Section: Anaesthesiology



## **Original Research Article**

SAFETY AND **SULPHATE** IN **RESPONSE** TO LAPAROSCOPIC CONTROLLED **STUDY** 

**EFFICACY** OF **MAGNESIUM** MODULATING **HEMODYNAMIC PNEUMOPERITONEUM** DURING PLACEBO-SURGERIES: SINGLE-BLIND RANDOMIZED

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### **ABSTRACT**

Background: Pneumoperitoneum during laparoscopic surgeries causes significant hemodynamic changes due to increased intra-abdominal pressure and hypercarbia, leading to elevated heart rate (HR) and blood pressure. Magnesium sulfate (MgSO<sub>4</sub>) may mitigate these effects through its vasodilatory and anti-catecholamine properties. The aim is to evaluate the efficacy and safety of magnesium sulfate in attenuating hemodynamic responses during laparoscopic surgeries. Materials and Methods: A randomized study was conducted on 60 patients aged 18-60 years undergoing elective laparoscopic surgeries. Patients were divided into two groups: Group A received MgSO<sub>4</sub> (30 mg/kg diluted in 100 mL normal saline), and Group B received 100 mL normal saline. Hemodynamic parameters, including HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), were monitored at baseline and during pneumoperitoneum. Adverse events and rescue drug requirements were documented. Result: MgSO4 significantly reduced HR, SBP, DBP, and MAP during pneumoperitoneum compared to the control group. None of the MgSO4 group required rescue drugs, while 10% of the control group required intervention for hypertension. Adverse effects were minimal, with one case of transient bradycardia in the MgSO<sub>4</sub> group. Conclusion: Magnesium sulfate effectively attenuates hemodynamic fluctuations during laparoscopic surgeries without significant adverse effects. It is a safe and effective option for managing pneumoperitoneum-induced hemodynamic changes.

### INTRODUCTION

Laparoscopy, a minimally invasive surgical technique, involves gas insufflation to separate the anterior abdominal wall from the viscera, enabling endoscopic access to the peritoneal cavity. Since its introduction in the mid-1950s, laparoscopy has revolutionized surgical practices by reducing bleeding, improving cosmesis, lowering medical costs, promoting early recovery, and minimizing postoperative complications. The evolution of laparoscopy to encompass more complex procedures has necessitated advancements in anesthetic techniques and innovations.

The initial step in laparoscopic surgery involves inserting a trocar or insufflation needle into the abdominal cavity, followed by gas insufflation (commonly CO<sub>2</sub>) to maintain an intra-abdominal pressure (IAP) of 12–15 mmHg.<sup>[1,2]</sup> An ideal insufflation gas should exhibit minimal peritoneal absorption, rapid excretion, non-combustibility, high blood solubility, and limited physiological effects. Laparoscopic cholecystectomy, now regarded as the "gold standard" for managing benign gallbladder diseases, has significantly transformed the treatment of gallbladder pathology.[3,4]

The advantages of laparoscopy include reduced postoperative complications such as pain, respiratory dysfunction, wound dehiscence, ileus, infections.<sup>[5,6]</sup> Additional benefits include quicker recovery, reduced intraoperative blood loss, and a lower inflammatory response.[6,7] However, laparoscopy also presents challenges, such as increased risk of injuries, longer operative times8, limited three-dimensional depth perception, [9] and hemodynamic changes caused by pneumoperitoneum and positioning (e.g., reverse Trendelenburg). Elevated intra-abdominal pressure and CO<sub>2</sub> absorption significantly affect respiratory, metabolic, and hemodynamic parameters. [10,11]

Anesthetic management during laparoscopic surgeries is often complicated by hemodynamic alterations induced by CO2 pneumoperitoneum and patient positioning. [12] These changes include increased arterial pressure, systemic vascular resistance, and reduced cardiac output. Elevated levels of vasopressin and catecholamines exacerbate these effects, contributing to tachycardia and hypertension. Various pharmacological agents, including opioids, β-blockers, α-adrenergic agonists, calcium channel blockers, vasodilators, lignocaine, have been used to mitigate stress responses with varying success. While some medications, such as opioids and calcium channel blockers, reduce hemodynamic responses, they may also impair cardiac output.

