square test, with p-value<0.05 considered significant.

Results: The demographic characteristics (mean age: group I 38.9±7.5 years, group II 41.0±8.8 years; gender ratio M:F - group I 17:13, group II 18:12; mean weight: group I 66.3±6.3 kg, group II 65.9±5.7 kg) were comparable between groups. Heart rate showed significant differences between groups during laryngoscopy (group I: 94.0±5.1 vs group II: 89.4±5.2 beats/min, p-value<0.001) and at intubation (group I: 97.7±5.5 vs group II: 93.5±5.0 beats/min, p-value=0.003). Systolic blood pressure at intubation was 131.0±6.2 mmHg in group I versus 124.8±5.4 mmHg in group II (p-value<0.001). Diastolic blood pressure at intubation was 80.7±4.7 mmHg in group I versus 76.4±4.8 mmHg in group II (p-value<0.001). Mean arterial pressure at intubation was 97.4±4.7 mmHg in group I versus 92.5±4.3 mmHg in group II (p-value<0.001). Both magnesium sulphate groups showed significant attenuation of the haemodynamic stress response to laryngoscopy and intubation compared to baseline values. Group II (50 mg/kg) demonstrated greater attenuation of blood pressure responses than group I (30 mg/kg), but with a higher incidence of hypotension (20% vs 10%).

Conclusion: Both doses of magnesium sulphate effectively attenuate the haemodynamic stress response to laryngoscopy and tracheal intubation. While the 50 mg/kg dose provided better blood pressure control, the 30 mg/kg dose had a more favourable side-effect profile. The choice of dosage should be individualised based on patient characteristics and the anticipated degree of haemodynamic stress.

Keywords: Anaesthesia, Blood pressure, Haemodynamics, Sympathoadrenal activation

INTRODUCTION

Endotracheal intubation is an essential component of general anaesthesia for maintaining upper airway patency and ensuring proper ventilation. However, laryngoscopy and tracheal intubation are considered critical events during the induction of general anaesthesia as they stimulate somatic and visceral nociceptive afferent fibres, leading to sympathoadrenal activation [1]. This sympathoadrenal response results in increased catecholamine levels, causing a rise in blood pressure, heart rate, increased myocardial oxygen demand, and occasionally dysrhythmias. These haemodynamic changes are most pronounced during stimulation of the epipharynx and least marked from stimulation of the tracheobronchial tree [2]. While these responses may be well-tolerated by healthy individuals, they can be detrimental in patients with cardiovascular disease, increased intracranial pressure, or other co-morbidities [3].

Cardiovascular stability during the perioperative period is one of the fundamental goals of anaesthesia practice. Various pharmacological approaches have been employed to attenuate the haemodynamic response to laryngoscopy and endotracheal intubation, including topical anaesthesia of the oropharynx, intravenous lidocaine, adrenergic blocking drugs, vasodilators, deep inhalational anaesthesia, opioids, and magnesium sulphate [3].

Magnesium sulphate has garnered significant attention as it uniquely suppresses the release of catecholamines from the adrenal medulla and adrenergic nerve terminals [4]. Often referred to as a 'natural physiological calcium channel blocker,' magnesium has been promoted as a safe component for balanced general anaesthesia [5,6]. It attenuates vasopressor effects through various mechanisms, including blockage of catecholamine release,

calcium channel antagonism, and promotion of vasodilation, thus decreasing cardiovascular adverse events [5,6]. The role of calcium in catecholamine release in response to sympa thetic stimulation is well-established. Magnesium competes with calcium for binding to membrane channels, effectively acting as a calcium antagonist and modifying calcium-mediated responses. Consequently, magnesium sulphate blocks the release of catecholamine stores and diminishes responses to adrenergic stimulation [7]. Studies have demonstrated that magnesium sulphate reduces serum epinephrine levels, leading to decreased atrial contraction, bradycardia, and vasodilation [8,9].

