Original Article

Comparison of Intravenous Magnesium Sulphate and Lidocaine Effects on Attenuating Haemodynamic Variables to Laryngoscopy and Intubation in Patient Undergoing General Anesthesia

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Abstract

Objective: To determine the effect of intravenous xylocaine and magnesium sulfate on attenuation of hemodynamic response to laryngoscopy and intubation in patients undergoing general anaesthesia.

Methods: This was a randomized controlled study carried out at operation theaters of services hospital lahore after obtaining approval from IRB of hospital. The data was collected over period of six month from 20.05.2020 to 20.12.2020 through electronic databases. 60 patients were divided into two groups of 30 each by lottery method in this randomized control trial. Intravenous magnesium sulphate 30 mg/ kg diluted in 50 ml normal saline 15 min before induction was administered in M group and 50 ml normal saline given in L group. Induction was done with propofol 2 mg/ kg, followed by suxamethonium 2 mg/ kg. I/V lignocaine 1.5 mg/kg diluted in N/S (5ml) was given as bolus in L group and 5 ml N/S IV bolus in M group 1 minute before intubation. Laryngoscopy was performed and the trachea was intubated after 1 minute. HR, systolic (SBP), diastolic (DBP) and mean arterial pressures (MAP) were measured just before securing intravenous access, just before induction, after intubation and 1,3,5 min post intubation.

Results: Mean age for both groups was 36.0 ± 12.8 and 38.2 ± 10.8 . Mean HR was significantly different between two groups immediately after intubation (p=0.010), and at 1, 3 and 5 minutes also (p=0.004, p=0.018 and p=0.024) respectively. No significant difference was seen in systolic, diastolic and mean blood pressures at intubation, 1 minute, 3 minutes and 5 minutes after intubation among the groups (p>0.05).

Conclusion: Both Magnesian Sulfate and lignocaine are effective in attenuating haemodynamic response to laryngoscopy and intubation but magnesium sulphate provides better efficacy in control of heart rate.

Key Words: Haemodynamic response, laryngoscopy, Intubation, magnesium sulphate, lignocaine.

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Introduction

Laryngoscopy and endotracheal intubation induces a stress response that occurs due to sympathoadrenal stimulation.^{1,2,3,4} This pressor response leads to various cardiovascular changes such as increase in heart rate, rise in arterial blood pressure from baseline

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and dysrhythmias.² These transient cardiovascular responses may not affect normal healthy individuals but may increase perioperative morbidity and mortality in patients with coexisting disease such as ischemic heart disease, hypertension, cerebrovascular disease and diabetes mellitus.^{1,2}

Several pharmacological and non-pharmacological methods were tried to minimize the adverse sympathoadrenal response at different times but few were found to be effective. Various drugs like lignocaine, magnesium sulphate, opioids, beta blockers, clonidine, labetalol, calcium channel blockers and vasodilators such as hydralazine have been used.¹

The use of I/V magnesium sulfate 15min before induction of anesthesia provide steady, smooth

reduction and control of MAP and HR after intubation. This attenuation in hemodynamic response results from the inhibition of catecholamine release from the adrenal medulla and thus indirect vasodilation of blood vessels leading to a decrease in blood pressure. It also has a systemic and coronary vasodilation effect by antagonizing calcium ion in vascular smooth muscle.⁵

2% lignocaine, an amide local anaesthetic, is most widely used to attenuate the stress response to laryngoscopy and intubation when given in dose of 1.5mg/kg intravenously 90 seconds before induction.² The beneficial effect of lidocaine is due to its direct cardiac depression and peripheral vasodilation, ability to suppress airway reflexes as well as antiarrhythmic properties.⁶ Lignocaine decreases airway reactivity by reducing release of substance P and its glycinergic action.⁷

Various studies have been done to compare intravenous magnesium sulphate with lignocaine and other drugs but efficacy is still controversial. The aims of the study to compare the effect of prophylactic use of IV magnesium sulfate with lidocaine on hemodynamics following laryngoscopy and intubation in patients undergoing general anaesthesia.

Methods

This was a randomized controlled study carried out at operation theaters of services hospital Lahore after obtaining approval from IRB of hospital. The data was collected over period of six months from 20.05. 2020 to 20.12.2020 through electronic databases. 60 patients scheduled for elective surgery were divided into two groups of 30 each by lottery method. Patients undergoing major head and neck surgeries under general anaesthesia with endotracheal intubation, aged 20-50 years of American Society of Anesthesiologists ASA I and II were included. Patients in whom difficult airway was anticipated, American Society of Anesthesiologists ASA III, patient with raised ICP, IHD, hypertensive and having diabetes mellitus were excluded.