Magnesium sulfate has emerged as a promising alternative due to its ability to attenuate stress responses while preserving cardiac output. It reduces serum epinephrine levels and systemic vascular resistance, improves cardiac output, and inhibits catecholamine and vasopressin release from endings.[13,14] adrenergic nerve Additionally, magnesium sulfate induces vasodilation directly and reduces vasoconstriction triggered vasopressin.<sup>[15,16]</sup> Its role in mitigating adverse hemodynamic responses during pneumoperitoneum and endotracheal intubation has been welldocumented.[17,18]

Other effects of pneumoperitoneum include transient reductions in hepatic blood flow, potentially elevating liver enzymes19. Employing low-pressure pneumoperitoneum has been shown to mitigate these adverse effects on hepatic and portal circulation19. Understanding the resorption patterns of pneumoperitoneum is crucial for managing postoperative outcomes.

This study evaluates the safety and efficacy of magnesium sulfate in attenuating hemodynamic responses during pneumoperitoneum in laparoscopic surgeries, contributing to advancements in anesthetic management.

# **MATERIALS AND METHODS**

Study Design and Setting: This prospective, randomized, placebo-controlled, single-blind study was conducted in the Department of Anaesthesiology at DY Patil University School of Medicine and Hospital, Nerul, Navi Mumbai. The study was carried out over two years after obtaining approval from the Institutional Ethical Committee (IEC Ref. No: DYP/IECBH/2022/283, dated 21/01/2023).

Informed written consent was obtained from all participants before enrolment. The study included patients undergoing laparoscopic surgeries with CO<sub>2</sub> pneumoperitoneum under general anesthesia. A total of 60 patients were enrolled and randomly divided into two groups: Group A and Group B, with 30 patients in each group.

#### **Sample Size Calculation**

The sample size for this study was calculated using the formula for a two-group comparison study:

$$n = rac{\left(2\sigma^2
ight) imes\left[Z_{lpha/2} + Z_eta
ight]^2}{d^2}$$

Where: ZZ represents the table value of alpha error from the standard normal distribution curve, Power  $(\rho) = 90\%$ , Significance level  $(\alpha) = 0.05$ , Standard deviation  $(\sigma) = 2$ , Effect size  $(\delta) = 1.5$ ,  $Z_{\alpha/2} = 1.96$ ,  $Z_{\beta} = 0.84$ . Using the above parameters, the calculated sample size per group was n=27.88. To simplify, a sample size of 30 patients per group was chosen, resulting in a total sample size of 60 patients for the study.

Selection Criteria: Patients for the study were selected based on specific inclusion and exclusion criteria to ensure a standardized and homogenous population. The study included adult patients aged 18 to 60 years undergoing elective laparoscopic surgeries under general anesthesia. Only patients classified as American Society of Anesthesiologists (ASA) Grade I and II and those who provided informed written consent were included. Patients were excluded if they were unwilling to participate, had morbid obesity, or presented with severe hepatic, endocrine, cardiac renal, or dysfunction. Additionally, pregnant and lactating patients were not considered for the study.

Randomization and Group Allocation: Patients were randomly assigned to one of two study groups using a sequentially numbered envelope method to ensure unbiased allocation. A total of 60 patients were divided equally between the two groups, with 30 patients in each group.

- Group A (n=30): Received magnesium sulphate (MgSO<sub>4</sub>) at a dose of 30 mg/kg diluted in 100 mL normal saline.
- Group B (n=30): Received 100 mL of normal saline as a placebo.

Perioperative Procedure and Monitoring: Upon arrival in the operation theatre, each patient's fasting status, written informed consent, and pre-anaesthetic checkup were verified. Routine ASA monitors, including pulse oximeter, non-invasive blood pressure (NIBP), and ECG, were attached to the patient, and baseline systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and oxygen saturation (SpO<sub>2</sub>) were recorded. An IV cannula was secured, and Ringer lactate infusion was initiated at 2–4 mL/kg/hr. Patients were pre-oxygenated with 100% oxygen for 3 minutes before administration of Inj. Glycopyrrolate (4 mcg/kg), Inj. Midazolam (0.03

mg/kg), and Inj. Fentanyl (2 mcg/kg). Anaesthesia induction was performed using intravenous propofol (2 mg/kg) until the eyelash reflexes were abolished, followed by Inj. Atracurium (0.7 mg/kg) to achieve neuromuscular blockade. Intermittent positive pressure ventilation was done for three minutes, laryngoscopy was performed using McIntosh blade and patient was intubated with appropriate size ETT and fixed after confirming position of ETT by chest auscultation and positive EtCo2.

