Original Article

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A Randomized Double Blind Comparative Study of Intravenous Magnesium Sulphate and Intravenous Lignocaine for Attenuation of Haemodynamic Response to Laryngoscopy and Tracheal Intubation

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Abstract

Introduction: Endotracheal intubation has become a standard part of anaesthesia and critical care management. All of these operations can cause sympathetic responses, and it's important to remember that many of these patients are extremely ill and at risk. The aim is to compare the efficacy of intravenous Magnesium sulphate with intravenous Lignocaine hydrochloride in attenuation of cardiovascular responses to laryngoscopy and intubation. **Subjects and Methods :** A Prospective Randomized Double Blind Study. The study was done in General Operation Theatre Complex of the Tirumala Hospitals, Vizianagaram, Andhra Pradesh.70 Patients, 35 in each group. The study comprised patients who were scheduled for ASA class I and II elective surgical operations under general anaesthetic. **Results:** Percent rise in the HR, SBP, DBP, and MAP were significantly less in Group B(magnesium) compared to Group A(lignocaine). $MgSO_4$ 30mg/kg given intravenous 90 seconds prior to laryngoscopy and intubation for attenuation of pressor response results in significant reduction in HR, SBP, DBP and MAP at 1 min after laryngoscopy and intubation and no significant changes after 3 min when compared to intravenous lignocaine 1.5mg/kg. **Conclusion:** Intravenous magnesium sulphate 30mg/kg 90 seconds prior to laryngoscopy and intubation is superior to lignocaine 1.5mg/kg prior to laryngoscopy and intubation.

Keywords: Intravenous magnesium sulphate, Lignocaine hydrochloride.

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Introduction

Endotracheal intubation has become a standard feature of anaesthesia and critical care, with circulatory responses to laryngeal and tracheal stimulation as reflex sympathoadrenal activation following laryngoscopy and tracheal intubation. Even though the increase in blood pressure and heart rate caused by laryngoscopy and intubation is only temporary, it can cause myocardial infarction, cardiac failure, cerebral haemorrhage, and an increase in intracranial pressure in high-risk patients. Changes in circulating catecholamine levels are greatly influenced by laryngoscopy and tracheal intubation. Norepinephrine, epinephrine and dopamine levels rise, but the raise in Norepinephrine levels is consistently associated with elevation of blood pressure and heart rate.[1] In fact, some writers consider the intubation period to be one of the most dangerous in surgical patients with coronary artery disease and cerebral aneurysms. The response is invariable, significant, often persistent, and of great worry, even if it is temporary.[2] The procedures of laryngoscopy and tracheal intubation are not only used in the operating room, but also in non-anaesthetic situations. Diagnostic laryngoscopy and fibreoptic bronchoscopy are two examples of situations where intubation may be required to prevent aspiration and protect the airway, as well as during mechanical ventilation. All of these operations can cause sympathetic responses, and it's important to remember that many of these patients are extremely ill and at risk. As a result, it's critical to find a way to reduce the sympathetic reaction to laryngoscopy and tracheal intubation. Many treatments focused at different levels of the reflex arc have been advised to reduce these deleterious hemodynamic effects.[1]

Topical administration and injection of local anaesthetic to the superior laryngeal nerve to block peripheral sensory recep-

tors and afferent input. Anesthetics, narcotics, alpha 2 receptor agonists, and other drugs block central mechanisms of integration and sensory information. Blockade of efferent pathway and effector sites i.v. lignocaine, beta blockers, calcium channel blockers, hydralazine etc. No single drug or technique is satisfactory. Deep anaesthesia, topical anaesthesia, use of ganglion blockers, beta blockers, antihypertensive drugs such phentolamine, sodium nitroprusside, nitroglycerine, calcium channel blockers are some of the approaches used to reduce intubation-related stress responses. Clonidine, an agonist for the adrenoreceptor, reduces the adrenergic hemodynamic stress response.[3] The purpose of this study was to see how efficient intravenous lignocaine and magnesium sulphate were at reducing the haemodynamic response to the powerful stimulation of laryngoscopy and endotracheal intubation.

Subjects and Methods

It is Prospective Randomized Double Blind Study on 70 patients with approval of the Institutional ethics and scientific committee, present clinical comparative study of IV Lignocaine and IV Magnesium Sulphate in the attenuation of sympathetic response to laryngoscopy and intubation was done. General anesthesia with endotracheal intubation was provided for a duration of 12 months, Jan 2015 to Dec 2015**.** The study was done in General Operation Theatre Complex of the Tirumala Hospitals, Vizianagaram, Andhra Pradesh.