Despite these recognised benefits, the effects of various doses of magnesium on haemodynamic responses to laryngoscopy have not been extensively investigated [8]. Previous studies have examined doses ranging from 30 mg/kg to 50 mg/kg, with varying results regarding efficacy and side-effects. Honarmand A et al., found that lower doses of magnesium sulphate (30 mg/kg or 40 mg/kg i.v.) were comparable to 50 mg/kg intravenously (i.v.) in significantly decreasing blood pressure changes following laryngoscopy [6]. Other studies by Kotwani MB et al., and Nandal S et al., suggested that 30 mg/kg provided adequate cardiovascular control without significant complications [7,10]. The existing literature lacks consensus on the optimal dose of magnesium sulphate that provides maximum haemodynamic stability with minimal adverse effects. Most studies have evaluated single doses rather than comparative dosing strategies, creating a gap in understanding dose-response relationships [9,10]. The present study was designed to compare the effects of two different doses of magnesium sulphate (30 mg/kg vs. 50 mg/kg) on the suppression of cardiovascular responses to laryngoscopy and endotracheal intubation during the first 10 minutes post-intubation. The primary outcome measure was the attenuation of haemodynamic stress response (heart rate and blood pressure changes) during laryngoscopy and intubation. Secondary outcome measures included the incidence of adverse events such as hypotension, bradycardia, and other complications.

MATERIALS AND METHODS

This single-blinded randomised clinical study was conducted at the Department of Anaesthesiology, SBKS Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed To Be University, Piparia, Vadodara, Gujarat, India, from January 2023 to June 2024. The study was approved by the Institutional Ethics Committee before patient enrollment [SVIEP/ON/medi/Srp/24/126], and written informed consent was obtained from all participants. The CTRI number was CTRI/2024/12/077749.

Sample size calculation: The sample size was calculated based on a previous similar study with a power of 80% and a significance level of 5%, using the mean difference in heart rate response between groups (15±5.2 beats/min) from Zhang J et al., resulting in 30 patients per group [9].

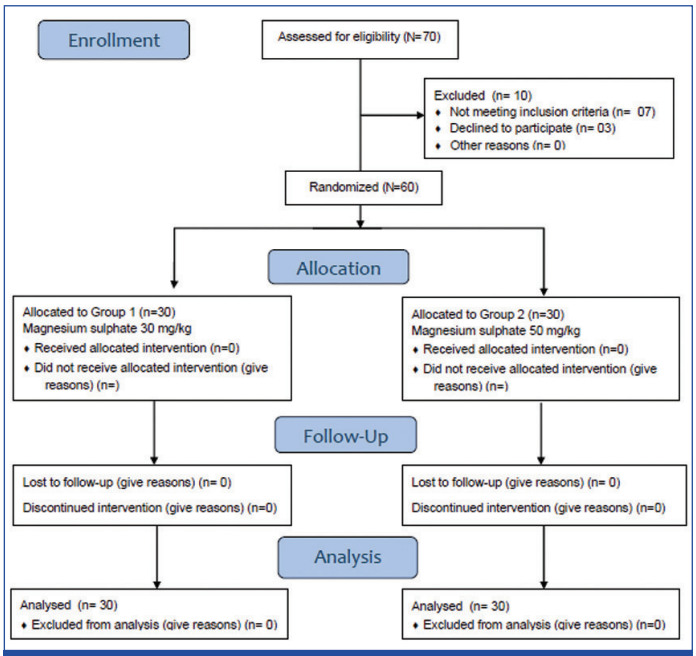
Inclusion criteria: It included patients of either gender aged 18-65 years, ASA physical status I or II, and scheduled for elective surgeries under general anaesthesia.

Exclusion criteria: It included major cardiovascular, cerebrovascular, hepatic, or renal diseases (ASA III, IV), arrhythmias or heart block, psychiatric conditions, pregnancy or lactation, taking beta-blockers or calcium channel blockers, history of allergy to study drugs, anticipated difficult airway, neuromuscular disease, serum magnesium level >2.5 mg/dL, premedication with strong opioids, multiple laryngoscopy attempts, laryngoscopy duration >30 seconds, laparoscopic procedure converted to open surgery.