Group A patients received an intravenous bolus of MgSO<sub>4</sub> (30 mg/kg body weight) diluted in 100 mL normal saline administered slowly over 10 minutes, while Group B patients received 100 mL normal saline over the same duration before pneumoperitoneum. Anaesthesia was maintained with a mixture of oxygen and air (50:50), 1-2% sevoflurane, and intermittent doses of atracurium. Pneumoperitoneum was established by insufflating the peritoneal cavity with CO<sub>2</sub>, maintaining an intraabdominal pressure (IAP) of 12 mmHg. Ventilation was adjusted to maintain EtCO2 levels between 35-40 mmHg.

Throughout the process, vital parameters such as heart rate (HR), blood pressure, ECG, temperature, EtCO<sub>2</sub>, and SpO<sub>2</sub> were continuously monitored. Bradycardia (HR < 60 beats/min) was treated with Inj. Atropine (0.6 mg IV), while hypotension (SBP drop > 20% or MAP < 60 mmHg) was managed with increased IV fluid infusion rates and Inj. Mephentermine in graded doses (0.3 mg). Hypertension (MAP > 110 mmHg) was addressed with bolus Inj. Propofol (20 mg IV) followed by Dexmedetomidine infusion which was used as a rescue drug for hemodynamic control.

Ondansetron (4 mg) was administered at the end of surgery to prevent postoperative nausea and vomiting. Neuromuscular blockade was reversed using Neostigmine (0.05 mg/kg) and Glycopyrrolate (8 mcg/kg), after which the patients were extubated upon achieving spontaneous respiration and full recovery.

HR and MAP were recorded at the following intervals: baseline, before pneumoperitoneum, at 5, 15, 60 30. 45, and minutes pneumoperitoneum, after release  $\alpha f$ pneumoperitoneum, and post-extubation. Patients were closely monitored in the recovery room for any adverse events such as bradycardia, hypotension, or hypertension.

**Statistical Analysis:** The collected data were organized into a tabulated format using Microsoft Excel and analyzed statistically with MedCalc software. Demographic data were evaluated using the Chi-square test and t-test, while the comparison of means between the two groups was performed using the ANOVA statistical tool. A p-value of less than 0.05 was considered statistically significant.

## **RESULTS**

The demographic characteristics, ASA grades, and comorbidity distribution were comparable between Group A and Group B (Table 1). The mean age was  $44.4 \pm 13.59$  years in Group A and  $49.60 \pm 10.22$ years in Group B, with no significant difference. Group A comprised 60% males and 40% females, while Group B had 53.33% males and 46.67% females, with no significant differences (p > 0.05). ASA Grade I was observed in 63.33% of Group A and 53.33% of Group B, and Grade II in 36.67% and 46.67%, respectively, with no significant difference (p>0.05). Hypertension was present in 20% of Group A and 13.33% of Group B, while diabetes mellitus was more common in Group B (30% vs. 6.67%in group A). Most patients had no comorbidities (63.33% in Group A and 56.67% in Group B). The comparable demographics of the sample population across the groups indicate that the allocation was random, ensuring that any differences in outcomes can be attributed to the intervention rather than preexisting baseline variations.

