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Effect of Intravenous Magnesium Sulphate Added to Paracetamol Lignocaine Regimen on Hemodynamic Response to Laryngoscopy and Intubation: A Randomized Double-Blind Clinical Study

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ABSTRACT

Background: Laryngoscopy and intubation cause marked sympathetic activation with rises in heart rate and blood pressure. Paracetamol and lignocaine provide limited control, especially of arterial pressure. Magnesium sulphate, with sympatholytic and calcium-antagonist actions, may improve stability. This study evaluates whether adding intravenous magnesium enhances attenuation of these hemodynamic responses.

Methods: This randomized, double-blind comparative study included 60 ASA I II patients aged 20-50 years undergoing elective surgery under general anaesthesia. Participants were allocated to two groups: Group PML received intravenous paracetamol, magnesium sulphate (50 mg/kg), and lignocaine; Group PL received paracetamol, placebo saline, and lignocaine. Hemodynamic parameters-heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP)-were recorded at baseline, post-induction, immediately after intubation, and at 1, 3, 5, 10, and 15 minutes.

Results: Baseline characteristics were comparable between groups. Heart rate responses remained similar at most time points, with Group PL showing significantly higher values at 15 minutes ($p < 0.05$). SBP, DBP, and MAP were significantly lower in Group PML at all post-intubation intervals ($p < 0.01$), indicating superior attenuation of pressor responses. Oxygen saturation remained stable in both groups.

Conclusion: The addition of magnesium sulphate to a paracetamol lignocaine regimen effectively blunts the hypertensive response to laryngoscopy and intubation, offering enhanced cardiovascular stability without compromising safety.

Keywords: Magnesium sulphate, Lignocaine, Paracetamol, Hemodynamic response, Laryngoscopy, Tracheal intubation

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INTRODUCTION

Laryngoscopy and tracheal intubation are integral components of general anaesthesia but are associated with a pronounced sympathetic response characterized by hypertension, tachycardia, arrhythmias, and increases in intracranial and intraocular pressures.[1,2] This pressor response is mediated by nociceptive stimulation of the upper airway, resulting in reflex activation of the sympathetic nervous system and a surge in circulating catecholamines.[1,3] While these transient hemodynamic changes are generally well tolerated in healthy individuals, they may precipitate myocardial ischemia, ventricular dysfunction, cerebrovascular events, or other serious complications in patients with pre-existing cardiovascular or cerebrovascular disease, or in those with poorly controlled hypertension.[1]

Various pharmacological strategies have been evaluated to attenuate the stress response to laryngoscopy and intubation, including deepening the plane of anaesthesia, beta-blockers, vasodilators, opioids, and local anaesthetic techniques. However, many of these modalities are limited by undesirable adverse effects such as hypotension, bradycardia, respiratory depression, or the need for invasive monitoring. Multimodal, opioid-sparing approaches using combinations of non-opioid analgesics and adjuvant agents are therefore increasingly preferred to optimize hemodynamic stability while minimizing drug-related complications.

Paracetamol is a centrally acting analgesic that inhibits prostaglandin synthesis within the central nervous system and modulates peripheral pain pathways, in addition to its antipyretic activity via hypothalamic action.[4] Intravenous paracetamol is widely used as part of perioperative multimodal analgesia due to its favourable safety profile. Lignocaine, an amide local anaesthetic, exerts its effect by blocking voltage-gated sodium channels and thereby inhibiting action potential propagation.[5,6] Intravenous lignocaine has been shown to blunt the pressor response to laryngoscopy and intubation when administered in appropriate doses prior to airway manipulation.[7,8]

Magnesium is the fourth most abundant cation in the body and an important intracellular ion that modulates numerous enzymatic and cellular processes. It acts as a physiological calcium antagonist and inhibits catecholamine release from adrenergic nerve endings and the adrenal medulla, thereby attenuating the sympathetic response to noxious stimuli.[9] Intravenous magnesium sulphate has been reported to reduce the blood pressure response associated with tracheal intubation, making it a potentially useful adjuvant in patients at risk of hemodynamic instability.[3,9]

Although previous studies have evaluated the effects of magnesium sulphate or lignocaine individually on the hemodynamic response to laryngoscopy and intubation, and a few have examined intravenous paracetamol in this context, the evidence regarding the combined use of these agents remains limited and inconsistent, particularly with respect to simultaneous control of both heart rate and blood pressure. Existing data suggest that blood pressure attenuation may be more consistent than heart rate control with many single-agent regimens.