A total of 70 patients were assessed for eligibility, 10 were excluded (7 did not meet the inclusion criteria, 3 declined to participate), and so, 60 patients were enrolled and completed the study.

Randomisation and group allocation: Computer-generated random numbers were used for randomisation by an independent researcher. Allocation concealment was maintained using sealed opaque envelopes. The anaesthesiologist administering the study drug was not involved in data collection (single-blind design). Measures to reduce bias included a standardised anaesthetic protocol and blinded outcome assessment. Patients were randomly divided into two equal groups using a computerised randomisation method :

- Group I (n=30): Magnesium sulphate 30 mg/kg i.v. diluted in 50 mL normal saline infusion administered over 10 minutes before induction [7].
- Group II (n=50): Magnesium sulphate 50 mg/kg i.v. diluted in 50 mL normal saline infusion administered over 10 minutes before induction [11] shown in Consolidated Standards of Reporting Trials (CONSORT) [Table/Fig-1]:



[Table/Fig-1]: CONSORT Flow diagram.

Study Procedure

The study drug was administered by a consultant anaesthesiologist present in the operating theatre who was not involved in the data collection.

Pre-operative assessment and preparation: A detailed pre-anaesthetic check-up was conducted for all patients one day before surgery. General parameters, including weight and airway assessment, were noted. Vital signs, including temperature, pulse rate, blood pressure, and respiratory rate, were recorded. Systemic examination of the respiratory, cardiovascular, abdominal, and central nervous systems was performed.

Pre-operative investigations included complete blood count, random blood sugar, blood urea, serum creatinine, serum electrolytes, serum magnesium level, Prothrombin Time/International Normalised Ratio (PT/INR), liver function tests, and Serum Glutamic Oxaloacetic Transaminase (SGOT), Serum Glutamic Pyruvic Transaminase (SGPT), total bilirubin, Electrocardiogram (ECG), chest X-ray, and serological tests for {Human Immunodeficiency Virus (HIV); Hepatitis C Virus (HCV); and Hepatitis B surface antigen (HBsAg)}. All pre-operative investigations were within normal limits for enrolled patients. Additional specific investigations were performed as required. Patients were kept nil per oral for 8 hours before surgery. Written informed consent was obtained from all patients meeting the inclusion criteria.

Anaesthetic management: After arrival in the operating theatre, patients were connected to a multiparameter monitor to record Heart Rate (HR), Non Invasive measurements of Systolic, Diastolic,

and Mean Arterial Pressure (SBP, DBP, MAP), continuous ECG, oxygen saturation (SpO_2, and end-tidal CO_2. An 18-gauge venous cannula was secured, and intravenous fluid (Ringer's lactate) was started.

Patients were premedicated with Inj. Glycopyrrolate 0.004 mg/kg i.v., Inj. Ondansetron 0.1 mg/kg i.v., Inj. Ranitidine 50 mg i.v., and Inj. Diclofenac Sodium AQ 75 mg i.v. The test drug was administered according to the allotted group 10 minutes before the induction of general anaesthesia as an infusion. After 10 minutes of infusion, patients were pre-oxygenated with 100% oxygen for 5 minutes and induced with Inj. Propofol 2.0-2.5 mg/kg i.v. Inj. Succinylcholine 2 mg/kg i.v. was administered to facilitate intubation after confirmation of bag and mask ventilation and loss of eyelash reflex.

The trachea was intubated with a cuffed endotracheal tube of appropriate size. Bilateral air entry and end-tidal CO_2 were confirmed, and the tube was secured. End-tidal CO_2 was maintained between 35-40 mmHg throughout the procedure. Anaesthesia was maintained with O_2 and N_2O at a 1:1 ratio and Isoflurane using a closed circle system. Inj. Atracurium (loading dose 0.5 mg/kg IV followed by 0.5 mg/kg/hr i.v. infusion) was administered for muscle relaxation until the end of surgery.

