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Medical Science

INTRAVENOUS MAGNESIUM SULPHATE IN ATTENUATING PRESSOR RESPONSE TO LARYNGOSCOPY AND INTUBATION

KEY WORDS:

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INTRODUCTION

Laryngoscopy and tracheal intubation form an integral part of general anaesthesia. Although the associated elevations in blood pressure (BP) and heart rate (HR) are brief, they may have detrimental effects in high-risk patients.[1,2] Intravenous (IV) magnesium sulphate has also been shown to reduce the haemodynamic changes associated with laryngoscopy and intubation.[3]

METHODS

After approval from the Institutional Ethics Committee, this study enrolled 100 patients of ASA I and II, aged 18–60 years, of either sex, scheduled for elective surgery under general anaesthesia using induction with propofol and maintenance with atracurium and sevoflurane. The exclusion criteria were refusal of consent; hepatic, renal or cardiovascular dysfunction; uncontrolled hypertension; epilepsy; pregnancy; postural hypotension; anticipated difficult airway; anticipated major blood losses and fluid shift; patients on sedatives and drug allergies. Pre-anaesthetic evaluation and relevant investigations were carried out in all patients. On the night before the surgery, informed written consent was obtained and tablet alprazolam 0.5 mg was given to all patients and fasted for 6 h.

Pre-operative ECG, SpO2, HR and blood pressures, systolic (SBP) and diastolic (DBP) were recorded. This was a participant and investigator blinded study. Group A (n = 50) received IV magnesium sulphate 30 mg/kg and Group B (n = 50), control group, received normal saline intravenously. Blinding was done by preparation of the injectable solution using magnesium sulphate or 0.9% saline (control) by an independent observer not involved in this study. Infusion of the study drug was given in 100 ml normal saline over 15–20 min 15 min before induction according to the groups allotted. SBP, DBP, HR, were recorded after 5 min of drug infusion. IV glycopyrrolate 0.01 mg/kg were given to all patients to avoid any bias.

Patients were pre-oxygenated for 3 min with 100%oxygen and then, anaesthesia was induced with propofol IV and then 1.5 mg/kg succinylcholine IV was administered. Laryngoscopy was carried out gently in <15 s. The pressor response was evaluated. Patients requiring >15 s for laryngoscopy, bronchospasm or laryngospasm were added in the demographic profile but excluded from statistical analysis. Perioperative hypotension was defined as SBP <90 mmHg and bradycardia as HR <50 beats/min. Arrhythmias were defined as supraventricular or premature ventricular beats >3/min or any rhythm other than sinus. Injection atropine 0.3 mg increments and mephentermine 6 mg bolus were used to treat bradycardia and hypotension, respectively. SpO2, HR, SBP and DBP were recorded at intubation, at the interval of 1 min till 5 min, then 2 min till 10 min and every 10 min for 30 min.

Subsequently, the maintenance of anaesthesia was carried out with sevoflurane and 66% nitrous oxide in oxygen with controlled ventilation using a closed circuit throughout. Injection Atracurium 0.5 mg/ kg IV was given as an initial dose for neuromuscular blockade and 0.01 mg//kg bolus IV when required. Residual neuromuscular blockade was reversed at the end of surgery using IV neostigmine 0.05 mg/kg and glycopyrrolate 0.02 mg/kg, followed by extubation. The primary outcome of interest was changes in BP and HR from baseline, at intubation and up to 30 min after intubation. Assuming the power of our study 80% and an alpha error of 0.05, sample size was calculated to be 40.[4]

Statistical Package for the Social Sciences (SPSS) 17.0 software was used for the statistical analysis of the compiled data by applying Chi-square test and ANOVA test. $P < 0.001$ was considered a highly significant difference and $P < 0.05$ was significant.