IV access was established with 20G cannula. Baseline heart rate, BP, ECG and Oxygen saturation was recorded. Intravenous magnesium sulphate 30 mg/kg diluted in 50 ml normal saline 15 min before induction was administered in M group and 50 ml normal saline given in L group. All patients were premedicated with nalbuphine 0.1 mg/kg intravenously and induc-

tion was done with propofol 2 mg/ kg, followed by suxamethonium 2 mg/ kg. I/V lignocaine 1.5 mg/kg diluted in N/S was given as bolus in L group and 5 ml N/S IV bolus in M group 1 min before intubation. A quick and gentle laryngoscopy not lasting for more than 15 second was then performed by one anesthetist and the trachea was intubated. Atracurium 0.25 mg/ kg intravenously was administered to maintain anaesthesia using oxygen with isoflurane 1% and IPPV.

Heart Rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressures (MAP) were measured just before securing intravenous access (baseline value), just before induction, after intubation and 1,3,5 min post intubation. Hypertension was considered when BP value was greater than 20% of baseline. Hypotension was considered when BP was less than 20% of baseline. Tachycardia was considered when HR was more than 20% of base line or HR greater than 100bpm. Bradycardia was labelled when HR was lower than 50 bpm.

The data for age, weight, SBP, DBP, MAP, and HR were all described by using "Mean±SD". The comparison between two groups at baseline, immediately after intubation, one, three and five minutes after intubation were made by using independent sample ttest. Comparison of each hemodynamic parameter with its baseline value within each group was made by using paired t-test. P-value 0.05 was considered statistically significant. Line graphs were used to present the changes from baseline to 5 minutes after intubation for each parameter.

Results

The mean age for both groups was 36.0 ± 12.8 and 38.2 ± 10.8 which was insignificantly different between two groups, beside this the weight, systolic diastolic and mean arterial pressure were all insignificantly different at baseline with p-values 0.755, 0.877, 0.692 and 0.824 respectively. The heart rate for L group at baseline was 93.0 ± 19.3 and that for M group was 86.7 ± 15.6 and this difference was also insignificant with p-value 0.168. (Table.1)

As the baseline values were all insignificant so direct comparisons were made at each instance at and after intubation between two groups. The systolic, diastolic and mean blood pressures were not found significant at intubation, and 1, 3 and 5 minutes after intubation between two groups. The mean heart rate, however, immediately after intubation was found significantly different between two groups with p-value 0.010, and at 1, 3 and 5 minutes also were significantly different with p-values 0.004, 0.018 and 0.024 respectively. (table.2)

When compared within group the systolic blood pressure raised a bit from baseline in both groups till

Table 1: Demographic and Hemodynamic Parameters atStart of Study after Randomization

	Group				P-value	
	L		Μ		comparison between	
	(n=30)		(n=30)			
	Mean	SD	Mean	SD	groups	
Age	36.0	12.8	38.2	10.8	0.474	
Weight (kgs)	66.6	10.2	67.5	12.7	0.755	
SBP	134.3	13.1	133.7	18.0	0.877	
DBP	82.7	12.4	83.9	11.6	0.692	
MAP	102.2	12.1	102.9	12.3	0.824	
HR	93.0	19.3	86.7	15.6	0.168	

immediate after intubation but this raise was insignificant. The p-value for immediately after intubation was 0.108 in group L and 0.057 in M. At one 1 minute after intubation the SBP declined and p-values for two groups were <0.001 and 0.005 respectively. The mean systolic blood pressure reached to 104 mmHg for group L and 105 mmHg for group M at 5 minutes and both were highly significant as compared to baseline with p-values <0.001. (Table.2, fig.1)

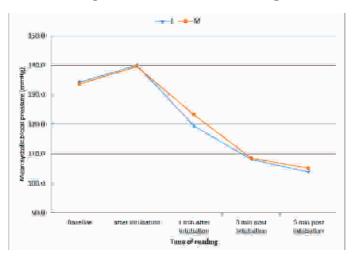


Figure.1: Systolic Blood Pressure for Patients in Two Groups from Baseline to 5 Minutes after Intubation The pattern for diastolic blood pressure was not diffe-

Table 2: Comparison of Hemodynamic Parameters from Baseline within Group and between Groups at Each
 Reading Time After Intubation