In this context, the present randomized double-blind clinical study was designed to assess whether the addition of intravenous magnesium sulphate to a regimen of intravenous paracetamol and intravenous lignocaine provides superior attenuation of the hemodynamic response to laryngoscopy and tracheal intubation. Specifically, the objective was to compare the combination of intravenous paracetamol lignocaine with and without intravenous magnesium sulphate with respect to changes in heart rate and arterial blood pressure during and after laryngoscopy and intubation in patients undergoing elective surgery under general anaesthesia.

MATERIALS AND METHODS

This randomized, double-blind, comparative clinical study was conducted in the Department of Anaesthesiology after obtaining approval from the Institutional Ethics Committee and written informed consent from all participants. Adult patients aged 18 60 years belonging to American Society of Anaesthesiologists (ASA) physical status I or II and scheduled for elective surgeries under general anaesthesia requiring endotracheal

intubation were eligible for inclusion. Exclusion criteria comprised anticipated difficult airway, pregnancy, cardiovascular or cerebrovascular disease, uncontrolled hypertension, renal or hepatic impairment, chronic analgesic or sedative use, hypersensitivity to study medications, and refusal to participate.

A total of 90 patients were enrolled and randomized into three equal groups (n=30 each) using a computer-generated randomization table. Allocation concealment was maintained through sequentially numbered, sealed, opaque envelopes prepared by an independent investigator. Drug preparation and administration were performed by an anaesthesiologist not involved in patient monitoring or data collection to ensure blinding of both participants and outcome assessors.

Patients in Group P received intravenous paracetamol 1 g diluted in 100 mL normal saline administered over 10 minutes prior to induction. Group PL received the same dose of intravenous paracetamol followed by intravenous lignocaine 1.5 mg/kg administered 90 seconds before laryngoscopy. Group PML additionally received intravenous magnesium sulphate 30 mg/kg diluted in 100 mL normal saline infused over 10 minutes prior to induction, along with paracetamol and lignocaine in identical doses and timings as Group PL. All study medications were colorless solutions prepared in identical syringes or infusion bottles to maintain blinding.

All patients were kept fasting according to standard guidelines and received tablet ranitidine 150 mg and tablet alprazolam 0.25 mg the night before surgery. In the operating room, baseline heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded using a multiparameter monitor. Intravenous access was established, and preloading with 10 mL/kg of normal saline was administered.

Anaesthesia was induced with intravenous propofol 2 mg/kg and fentanyl 2 μg/kg. Neuromuscular blockade was achieved with intravenous vecuronium 0.1 mg/kg. After three minutes of controlled ventilation with 100% oxygen, direct laryngoscopy was performed using a Macintosh blade by an experienced anaesthesiologist with a standardized intubation technique to minimize variability. Intubation was completed within 15 seconds in all patients; any attempt exceeding this duration or requiring more than one attempt resulted in exclusion from analysis.

Hemodynamic parameters (heart rate, SBP, DBP, MAP) were recorded at baseline, immediately after drug administration, at induction, at laryngoscopy, and at 1,3,5, and 10 minutes post-intubation. Any episode of hypotension (SBP <90 mmHg or >20% decrease from baseline) was treated with intravenous fluids and mephentermine 3 6 mg. Bradycardia (HR <50 bpm) was managed with intravenous atropine 0.6 mg.

The primary outcome measure was attenuation of the hemodynamic response to laryngoscopy and intubation, assessed by comparing changes in heart rate and arterial pressures across the three groups. Secondary outcomes included incidence of adverse events such as hypotension, bradycardia, arrhythmias, or signs of drug intolerance.

Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation and compared using one-way ANOVA followed by post-hoc testing. Categorical variables were analyzed using chi-square or Fisher's exact test. A p-value <0.05 was considered statistically significant.

RESULTS

The study included 60 patients, with 30 participants in each treatment arm. Baseline demographic and physiological characteristics including age, gender distribution, BMI, ASA physical status, and Mallampati class were well matched between the groups (Table 1).

Heart rate decreased after intravenous paracetamol in both groups, without significant intergroup differences at most time points. A statistically significant rise was observed in Group PL at 15 minutes post-intubation ($p = 0.018$), indicating better sustained attenuation of tachycardic response in Group PML (Table 2).