Table 1: Demographic Characteristics, ASA Grades, and Comorbidity Distribution Between Group A and Group E
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Parameter	Category	Group A (n=30)	Group B (n=30)	P Value
Age Distribution	18-40	13 (43.33%)	6 (20.00%)	0.1594 (NS)
	40-50	10 (33.33%)	17 (56.67%)	
	50-60	7 (23.33%)	7 (23.33%)	
	$Mean \pm SD$	$44.4 \pm 13.59$	$49.60 \pm 10.22$	
Sex Distribution	Male	18 (60.00%)	16 (53.33%)	0.7316 (NS)
	Female	12 (40.00%)	14 (46.67%)	0.6949 (NS)
ASA Grade	I	19 (63.33%)	16 (53.33%)	0.6121 (NS)
	II	11 (36.67%)	14 (46.67%)	0.5485 (NS)
Comorbidity	HTN	6 (20.00%)	4 (13.33%)	
	DM	2 (6.67%)	9 (30.00%)	
	Other	3 (10.00%)	0 (0.00%)	
	None	19 (63.33%)	17 (56.67%)	

NS- Non-Significant

The hemodynamic parameters, including SBP and DBP, were monitored at various time intervals for both groups (Table 2). At baseline, no significant differences in SBP and DBP were observed between

Group A and Group B (p> 0.05). Similarly, values remained comparable 15 minutes before pneumoperitoneum.

Following the initiation of PP, significant elevations in SBP and DBP were noted in Group B compared to Group A at 5 minutes and 10 minutes post-PP (p< 0.0001). At 15 minutes post-PP, SBP remained significantly higher in Group B (P = 0.0067), while DBP showed no significant difference (p> 0.05). By 30 minutes post-PP, SBP differences persisted (p = 0.0382), but DBP remained similar between the

groups (p> 0.05). At later intervals, including 45 and 60 minutes post-PP, as well as after the release of PP and extubation, no significant differences in SBP or DBP were observed between the groups (p> 0.05). These findings indicate transient hemodynamic changes following PP, particularly in Group B, which normalized during the recovery period.

Table 2: Hemodynamic Parameters - SBP and DBP Across Time Points.

Time (min)	Mean SBP			Mean DBP		
	Group A	Group B	P Value	Group A	Group B	P Value
Baseline	$118.23 \pm 7.10$	$120.80 \pm 7.89$	0.19	$74.67 \pm 4.46$	$75.96 \pm 4.36$	0.2619
15 Min Before PP	$113.10 \pm 9.51$	$116.87 \pm 5.59$	0.0663	$73.90 \pm 5.27$	$75.13 \pm 4.69$	0.3436
5 Min After PP	$127.43 \pm 6.90$	$142.57 \pm 7.27$	< 0.0001	$83.77 \pm 3.79$	$93.90 \pm 4.92$	< 0.0001
10 Min After PP	$131.23 \pm 5.56$	$140.57 \pm 6.85$	< 0.0001	$84.70 \pm 4.66$	$92.27 \pm 5.38$	< 0.0001
15 Min After PP	$124.23 \pm 7.50$	$130.00 \pm 8.36$	0.0067	$79.30 \pm 7.54$	$78.80 \pm 5.39$	0.7687
30 Min After PP	$121.70 \pm 9.73$	$126.53 \pm 7.80$	0.0382	$78.53 \pm 7.29$	$78.30 \pm 5.55$	0.8911
45 Min After PP	$121.13 \pm 8.24$	$123.93 \pm 8.83$	0.2092	$76.53 \pm 5.89$	$77.30 \pm 4.07$	0.5581
60 Min After PP	$120.56 \pm 9.30$	$122.23 \pm 8.83$	0.4785	$75.93 \pm 7.84$	$76.96 \pm 8.28$	0.6226
After Release of PP	$118.97 \pm 7.13$	$120.46 \pm 7.09$	0.4203	$73.93 \pm 4.70$	$76.30 \pm 5.44$	0.0762
After Extubation	$135.07 \pm 7.19$	$135.50 \pm 7.20$	0.8178	$82.87 \pm 5.85$	$83.63 \pm 7.54$	0.6643

SBP: Systolic blood pressure, DBP: Diastolic blood pressure; PP: pneumoperitoneum