		L		P-value		
	Mean ± SD	P-value Within group comparison to base line	Mean ± SD	P-value Within group comparison to base line	comparing two groups	
SBP						
immediately after intubation	140.1 ± 18.1	0.108	139.6 ± 23.1	0.057	0.931	
1min after intubation	119.4 ± 16.9	< 0.001	123.2 ± 15.4	0.005	0.359	
3min post intubation	108.2 ± 14.7	< 0.001	108.5 ± 13.1	< 0.001	0.934	
5min post intubation	103.9 ± 13.2	< 0.001	105.1 ± 14.6	< 0.001	0.740	
DBP						
immediately after intubation	94.6 ± 14.9	0.001	95.1 ± 16.0	< 0.001	0.914	
1min after intubation	77.4 ± 12.6	0.054	82.9 ± 16.5	0.762	0.152	
3min post intubation	68.0 ± 13.0	< 0.001	70.0 ± 13.7	< 0.001	0.556	
5min post intubation	64.0 ± 10.9	< 0.001	66.7 ± 12.4	< 0.001	0.381	
MAP						
after intubation	109.7 ± 16.8	0.027	110.3 ± 15.9	0.008	0.881	
1min post intubation	92.4 ± 13.1	0.002	94.0 ± 17.1	0.008	0.692	
3min post intubation	82.1 ± 13.4	< 0.001	83.1 ± 13.0	< 0.001	0.763	
5min post intubation	77.6 ± 11.7	< 0.001	80.2 ± 13.2	< 0.001	0.410	
HR						
post-intubation	104.2 ± 13.2	0.003	95.8 ± 11.2	0.003	0.010	
1mins after intubation	98.2 ± 13.1	0.099	88.6 ± 11.4	0.504	0.004	
3mins after intubation	90.7 ± 14.9	0.402	82.6 ± 10.6	0.107	0.018	
5min after intubation	86.0 ± 12.4	0.006	78.6 ± 12.4	0.001	0.024	

rent than systolic. It also raised immediately after intubation to 94.6 and 95.1 mmHg in two groups and this raise was significant with p-value 0.001 in L and <0.001 in M group. The decline after one minute was insignificant in group L with p-value 0.054 and in group M with p-value 0.762. Later DBP continued to decline significantly in both groups with p-values <0.001. (Table.2, fig.2)

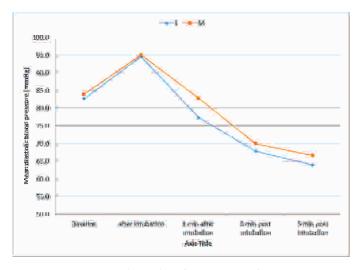


Figure.2: Diastolic Blood Pressure for Patients in Two Groups from Baseline to 5 Minutes After Intubation

The mean arterial pressure increased significantly in both groups immediately after intubation (p=0.002 & p=0.008) and then declined in both groups significantly at each reading time as compared to baseline. At five minutes the MAP was 77.6 ± 11.7 in group L and 80.2 ± 13.2 in group M. (Table.2, Fig.3)

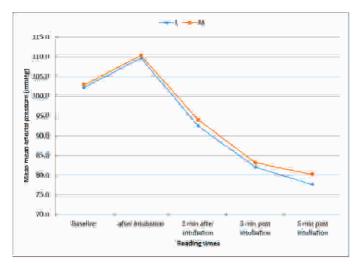


Figure.3: Mean Arterial Pressure for Patients in Two Groups from Baseline to 5 Minutes after Intubation

The heart rate in group L rose to 104.2±13.2 immediately after intubation and this raise was highly significant with p-value 0.003. In group M the heart rate increased to 95.8 ± 11.2 and this increase was also significant with p-value 0.003. It declined after one minute in both groups but the decline was insignificant in both groups with p-values 0.099 and 0.504 respectively. Even after 3 minutes of intubation the heart rate was insignificantly different in both groups with p-values 0.402 and 0.107. Later, after 5 minutes, the heart rate declined in group L to 86.0 ± 12.4 with p-value 0.006 and in group M to 78.6 ± 12.4 with a p-value 0.001. (Table.2, Fig.4)

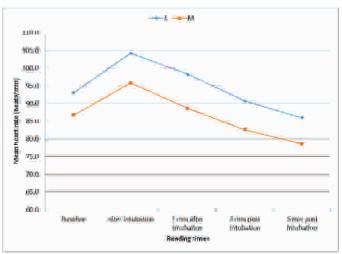


Figure.4: Heart rate for Patients in Two Groups from Baseline to 5 Minutes after Intubation