Table 1: Baseline Characteristics of Study Participants (n = 60)

Variable	Group PML (n=30)	Group PL (n=30)	p-value
Gender			0.559
Female	23 (76.7%)	21 (70%)	
Male	7 (23.3%)	9 (30%)	
Age (years)	47.43 ± 15.69	40.57 ± 14.56	0.084
Weight (kg)	54.30 ± 10.72	60.67 ± 13.49	0.223
Height (cm)	153.33 ± 8.84	158.10 ± 8.63	0.896
BMI (kg/m^2)	23.15 ± 4.32	24.31 ± 5.13	0.347
ASA			0.347
Grade I	16.7%	26.7%	
Grade II	83.3%	73.3%	
Mallampati Grade			0.632
I	30%	20%	
II	53.3%	56.7%	
III	16.7%	20%	
IV	0%	3.3%	

Table 2: Heart Rate (beats/min) at Different Time Points

Time Point	Group PML (Mean ± SD)	Group PL (Mean ± SD)	p-value
Before IV Paracetamol	83.83 ± 8.99	84.00 ± 8.27	0.941
After IV Paracetamol	77.73 ± 8.10	80.67 ± 6.83	0.135
Post Induction	78.80 ± 8.46	75.20 ± 8.22	0.100
Immediately After Intubation	82.63 ± 11.60	84.07 ± 15.52	0.687
1 min	78.43 ± 7.55	82.73 ± 14.47	0.155
3 min	77.30 ± 10.51	79.77 ± 12.81	0.418
5 min	73.37 ± 8.63	74.23 ± 9.98	0.720
10 min	68.30 ± 8.16	73.40 ± 12.76	0.070
15 min	69.33 ± 9.06	76.80 ± 14.11	0.018

Table 3: Systolic, Diastolic, and Mean Arterial Pressures (mmHg)

Time Point	Group PML (Mean ± SD)	Group PL (Mean ± SD)	p-value
Systolic Blood Pressure (SBP)			
Before IV PCM	123.53 ± 8.18	119.00 ± 15.61	0.164
After IV PCM	111.73 ± 9.83	120.73 ± 18.73	0.023
Post Induction	105.70 ± 10.01	103.73 ± 16.11	0.572
Immediately After	105.90 ± 11.14	123.60 ± 23.67	<0.001
1 min	94.97 ± 8.07	120.17 ± 29.65	<0.001
3 min	93.40 ± 11.09	113.17 ± 22.56	<0.001
5 min	91.60 ± 7.13	108.77 ± 19.45	<0.001
10 min	93.00 ± 7.80	111.00 ± 17.06	<0.001
15 min	93.83 ± 7.80	115.87 ± 13.01	<0.001
Diastolic Blood Pressure (DBP)			
Before IV PCM	74.30 ± 5.71	74.00 ± 7.24	0.859
After IV PCM	71.77 ± 6.38	74.63 ± 7.94	0.129
Post Induction	66.30 ± 8.66	62.27 ± 8.15	0.068
Immediately After	71.23 ± 8.30	81.13 ± 14.92	0.002
1 min	65.27 ± 5.76	79.57 ± 15.60	<0.001
3 min	64.27 ± 6.38	74.07 ± 13.27	0.001
5 min	63.10 ± 6.04	70.87 ± 11.53	0.002
10 min	62.53 ± 7.22	70.93 ± 12.86	0.003
15 min	64.77 ± 6.34	77.27 ± 8.76	<0.001
Mean Arterial Pressure (MAP)			
Before IV PCM	90.27 ± 5.96	88.97 ± 9.53	0.529
After IV PCM	86.30 ± 5.92	90.37 ± 11.20	0.084
Post Induction	78.23 ± 8.08	74.27 ± 13.29	0.168
Immediately After	82.43 ± 10.35	95.83 ± 17.87	0.001
1 min	74.43 ± 6.91	92.97 ± 20.73	<0.001
3 min	74.50 ± 8.36	85.97 ± 17.55	0.002
5 min	73.27 ± 6.92	82.60 ± 15.15	0.003
10 min	72.57 ± 7.09	83.30 ± 14.71	0.001
15 min	73.77 ± 5.74	89.33 ± 13.75	<0.001

A consistent and clinically meaningful attenuation of systolic, diastolic, and mean arterial pressures was observed in Group PML following intubation. In contrast, Group PL demonstrated marked hypertensive responses immediately after intubation and throughout the subsequent monitoring period. These intergroup differences were significant at all post-intubation time points for SBP, DBP, and MAP (all $p < 0.01$), confirming superior hemodynamic stability with the addition of magnesium sulphate (Table 3).

Oxygen saturation remained stable (≥99.97%) in both groups throughout all measurement intervals with no significant differences, indicating adequate oxygenation during induction and intubation in both treatment protocols (Table 4).