Further HR, MAP, and SpO<sub>2</sub>, were assessed across different time points for both groups (Table 3). At baseline, no significant differences in HR, MAP, or SpO<sub>2</sub> were observed between Group A and Group B (p> 0.05). Similarly, these parameters remained comparable 15 minutes before pneumoperitoneum. Following the initiation of PP, significant increases in HR and MAP were observed in Group B compared to Group A at 5 minutes and 10 minutes post-PP (p < 0.0001). At 15 and 30 minutes post-PP, both HR and MAP differences between the groups were no longer

significant. SpO<sub>2</sub> levels remained consistently at 100% in both groups throughout these time points. At later intervals, including 45 and 60 minutes post-PP, as well as after the release of PP and extubation, no significant differences in HR, MAP, or SpO<sub>2</sub> were noted between the groups. These findings highlight transient elevations in HR and MAP in Group B during the early PP phase, with normalization in subsequent time points. SpO<sub>2</sub> levels remained stable in both groups across all measurements.

Table 3: Hemodynamic Parameters – HR, MAP and Spo2 Across Time Points

Time in Minutes	Mean HR			Mean MAP			Mean	SpO <sub>2</sub>
	A	В	P Value	A	В	P Value	A	В
Base Line	$77.00 \pm 7.8$	$77.67 \pm 6.7$	0.723	$89.04 \pm 4.0$	$90.92 \pm 4.2$	0.0869	100	100
15 Min before PP	$76.47 \pm 5.4$	$80.03 \pm 9.7$	0.0809	$87.33 \pm 5.2$	$89.04 \pm 4.0$	0.1643	100	100
5 Min after PP	$89.60 \pm 5.9$	$105.8 \pm 9.6$	< 0.0001	$98.32 \pm 4.0$	$112.6 \pm 4.8$	< 0.0001	-	-
10 Min after PP	$88.77 \pm 5.0$	$101.6 \pm 8.8$	< 0.0001	$100.21 \pm 4.2$	$108.3 \pm 5.0$	< 0.0001	-	-
15 Min after PP	$84.47 \pm 6.4$	$85.87 \pm 9.5$	0.5071	$94.18 \pm 6.4$	$95.86 \pm 5.0$	0.2672	100	100
30 Min after PP	$82.47 \pm 7.5$	$84.20 \pm 7.8$	0.3879	$92.92 \pm 6.6$	$94.42 \pm 4.8$	0.3222	100	100
45 Min after PP	$81.33 \pm 7.2$	$80.4 \pm 10$	0.6896	$91.40 \pm 5.4$	$92.84 \pm 4.7$	0.2786	100	100
60 Min after PP	$75.80 \pm 7.3$	$76.60 \pm 9.1$	0.7095	$90.81 \pm 7.5$	$92.05 \pm 7.6$	0.5291	100	100
After PP	$73.33 \pm 6.0$	$75.9 \pm 10.5$	0.2387	$88.94 \pm 4.2$	$91.02 \pm 4.3$	0.0639	100	100
After extubation	$85.80 \pm 8.1$	$88.7 \pm 10.8$	0.2467	$100.27 \pm 5.0$	$100.9 \pm 5.7$	0.6419	100	100

HR: Heart rate, MAP: Mean arterial pressure; Spo2: Oxygen saturation, PP: pneumoperitoneum

The need for rescue drugs was observed exclusively in Group B, where 3 patients (10.00%) required intervention, while no such requirement was reported in Group A (Table 4). Regarding adverse events, hypotension was not observed in either group. Bradycardia was reported in 1 patient in Group A but was absent in Group B, with no statistically

significant difference between the groups (p> 0.05). Hypertension was noted in 3 patients in Group B, whereas no cases were reported in Group A, with a trend towards significance (p= 0.0780) (Table 4). Overall, Group A demonstrated fewer adverse events and no requirement for rescue drugs compared to Group B.

**Table 4: Need for Rescue Drug and Adverse Events** 

Parameter	Group A	Group B	P Value
Need for Rescue Drug	0 (0.00%)	3 (10.00%)	=
Adverse Events			
Hypotension	0	0	>0.05
Bradycardia	1	0	>0.05
Hypertension	0	3	0.0780