70 Patients scheduled for various elective surgical procedures, under general anaesthesia, belonging to ASA class I and II were included in the study. The hospital being a charitable & free hospital, the sample population of patients consisted of people from different parts of the country with varying socioeconomic status.

Sample size determination

Sample size formula for comparing two groups-

$N = 2 (Z_\alpha + Z_\beta)^2/(\mu 1 - \mu 2)2$

Where,

Z_α – Standardized score cutting off $\alpha/2$ proportion of each trail of a standard normal distribution.

Z_β – Standardized score cutting off the upper β proportion.

2 – Common variance of the two groups.

$\mu 1$-$\mu 2$ – difference in means of the two groups

By Mean Arterial Pressure values at first minute after intubation of a similar study by Navid Nooraei et al,[4] a sample size of 34 in each group were derived

MAP of Lignocaine group at 1^{st} min- 97.6±5.4

MAP of Magnesium group at 1^{st} min- 93.6±6.3

$N= 2(1.96-0.842)^2/(4/5.85)^2 \approx 34$ in each group.

The sampled patients were randomly allocated to either Lignocaine (Group A) or Magnesium(Group B) group by computer generated random numbers using Graph pad Software, http://www.graphpad.com/quickcalcs/randomize2.cfm

Inclusion Criteria

Patients aged between 20 to 60 years of both the sex of ASA physical status class I and II, Mallampatti grade I and II indergoing Elective surgeries under general anaesthesia.

Exclusion Criteria

Patients with hypertension, cardiac, coronary, renal, hepatic, cerebral diseases, peripheral vascular diseases and electrolyte imbalance, Patients with difficult airway and obese patients or those who require more than one attempt for laryngoscopy and intubation, patients with endocrinal disease like hypothyroidism, hyperthyroidism, diabetes mellitus, Pregnant and nursing women.

Preoperative assessment

Pre-anaesthetic evaluation was done on the evening before surgery. A routine preanaesthetic examination was conducted assessing and all basic investigaions done assessment and investigations. They were informed about the study and an informed consent was taken in all the patients. 70 cases were divided into two groups with 35 cases in each group.

Lignocaine group (Group A)

Patients received 1.5mg/kg of preservative free 2% lignocaine intravenously 90 seconds before laryngoscopy.

Magnesium group (Group B)

Patients received 30 mg/kg of magnesium 90 seconds prior to laryngoscopy.

The following cardiovascular parameters were recorded in all patients.

Heart rate [HR] in beats per minute.

Systolic blood pressure [SBP] in mm of Hg

Diastolic blood pressure [DBP] in mm of Hg

Mean blood pressure[MBP] in mm of Hg

The above cardiovascular parameters were monitored in the following time interval of Base line (before giving study drug), Post induction (Pre laryngoscopy)1,3,5 and 10 min after laryngoscopy and intubation

All the patients were pre oxygenated for 3 minutes with 100% oxygen. Induction was achieved Inj. Thiopentone sodium 5mg/kg i.v. given as a 2.5% solution and opioid analgesic Fentanyl 2mcg/kg. After checking the ability to ventilate, Inj.vecuronium was administered at a dose of 0.1mg/kg i.v. 3 minutes before laryngoscopy. Randomly selected 35 patients were given IV. Lignocaine 1.5mg/kg 90 seconds prior to laryngoscopy and 35 randomly selected patients were

given IV Magnesium sulphate 30mg/kg. The study solution was prepared in a 5 ml syringe by an anaesthesiologist who handed over the syringe in a coded form to the attending anaesthesiologist who records the parameters after injecting the drug. Laryngoscopy was done at the end of 3 mins after vecuronium injection using rigid laryngoscope with standard Macintosh blade. Intubation was done with appropriate sized, disposable, high volume low pressure cuffed endotracheal tube. Laryngoscopy and intubation was done by an experienced anesthesiologist in all cases. Heart rate, systolic and diastolic blood pressure were recorded post induction and at 1, 3, 5 and 10 minute intervals from the onset of laryngoscopy. Patients were connected to closed circuit and anesthesia was maintained with oxygen (50%), air (50%), isoflurane 1% and non-depolarizing muscle relaxant vecuronium bromide at a dose of 0.05 mg/kg i.v. and IPPV. Adequacy of ventilation was monitored by EtCO2 and SPO_2 was maintained at 99-100%. Positioning and surgery was withheld till the completion of recording upto 10 miniutes.