Table 4: Oxygen Saturation (%) at All Time Points

Time Point	Group PML (Mean ± SD)	Group PL (Mean ± SD)	p-value
Before IV Paracetamol	100.00	100.00	
After IV Paracetamol	100.00	100.00	
Post Induction	100.00	99.97 ± 0.18	0.321
Immediately After Intubation	100.00	99.97 ± 0.18	0.321
1 min	100.00	99.97 ± 0.18	0.321
3 min	100.00	99.97 ± 0.18	0.321
5 min	100.00	100.00	
10 min	100.00	99.97 ± 0.18	0.321
15 min	100.00	100.00	

DISCUSSION

Laryngoscopy and tracheal intubation are well known to provoke significant sympathetic stimulation, resulting in abrupt increases in blood pressure and heart rate due to reflex catecholamine release from airway manipulation.[1,2] These hemodynamic surges may be tolerated by healthy individuals but can pose substantial peri-operative risk to patients with cardiovascular, cerebrovascular, or metabolic comorbidities. Therefore, numerous pharmacological strategies have been evaluated to blunt this response, including beta-blockers, opioids, vasodilators, calcium channel blockers, and local anaesthetic agents. Each, however, carries limitations such as hypotension, bradycardia, respiratory depression, or inconsistent efficacy.[10,11]

Lignocaine is widely used to attenuate airway reflexes and reduce the pressor response during intubation. Its mechanism involves prolongation of inactivation of voltage-gated sodium channels, thereby reducing nerve conduction and suppressing airway reactivity.[5] Earlier studies demonstrated variable but generally favourable effects of lignocaine on heart rate and arterial pressure when administered prior to laryngoscopy.[7] However, many investigators have reported that lignocaine provides only partial attenuation of sympathetic surges, particularly in hypertensive individuals or during rapid sequence induction.[10,12]

Magnesium sulphate has emerged as an attractive adjunct because of its dual mechanisms: inhibition of catecholamine release from the adrenal medulla and adrenergic nerve terminals, and direct vasodilatory action by antagonizing calcium-mediated vascular smooth muscle contraction.[9,13] It has been shown to suppress hormonal stress responses during laryngoscopy and intubation, producing more stable hemodynamic profiles. Prior trials suggested that magnesium may be more effective than lignocaine in attenuating blood pressure elevation, although effects on heart rate have been inconsistent.[14,15]

The present study evaluated whether adding intravenous magnesium sulphate to a regimen of paracetamol and lignocaine would further attenuate the cardiovascular response to laryngoscopy and intubation. The baseline demographic and physiological parameters between the two groups were comparable, ensuring valid intergroup comparison. Paracetamol infusion has been shown to reduce catecholamine-induced tachycardic responses through central COX inhibition, and both groups demonstrated modest heart rate reduction after paracetamol administration.[4]

Consistent with previous findings, heart rate responses in both groups showed minimal difference at most time points, with a significant intergroup difference emerging only at

15 minutes post-intubation. This aligns with observations of prior studies where magnesium did not consistently blunt tachycardia immediately after intubation but showed delayed beneficial effects.[14,16] In contrast, systolic, diastolic, and mean arterial pressures showed marked and consistent attenuation in the magnesium group across all post-intubation intervals. This robust finding corroborates earlier reports that magnesium sulphate is superior to lignocaine in stabilizing arterial pressure following airway instrumentation.[17,18]

The hemodynamic patterns observed sharp rises in SBP and DBP in the lignocaine group versus significantly blunted responses in the magnesium group underscore the potent calcium-antagonist and sympatholytic properties of magnesium. Similar outcomes were reported by James et al., who demonstrated suppression of catecholamine release with intravenous magnesium during laryngoscopy.[12] The return of blood pressure parameters toward baseline within 5-10 minutes in the magnesium group parallels the findings of Padmawar S et al. and Allen RW et al., who noted more rapid hemodynamic normalization with magnesium compared to lignocaine.[17,19]

Importantly, oxygen saturation remained stable and comparable between groups throughout the peri-intubation period, indicating that neither drug combination adversely affected respiratory or oxygenation status.

Limitations

The study involved a relatively small sample size and included only ASA I - II patients, which may limit generalizability to high-risk populations. Serum magnesium levels were not measured, preventing correlation of pharmacodynamic responses with serum concentrations. Furthermore, the study did not evaluate postoperative outcomes or potential delayed hemodynamic effects. Future studies with larger and more diverse populations, including high-risk cardiac patients, would help clarify the full clinical implications of magnesium use in airway management.

Conclusion

The addition of intravenous magnesium sulphate to a regimen of paracetamol and lignocaine provides significantly better attenuation of the hemodynamic response to laryngoscopy and tracheal intubation compared with paracetamol lignocaine alone. While both groups demonstrated similar control of heart rate, magnesium produced markedly greater suppression of systolic, diastolic, and mean arterial pressures at all post-intubation intervals. These findings support the use of magnesium as an effective and safe adjunct for achieving improved cardiovascular stability during airway instrumentation.

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Availability of Data: The data supporting this study's findings are available upon reasonable request to corresponding author.

Declaration of Non-use of Generative AI: The authors affirm that no generative artificial intelligence tools were utilized in the design, analysis, interpretation of data, or preparation of this manuscript. All content is the result of the authors' original work.

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