## **DISCUSSION**

During laparoscopic surgeries, pneumoperitoneum represents a complex pathophysiological phase significant characterized by hemodynamic fluctuations.<sup>[20]</sup> The insufflation of carbon dioxide (CO<sub>2</sub>) and positional changes during surgery can lead to increased HR and elevated blood pressure due to CO<sub>2</sub> retention. These hemodynamic alterations arise from elevated intra-abdominal pressure and hypercarbia, which activate the renin-angiotensinaldosterone system (RAAS). This activation results in heightened systemic and pulmonary vascular resistance, increased arterial pressure, and reduced cardiac output.<sup>[21]</sup> To mitigate these hemodynamic changes, various pharmacological agents are employed during premedication and induction. Magnesium sulfate has emerged as an effective agent, known for its ability to inhibit catecholamine release from adrenal glands and adrenergic nerve terminals. Additionally, its vasodilatory properties help in reducing blood pressure.<sup>[22]</sup>

In this randomized comparative study, we evaluated the efficacy of magnesium sulfate in attenuating the hemodynamic responses associated with pneumoperitoneum along with assessment of its safety profile by observing any adverse effects associated with its use.

Patients aged 18 to 60 years were included in the study, as hemodynamic responses to interventions and stressors, such as pneumoperitoneum, are more pronounced in younger patients and tend to diminish with advancing age. This range ensures a balanced assessment of the impact of MgSO<sub>4</sub> on hemodynamic parameters across an appropriate clinical population. Similar age demographics have been observed in previous studies investigating the effects of MgSO<sub>4</sub>. Ashutosh Singh et al. (2016) reported mean ages of 44.18 years in the control group and 42.45 years in the MgSO<sub>4</sub> group, indicating a comparable age distribution.<sup>[23]</sup> Paul S et al. (2013) similarly observed mean ages of 43.7 years and 41.4 years in the MgSO<sub>4</sub> and control groups, respectively.[24] The gender distribution in our study was comparable to the findings of Karthikeyan et al., who reported 46.7% males and 53.3% females in the MgSO<sub>4</sub> group, and 56.7% males and 43.3% females in the control group.<sup>[22]</sup> Our ASA grade distribution aligns Bagle et al., who observed 51.7% ASA I and 48.2% ASA II patients in the MgSO<sub>4</sub> group, and 37.9% ASA I and 51.7% ASA II patients in the control group.<sup>[20]</sup> The comorbidity distribution in our study showed no statistically significant difference between the groups, making them comparable. Similarly, Sravanthi et al. (2023) reported 4 cases of hypertension in the MgSO<sub>4</sub> group and 3 cases in the control group.<sup>[25]</sup>

In our study, HR trends showed a significant decrease in the MgSO<sub>4</sub> group compared to the control group at 5 and 10 minutes after pneumoperitoneum, highlighting MgSO<sub>4</sub>'s efficacy in attenuating the

tachycardic response during this phase. These findings are consistent with previous studies. Karthikeyan et al. demonstrated a significantly lower HR in the MgSO<sub>4</sub> group compared to the control group.<sup>[22]</sup> Similarly, Reddy et al. reported that HR in the MgSO<sub>4</sub> group was significantly lower at 5, 10, and 20 minutes after pneumoperitoneum compared to the control group, with highly significant p-values (<0.0001) at all time points.<sup>[21]</sup> Paul S et al. also observed that HR in the MgSO<sub>4</sub> group was significantly lower throughout pneumoperitoneum, after its release, and post-extubation (p<0.05). However, in contrast, our study noted significant differences only at the 5- and 10-minute marks.

In our study, MgSO<sub>4</sub> demonstrated a significant decrease in SBP at 5, 10, 15, and 30 minutes after pneumoperitoneum compared to the control group, highlighting its role in mitigating hemodynamic stress during laparoscopic procedures. These findings align with previous research. Reddy et al. reported that SBP was significantly lower in the MgSO<sub>4</sub> group at 5, 10, and 20 minutes after pneumoperitoneum.[21] Similarly, Karthikeyan et al. observed a significant reduction in SBP in the MgSO<sub>4</sub> group compared to the placebo group, with the effect lasting up to 60 minutes post-pneumoperitoneum (p < 0.05)22. Jee et al. also noted significantly lower systolic arterial pressure in the MgSO<sub>4</sub> group at 10, 20, and 30 minutes post-pneumoperitoneum compared to their control group (p < 0.05).[26]

Our study demonstrated a significant reduction in DBP in MgSO<sub>4</sub> group at 5 and 10 minutes after pneumoperitoneum compared to the control group, highlighting its efficacy in controlling hemodynamic responses during laparoscopic procedures. Our findings are supported by Reddy et al., who observed that DBP was significantly lower in the MgSO<sub>4</sub> group compared to the control group at 5, 10, 15, and 20 minutes post-pneumoperitoneum.<sup>[21]</sup> The extended duration of significant differences in their study could be attributed to the higher dose of MgSO<sub>4</sub> (50 mg/kg) used, compared to the dosage administered in our study.