Statistical Methods

In the present study, descriptive statistical analysis was used. Continuous measurement findings are displayed as Mean SD (Min-Max), while categorical measurement results are presented as a number (percent). The significance is determined at a 5% level of significance. SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0, and R environment ver.2.11.1 were used to analyse the data, and Microsoft Word and Excel were used to create graphs, tables, and other images.

Results

The above table shows the age distribution in Lignocaine(Group A) and Magnesium(Group B) groups. The age range is 20-60 years in both the groups. There was no statistically significant difference between the groups with regard to age and so they are comparable. (p=0.66).

Table 1: Gender distribution of patients studied

Gender	Study groups		P-Value
	A	B	
Age in mean years	38.03 (12.84)	39.23 (9.46)	P=0.66
Male(number and percentages)	27 (77.1%)	25 (71.4%)	p= 0.58 (NS)
Female (number and percentages)	8 (22.9%)	10 (28.6%)	
Total	35	35	70

Samples are gender matched with P=0.58 In group A 77.1% of the patients are male and 22.9% are females. In group B 71.4% of the patients are males and 28.6% are females. Both the Groups are comparable with respect to gender.

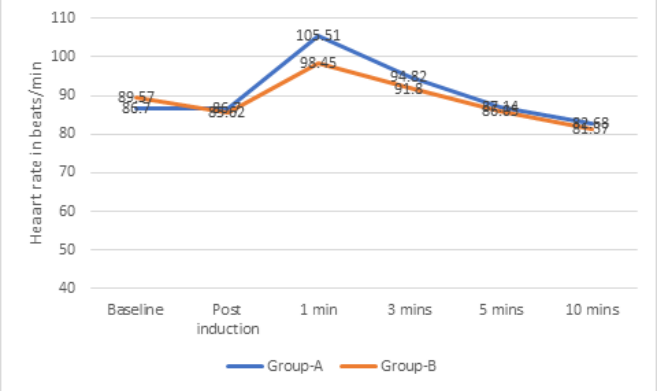

Figure 1: Comparison of Heart rate (bpm) in the groups studied.

observed after induction which was not significant statistically. Statistically significant increase in heart rate from baseline was observed only at 1 min after laryngoscopy and reached baseline by 5min. Although there was a statistically significant decrease in HR below baseline at 10 min after laryngoscopy, it was not clinically significant.

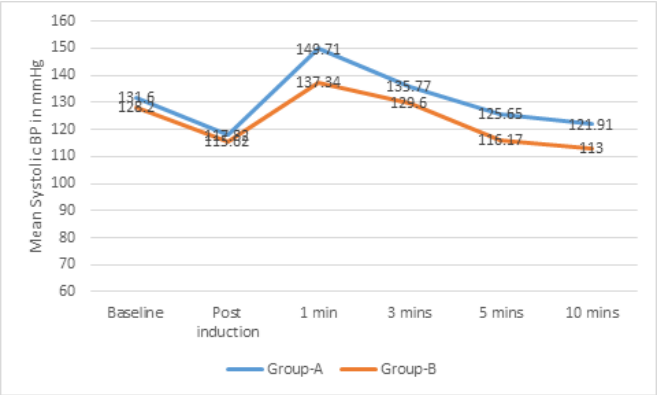

Figure 2: Comparison of Systolic Blood Pressure (SBP) (mm Hg) in the two groups studied.

Intergroup comparison between the two groups showed that the SBP at 1, 5 and 10 minute after laryngoscopy in magnesium group were statistically significantly lower compared to lignocaine group (P<0.05). At 3 minute SBP was lower in magnesium group but not statistically significant when compared with lignocaine group.

In Magnesium group the post induction DBP values was significantly lower compared to its baseline with P value of 0.02. At 1 minute there was slight increase in mean DBP

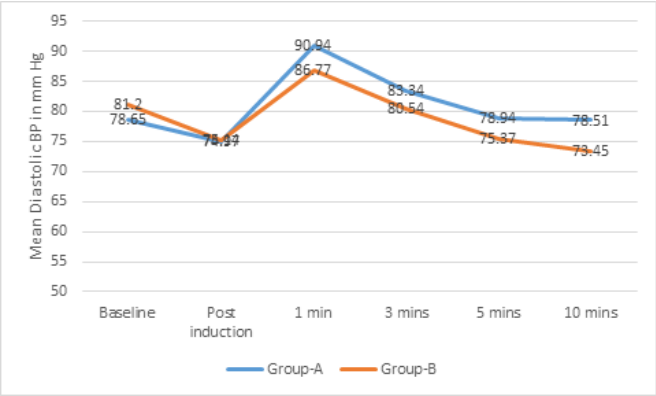

Figure 3: Comparison of Diastolic Blood Pressure (DBP) (mm Hg): Within group Analysis.

with P=0.03 which returned near baseline value by 3minute. At 5 min and 10 minutes values were significantly lower (P=0.02 and P=0.001 respectively) when compared to its baseline but this decrease was not clinically significant. Where as in Lignocaine group statistically significant increase in DBP compared to baseline was seen only at 1 minute after laryngoscopy with P value of <0.001.