Discussion

Laryngoscopy and intubation, being noxious stimuli, incite remarkable sympathetic activity. Studies have shown an increase in heart rate with intubation and a greater increase in blood pressure with direct laryngoscopy.⁶ This pressor response, arises 30s after laryngoscopy and intubation and returns to baseline values steadily within 5–10 min. These transitory responses usually produce no consequences in healthy individuals but may be harmful to the patients having reactive airways, hypertension, coronary artery disease, myocardial insufficiency and cerebrovascular diseases.¹

Common factors precipitating the pressor response to laryngoscopy and intubation are light planes of anaesthesia, prolonged time for the procedure, elevation of vagally innervated posterior part of epiglottis by straight/ Miller blade, anatomically difficult view, greater force used to displace the tongue and more manipulations/ attempts at laryngoscopy and intubation. Several drugs and maneuvers have been used for mitigating this stress response with variable benefits and side effects. Magnesium Sulphate and lignocaine 2% appear to meet the characteristic of a drug to prevent these sympathetic response. Comparison of magnesium sulphate with lignocaine in our study did not show significant difference in systolic, diastolic and mean blood pressures at intubation, 1 minute, 3 minutes and 5 minutes after intubation among the groups (p> 0.05). However, the mean heart rate decreased significantly in Magnesium group after 1, 3 and 5 minutes of intubation when compared to Lignocaine (p=0.01, p=0.00, and p=0.02).

In 2017, Bhalerao NS showed similar result to our study as there was no significant increase in BP after laryngoscopy when compared with base line with use of intravenous MgSO4 50 mg/kg and lignocaine 2mg/kg in hypertensive patients.⁸

In accordance to our study, Rajan Sunil observed there was a statistically significant decrease in HR from the pre-induction values in MgSO4 group (50mg/kg) than lignocaine group up to 15 min following intubation. Although there was decrease in systolic blood pressure, diastolic blood and mean arterial pressure from induction values in both groups, there was no significant difference between the groups.⁹

Kiaee M et al concluded similar results regarding heart rate changes when comparing magnesium sulphate with lignocaine. Difference in results were shown in systolic, diastolic and mean arterial pressures. In contradiction to our study results they found greater decrease in systolic, diastolic and mean arterial pressures with lignocaine.⁵

Padmawar S in 2016 found comparable results to our study. The difference in attenuation of heart rate was significant among groups but no significant difference was seen in SBP, DBP and MAP in comparison of lignocaine and magnesium sulphate groups at intubation, 1 minute, 3 minute and 5 minutes after intubation. Magnesium sulphate group was found better than lignocaine.²

Consistent with our results, significant fall in heart rate was noted by Vallabha et al following induction, laryngoscopy, and intubation when comparing magnesium (30mg/kg) with lignocaine (1.5mg/kg) group. The decrease in mean arterial pressure was significant unlike the results of our study.³

Nooraei N et al used 60mg/kg magnesium sulphate and lignocaine 1.5mg/kg in 60 patient and concluded

there was significant decrease in heart rate with lignocaine at 3rd and 4th minutes after intubation that were contrary to our results. But similarity was seen in the results of systolic, diastolic and mean arterial pressures with no significant difference among both groups.¹⁰

In 2017 CCRGA evaluated 56 patient and found different result from our study. There was increase in heart rate and blood pressure after laryngoscopy and intubation compared to baseline. The reason may be they used bispectral index in their study to assess the depth of anesthesia which is not used in our study.

In 2011 SH Majid Waseem et all found different result from our study. They found lignocaine was more effective and efficacious than magnesium sulphate. In 89 patients they used intravenous 1% lignocaine 1mg/kg and magnesium sulphate 10 mg/kg instead of 1.5mg/kg lignocaine and 30mg/kg magnesium sulphate intravenous in our study.¹¹

We did not monitor depth of anaesthesia due to unavailability of Bispectral Index and also used suxamethonium for intubation instead of nondepolarizing muscle relaxant. In future, research can be planned to evaluate these limitations of our study.

Conclusion

Both Magnesian Sulfate (30mg/kg) and IV lignocaine (1.5mg/kg) are effective in attenuating haemodynamic response to laryngoscopy and intubation without any deleterious effects. However, magnesium sulphate provides better efficacy in control of heart rate.

Conflict of Interest: None

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