Patients were mechanically ventilated on volume control mode to maintain eucapnia. Haemodynamic parameters (HR, SBP, DBP, MAP) were recorded at baseline, after administration of the test drug, after induction of general anesthesia, during laryngoscopy and intubation, and at 1, 3, 5, 7, and 10 minutes after intubation.

Management of adverse events: Bradycardia (HR <50/min) was treated with Inj. Atropine 0.6 mg i.v. Hypotension (SBP >20% decrease from baseline) was initially treated with 200 mL of bolus Ringer's lactate fluid, followed by incremental doses of 6 mg Inj. Mephentermine i.v. if there was no improvement with fluid therapy. Haemodynamic stability was defined as changes between -20% to +20% from baseline blood pressure.

Post-operative anaesthetic protocol: After completion of surgery, neuromuscular blockade was reversed with Inj. Neostigmine (0.05 mg/kg) i.v. and Inj. Glycopyrrolate (0.008 mg/kg) i.v. Tracheal extubation was performed when extubation criteria were met (sustained head lift for 5 seconds, adequate tidal volume, and response to verbal commands). Patients were monitored in the post-anaesthesia care unit for 2 hours before transfer to the ward.

Data collection and outcome measurements: The following parameters were recorded:

1. Demographic data (age, gender, weight, height, ASA status)
2. Haemodynamic parameters (HR, SBP, DBP, MAP) at specified time points up to 10 minutes post-intubation
3. Post-operative complications, including nausea, vomiting, headache, flushing, itching, and confusion

STATISTICAL ANALYSIS

Data were collected, tabulated, and analysed using Statistical Package for Social Sciences (SPSS) version 20.0. Numerical variables were presented as mean and Standard Deviation (SD), while categorical variables were presented as frequency and percentage. For numerical variables, the unpaired Student's t-test was used for between-groups comparisons, while for categorical variables, the Chi-square test was used. Repeated measures One-way Analysis of Variance (ANOVA) was used to analyse changes in haemodynamic parameters over time. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 60 patients completed the study, with 30 patients in each group. The demographic characteristics, including age, gender, weight, height, Body Mass Index (BMI), and ASA status, were comparable between the two groups with no statistically significant differences, indicating successful randomisation [Table/Fig-2].

Parameters	Group I (n=30)	Group II (n=30)	p-value
Age (years)	38.9±7.5	41.0±8.8	0.32
Gender (M:F)	17:13	18:12	0.79
Weight (kg)	66.3±6.3	65.9±5.7	0.79
Height (cm)	164.6±5.3	164.1±6.0	0.72
BMI (kg/m²)	24.5±2.7	24.5±2.8	0.97
ASA status (I:II)	22:8	20:10	0.57

[Table/Fig-2]: Demographic characteristics of patients in the two groups.
Values are presented as (mean±SD) or ratio. p-value <0.05 is considered statistically significant.

Heart Rate Response

Group II (50 mg/kg) demonstrated significantly better heart rate control during laryngoscopy and intubation compared to group I (30 mg/kg), with peak increases of 14.3% vs 19.4% respectively (p-value<0.05) as shown in [Table/Fig-3]. Both groups returned to baseline by 10 minutes post-intubation, with no significant difference between them at this time point (p-values=0.37).

Time point	Group I (n=30)	Group II (n=30)	p-value
Baseline	81.8±4.2	81.8±4.6	0.96
After pre-medication	81.6±4.5	80.4±5.1	0.32
After induction	80.2±4.0	78.8±4.7	0.20
During laryngoscopy	94.0±5.1	89.4±5.2	<0.001
At intubation	97.7±5.5	93.5±5.0	0.003
1 min post-intubation	95.3±5.3	91.2±5.2	0.004
3 min post-intubation	90.6±5.1	85.1±5.0	<0.001
5 min post-intubation	86.6±4.9	82.9±4.4	0.003
7 min post-intubation	84.1±4.6	80.8±4.2	0.005
10 min post-intubation	81.2±4.2	80.1±5.0	0.37

[Table/Fig-3]: Heart rate changes during and after intubation.
Values are presented as mean±SD (beats/min). A p-value <0.05 is considered statistically significant.