RESULTS

Demographic data between groups were similar [Table 1]

Variables	Group A	Group B	pvalue
Age	50.07±10.37	51.13±8.6	0.669
Weight	62.93±14.12	63.17±12.97	0.842
Gender	26/24	25/25	0.605
ASA	25/25	25/25	nil

Mean HR just before induction was 74±5.91/min in group A and 75.47±5.21/min in group B without significance difference (p =0.312) while at induction time it started to show significance difference (p <0.001) with a mean of 82.474.49/min in group A and 104.24 6.2/min in group B. In the subsequent reading at 5min, 10 min and 20min, mean HR showed the same significance ($p < 0.001$). After 30 minutes, mean HR was 77.4 3.83 /min in group A and 79.23 5.19/min.in group B with statistically insignificant value ($p = 0.124$) (Table 2)

Regarding mean MAP before induction it was 77.27 ±6.55 mmHg in group A and 76.97 ±7.3 mmHg in group B which was statistically insignificant (p=0.861). While at time of intubation it was 80.5 6.45 mmHg in group A and 95.73 6.68 mmHg in group B with significant difference ($p < 0.001$). At the following check points (at 60 seconds, 5min, 10 min and 20min), it showed the same significant difference ($p < 0.001$). But 30 minutes after mean MAP was 74.53±5.26 mmHg in group A and 76.73 ±4.91 mmHg in group B with no significant difference (p =0.1). (Table 3)

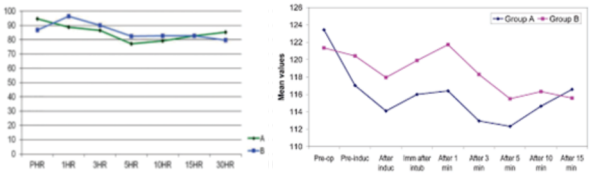
Table 2: Comparison between groups A and B for Mean heart rate (HR)/ minute

Variable	Group A	Group B	pvalue
Preoperative	74±5.91	75.47±5.21	0.312
Induction	76.7±5.98	97.2±5.81	<0.001
After 60 second	82.47±4.49	104.27±6.2	<0.001
After 5 minutes	80.13±6.33	96.77±8.63	<0.001
After 10 minutes	78.3±4.81	93.8±6.33	<0.001
After 20minutes	75.67±4.67	84.03±4.31	<0.001
After 30 minutes	77.4±3.83	79.23±5.19	0.124

Data are represented in Mean values ± SD

Table 3: Comparison between groups A and B for mean arterial blood pressure (MAP) in mmHg

Variable	Group A	Group B	pvalue
Preoperative	77.27±6.55	76.97±7.3	0.861
Induction	80.5±6.45	95.73±6.68	<0.001
After 60 second	86.07±6.12	103.33±6.76	<0.001
After 5 minutes	75.53±3.89	92.53±6.36	<0.001
After 10 minutes	76.33±5.7	86.53±6.05	<0.001
After 20minutes	75.5±5.67	82.07±4.63	<0.001
After 30 minutes	74.53±5.26	76.73±4.91	0.10



DISCUSSION

Laryngoscopy and intubation are known to cause an increase in HR and blood pressure.[1,3,5] Magnesium sulphate in a bolus dose of 30 mg/kg was observed to attenuate the adverse haemodynamic responses without any hypotension or bradycardia.[6] Infusion of magnesium sulphate produces a fall in HR and BP by directly blocking the release of catecholamines from both adrenal gland and adrenergic nerve terminals and indirectly through negative feedback mechanism.[3] It also acts directly on the blood vessels leading to vasodilatation and decreases vasopressin-stimulated vasoconstriction.[7]

Magnesium sulphate (MgSo4) is non-competitive N-Methyl-D-Aspartate (NMDA) receptor antagonist and a calcium channel blocker, it has an analgesic effect by blocking nociceptive central sensitization. It has also an ability to reduce peripheral nociception by reduction of catecholamine release.[8,9]

In this study, sudden increase in MAP and HR has been noticed at induction –intubation time with a mean rise of 3.2 mmHg and 76.7/min respectively in magnesium group versus 18.8 mmHg and 97.2/min in saline group with a significant difference between both groups. The peak rise was noticed after 60 seconds of (8.7, 26.3 mmHg and 82.47, 104.27/min in both groups). This was the same peak time recorded by **Arshad A et al.**[10] for this noxious stimulus to cause its maximal pressor effect.

CONCLUSION

Intravenous magnesium sulphate effectively attenuated both HR and arterial blood pressure responses to laryngoscopy and intubation.

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