Further in our study, MgSO<sub>4</sub> significantly reduced MAP at 5 and 10 minutes after pneumoperitoneum compared to the control group, with results being statistically significant (p<0.05). This suggests that MgSO<sub>4</sub> effectively attenuates hemodynamic fluctuations during the initial phases pneumoperitoneum. Our findings align with those of Tan et al., who observed a significant reduction in MAP in the MgSO<sub>4</sub> group (30 mg/kg) at 5 and 10 minutes post-pneumoperitoneum compared to the control group. They noted that these intervals represent the periods of maximum hemodynamic fluctuation during pneumoperitoneum, making them the most critical time for MgSO<sub>4</sub> to exert its antihypertensive effect. However, the hemodynamic fluctuations at 30 and 60 minutes were less pronounced, indicating that MgSO4 does not significantly affect normal blood pressure but is effective in mitigating hypertension associated with

pneumoperitoneum.<sup>[27]</sup> Paul et al. also reported significant reductions in MAP throughout pneumoperitoneum, following its release, and after extubation in patients receiving MgSO<sub>4</sub> (30 mg/kg) compared to the control group. These results further support the use of MgSO<sub>4</sub> in controlling MAP during laparoscopic surgeries.<sup>[24]</sup>

In our study, there were no changes in SpO2 levels from baseline to after extubation in both the MgSO<sub>4</sub> and control groups, indicating that MgSO<sub>4</sub> did not adversely affect oxygen saturation levels.

In our study, none of the patients in group A (MgSO<sub>4</sub> group, 30 mg/kg) required a rescue drug, while 3 patients (10%) in group B (control group, 100 ml NS) required intervention for hypertension. This indicates that MgSO<sub>4</sub> effectively managed intraoperative hemodynamic fluctuations, minimizing the need for additional pharmacological interventions.

Our findings are consistent with the study by Paul et al., who reported that 12 patients in the control group required rescue drugs for hypertension, highlighting the efficacy of MgSO<sub>4</sub> in attenuating hypertensive episodes during surgery.<sup>[24]</sup> Further during our study, adverse effects were minimal and clinically insignificant. No patient in either group experienced hypotension. However, one patient in group A (MgSO<sub>4</sub> group) developed bradycardia with a HR of 54 bpm after the release of pneumoperitoneum. This represented a 20% decrease from the patient's baseline heart rate of 62 bpm. Since this change was not clinically significant and depth of anesthesia was not monitored, the bradycardia could have resulted from a deep plane of anesthesia or the residual effects of MgSO<sub>4</sub>. In group B (control group), 3 patients experienced hypertension during the procedure. Thus the adverse effects were not significant and highlight the safety profile of MgSO<sub>4</sub> when used for hemodynamic control in laparoscopic surgeries.

The limitations of our study include a small sample size, the absence of catecholamine level measurement, and a lack of depth of anesthesia monitoring, with rescue drugs administered based on heart rate and blood pressure alone. High-risk hypertensive and cardiac patients were also excluded, limiting the generalizability of the findings.

## **CONCLUSION**

Our study demonstrates that the administration of magnesium sulfate (30 mg/kg) before pneumoperitoneum effectively attenuates hemodynamic response in patients undergoing laparoscopic surgeries. This effect is likely due to its vasodilatory properties and the reduced release of catecholamines and vasopressin. Magnesium sulfate significantly reduces heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure without causing any notable adverse effects. Further research involving varied doses, diverse patient populations, and the measurement of serum catecholamine levels is warranted to better understand its mechanism of action and optimize its clinical application.

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