Blood Pressure Response

Group II showed superior blood pressure control with SBP and DBP actually remaining below baseline during intubation (-1.7% and -0.8% respectively), while group I showed moderate increases (3.4% and 4.9% above baseline) as demonstrated in [Table/Fig-4]. These significant differences persisted throughout the 10-minute monitoring period (p-value<0.001 for most blood pressure parameters). Group II consistently maintained lower systolic, diastolic, and mean arterial pressures compared to group I, though by 10 minutes post-intubation, both groups showed a gradual return toward baseline values. All patients had respiratory rates between 12-16 breaths/min preoperatively.

Oxygen Saturation

The SpO_2 levels were well maintained in both groups with no clinically significant differences as shown in [Table/Fig-5]. Both groups showed minimal transient decreases during intubation and returned to baseline by 5 minutes with no significant between-group differences throughout the 10-minute study period (p-value >0.05).

Group II had a higher incidence of hypotension (20% vs 10%) and showed trends toward increased bradycardia (10% vs 3.3%), flushing (16.7% vs 10%), and headache (10% vs 3.3%), though these differences were not statistically significant as depicted in [Table/Fig-6]. No significant differences were observed in nausea or vomiting between groups.

DISCUSSION

Laryngoscopy and endotracheal intubation are known to cause significant sympathoadrenal responses, which can lead to

Time point	SBP (mmHg)			DBP (mmHg)			MAP (mmHg)		
	Group I	Group II	p-value	Group I	Group II	p-value	Group I	Group II	p-value
Baseline	126.7±4.7	126.9±4.6	0.87	76.9±4.3	77.0±4.6	0.94	93.4±3.5	93.6±3.9	0.82
After pre-medication	117.1±5.3	110.3±4.2	<0.001	70.2±3.7	67.7±4.4	0.02	85.8±3.4	81.9±3.5	<0.001
After induction	110.8±4.1	104.0±3.9	<0.001	66.9±4.2	64.1±4.0	0.01	81.5±3.2	77.4±3.1	<0.001
During laryngoscopy	127.8±5.4	120.3±4.6	<0.001	77.2±4.4	74.0±4.6	0.01	94.0±4.2	89.4±3.9	<0.001
At intubation	131.0±6.2	124.8±5.4	<0.001	80.7±4.7	76.4±4.8	<0.001	97.4±4.7	92.5±4.3	<0.001
1 min post-intubation	128.9±5.8	122.0±5.2	<0.001	79.2±4.6	74.8±5.0	<0.001	95.7±4.5	90.5±4.2	<0.001
3 min post-intubation	126.5±5.5	118.9±5.0	<0.001	77.4±5.0	72.3±5.3	<0.001	93.8±4.5	87.9±4.5	<0.001
5 min post-intubation	122.1±4.9	116.4±5.5	<0.001	74.9±4.5	70.8±5.3	0.002	90.6±4.1	85.9±4.7	<0.001
7 min post-intubation	120.4±5.1	114.2±5.3	<0.001	73.5±4.7	69.3±5.1	0.001	89.1±4.3	84.3±4.5	<0.001
10 min post-intubation	119.7±5.4	112.8±5.2	<0.001	72.6±4.7	68.6±5.5	0.003	88.3±4.3	83.3±4.7	<0.001

[Table/Fig-4]: Blood pressure parameters during and after intubation.
Values are presented as mean±SD. SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, MAP = Mean Arterial Pressure. p-value <0.05 is considered statistically significant.

Time Point	Group I (n=30)	Group II (n=30)	p-value
Baseline	99.0±0.8	98.9±0.9	0.65
After pre-medication	99.0±0.7	99.0±0.8	1.00
After induction	99.1±0.6	99.0±0.7	0.55
During laryngoscopy	98.9±0.8	98.8±0.9	0.52
At intubation	98.8±0.9	98.7±1.0	0.60
1 min post-intubation	99.0±0.7	98.9±0.8	0.48
3 min post-intubation	99.1±0.6	99.0±0.7	0.39
5 min post-intubation	99.2±0.5	99.1±0.6	0.32
7 min post-intubation	99.2±0.5	99.1±0.6	0.55
10 min post-intubation	99.3±0.4	99.2±0.5	0.52

[Table/Fig-5]: SpO_2 changes during and after intubation.
Values are presented as mean±SD (%). p-value <0.05 is considered statistically significant.

Adverse events	Group I (n=30) n (%)	Group II (n=30) n (%)	p-value
Hypotension	3 (10%)	6 (20%)	0.28
Bradycardia	1 (3.3%)	3 (10%)	0.30
Nausea	3 (10%)	3 (10%)	1.00
Vomiting	1 (3.3%)	2 (6.7%)	0.55
Flushing	3 (10%)	5 (16.7%)	0.45
Headache	1 (3.3%)	3 (10%)	0.30

[Table/Fig-6]: Incidence of adverse events.
The p-value <0.05 is considered statistically significant.

haemodynamic instability [1]. This randomised clinical study aimed to compare the efficacy of two different doses of magnesium sulphate (30 mg/kg vs. 50 mg/kg) in attenuating these responses during the first 10 minutes after intubation. The demographic characteristics of the patients in the two groups were comparable, ensuring that any differences observed in the haemodynamic parameters could be attributed to the study interventions rather than inherent differences among the groups.

The present study demonstrated that both doses of magnesium sulphate effectively attenuated the heart rate response to laryngoscopy and endotracheal intubation compared to baseline values [2,3]. After administration of magnesium sulphate, both groups showed minimal changes in heart rate, suggesting that magnesium's effect on heart rate is less pronounced than its effect on blood pressure [4]. During laryngoscopy and immediately after intubation, group I (30 mg/kg) showed a greater increase in heart rate (up to 19.4% above baseline) compared to group II (50 mg/kg), which showed a more modest increase (up to 14.3% above baseline). This difference was statistically significant (p-value<0.05) and indicates that the higher dose of magnesium sulphate provided better control of the heart rate response during the critical phase of intubation [5]. These findings on heart rate control are consistent with those of Honarmand A et al., who found that different doses of magnesium sulphate effectively reduced cardiovascular instability related to laryngoscopy and tracheal intubation, with higher doses showing better control [6]. However, the results differ slightly from those of Kotwani MB et al., who reported that 30 mg/kg of magnesium sulphate provided adequate cardiovascular control while higher doses were associated with transient tachycardia [7]. In the current study, both doses effectively controlled heart rate, with the 50 mg/kg dose providing better control without inducing tachycardia. Parul J, also reported similar findings in maxillofacial surgeries, demonstrating effective heart rate attenuation with magnesium sulphate [8]. Similar dose-dependent effects have been observed in other studies examining magnesium's cardiovascular effects [9,12].

Both systolic and diastolic blood pressure responses showed similar patterns in this study. After administration of magnesium sulphate, there was a decrease in blood pressure parameters in both groups, with group II (50 mg/kg) exhibiting a more pronounced decrease than group I (30 mg/kg). This is consistent with the vasodilatory effects of magnesium, which are dose-dependent [13]. During laryngoscopy and intubation, group I showed moderate increases in SBP (3.4% above baseline) and DBP (4.9% above baseline), while group II actually maintained values below baseline for both SBP (-1.7%) and DBP (-0.8%). This remarkable attenuation of the pressor response in group II highlights the superior efficacy of the higher dose in controlling blood pressure during the most critical phase of airway management [14]. Comparing these blood pressure findings with previous studies, they align with Nandal S et al., who found that magnesium at a dose of 30 mg/kg i.v. was optimal for attenuating the stress response in controlled hypertensive patients, as further increases in dose could lead to significant hypotension [10]. The current study in normotensive patients supports this observation, noting a higher incidence of hypotension (20% vs. 10%) with the 50 mg/kg dose. Tailor R also reported similar findings regarding the dose-dependent efficacy of magnesium sulphate in attenuating haemodynamic responses [15]. Recent studies by Seal A et al., and Sawant U, and Sen J have further validated these dose-dependent effects in various surgical populations [16,17].

The SpO_2 levels were well maintained throughout the study period in both groups. After administration of magnesium sulphate, there were

no significant changes in SpO_2 in either group. During laryngoscopy and intubation, there was a slight but clinically insignificant decrease in SpO_2 in both groups, similar to the results by Wang H et al., [18]. The SpO_2 levels returned to baseline by 5 minutes post-intubation and remained stable throughout the remainder of the 10-minute monitoring period. There were no statistically significant differences between the two groups at any time point (p-value>0.05 for all comparisons) [19].

The incidence of adverse events was generally low in both groups, indicating the safety of magnesium sulphate at both doses studied during the immediate post-intubation period. Similar findings have been observed by Lee JH et al., [20]. However, group II (50 mg/kg) had a higher incidence of hypotension (20%) compared to group I (10%), suggesting a dose-dependent risk of this adverse effect. This finding is consistent with the known vasodilatory effects of magnesium and highlights the importance of careful dosing to balance efficacy and safety [21]. Similar adverse event profiles have been reported by Montazeri K et al., in their dose-response study [22].

The findings of the current study have several important clinical implications for the immediate post-intubation period. First, they confirm that magnesium sulphate is an effective agent for attenuating the haemodynamic stress response response to laryngoscopy and endotracheal intubation during the critical first 10 minutes, which is particularly relevant for patients at risk of adverse consequences from haemodynamic instability, such as those with cardiovascular disease [23]. Second, the study provides guidance on dosing magnesium sulphate for this indication. While the 50 mg/kg dose provided better control of haemodynamic parameters during the immediate post-intubation period, it was associated with a higher incidence of hypotension [24]. The 30 mg/kg dose, while slightly less effective in controlling haemodynamic parameters, had a more favourable side-effect profile. This suggests that the choice of dose should be individualised based on patient characteristics and the anticipated degree of haemodynamic stress. Third, the findings demonstrate that both doses effectively return haemodynamic parameters to near-baseline levels by 10 minutes post-intubation, suggesting that the immediate period following intubation is when the protective effects of magnesium sulphate are most critical [25].

Limitation(s)

The present study has several limitations. First, it was a single-blind study rather than a double-blind study, which may introduce observer bias. Second, the study was conducted in ASA I and II patients, and results may not be generalisable to patients with significant co-morbidities. Third, the study only monitored haemodynamic parameters for 10 minutes post-intubation; longer monitoring might reveal additional differences between doses. Fourth, serum magnesium levels were not measured after drug administration, which could have provided insights into the pharmacokinetic-pharmacodynamic relationships. Fifth, the study did not include a control group without magnesium sulphate, which would have better quantified the absolute benefit of magnesium administration.

CONCLUSION(S)

Both doses of magnesium sulphate (30 mg/kg and 50 mg/kg) effectively attenuated the haemodynamic stress response to laryngoscopy and tracheal intubation during the first 10 minutes post-intubation. The 50 mg/kg dose provided better control of blood pressure parameters during the immediate period but was associated with a higher incidence of hypotension. The 30 mg/kg dose, while slightly less effective in controlling blood pressure, had a more favourable side effect profile. These findings suggest that magnesium sulphate is a valuable agent for maintaining haemodynamic stability during laryngoscopy and endotracheal intubation, with the optimal dose depending on individual patient characteristics and the anticipated degree of haemodynamic stress. Careful monitoring and prompt management of potential adverse effects, particularly hypotension with higher doses, are essential for patient safety during the immediate post-intubation period. Both groups demonstrated effective return to near-baseline haemodynamic parameters by 10 minutes post-intubation, confirming that the critical period for haemodynamic control is during and immediately following laryngoscopy and intubation.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Sep 08, 2025
- Manual Googling: Nov 01, 2025
- iThenticate Software: Nov 03, 2025 (15%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Aug 13, 2025**
Date of Peer Review: **Sep 09, 2025**
Date of Acceptance: **Nov 05, 2025**
Date of Publishing: **Jan 01, 2026**