Intergroup comparison between the two groups showed that the MBP at 1 minute after laryngoscopy in magnesium group was statistically significantly lower compared to lignocaine group (P=0.03). At 3, 5 and 10 minutes MBP was lower in magnesium group but not statistically significant when compared with lignocaine group.

Discussion

In many high-risk patients, the sequence of anaesthesia induction, laryngoscopy, and tracheal intubation is linked with significant haemodynamic alterations and autonomic reflex activation, which may be cause for worry. Intubation and laryngoscopy are linked to an increase in heart rate, blood pressure, and the occurrence of cardiac arrhythmias. Within 5 minutes of opening laryngoscopy, these potentially harmful changes vanish. Although these blood pressure and heart rate reactions are temporary and short-lived, they can be harmful in high-risk patients, particularly those with cardiovascular illness, elevated intracranial pressure, or cerebral blood vessel abnormalities.[5]

The cardiovascular changes related with laryngoscopy and intubation are influenced by a variety of variables. The pressor response is influenced by age, medicines, anaesthetic depth, hypoxia, hypercarbia, and other factors. As people get older, these heart rate differences become less noticeable. The changes in young patients are more pronounced. In geriatric patients, substantial changes in haemodynamic parameters are common. 5 We chose a 20-to-60-year-old age range for our study.

Antihypertensive medicines may cause a decrease in pressor response in patients. Patients taking antihypertensive drugs were excluded from the trial. The medicine must be effective regardless of patient cooperation, prevent cerebral blood flow disruption, and reduce patient arousal. It should neither be time intensive, nor should it have an impact on the duration and modality of the following anaesthetic. The above criteria appear to be met by intravenous bolus lignocaine and magnesium sulphate.

Lignocaine reduces an increase in intracranial pressure and an increase in intraocular pressure associated with laryngo-tracheal stimulation, in addition to attenuating cardiovascular reactions to laryngoscopy and intubation. Extubation-related cough is also suppressed. For many years, magnesium ions have been recognised to block catecholamine release from both the adrenal glands and peripheral adrenergic nerve terminals. It also causes immediate vasodilation. MgSO4 has been shown in numerous studies to reduce cardiovascular reactions to endotracheal intubation.[6]

Here an attempt has been made in this study to compare magnesium with lignocaine to find out whether it can be used as a suitable alternative to lignocaine or not. This clinical study is a randomized, prospective, double blind study, was done in 70 patients randomly allocated into two groups. Patients belonging to Group A received 1.5mg/kg of lignocaine intravenously 90 seconds prior to laryngoscopy and intubation. In Group B patients received intravenous magnesium sulphate 30mg/kg 90 seconds prior to laryngoscopy and intubation.

James F. Hamill and Robert Bedford F et al used similar dose of lignocaine and found it to be effective in attenuation of sympathetic responses of laryngoscopy and intubation, so dose of 1.5mg/kg of lignocaine was chosen in our study.[7]

In 2005, K. Montazeri et al and in 2013, Panda NB et al have done studies on optimal dose of magnesium sulphate for attenuation of hemodynamic response to laryngoscopy and intubation and concluded that 30mg/kg as optimal dose and smaller dose was less effective and larger doses resulted in complications.[8,9] So 30mg/kg of magnesium sulphate was chosen in our study.

Both the groups were well matched for demographic data and no statistically significant difference were found between groups with regard to age and gender.

In lignocaine group, the heart rate decreased from baseline after induction, which was not statistically significant (P=0.77) and there was statistically significant increase following intubation only at 1 min and 3 min (P<0.001 and P=0.002 respectively). At 5-minute heart rate was still above baseline and reached below baseline at 10 minute but these were not

Table 2: Comparison of Mean Blood Pressure (MBP) (mm Hg) in two groups studied.

MBP	Group	N	Mean	SD	Mean differ-	95% CI of difference		t	df	p-value
						Lower	Upper			
Baseline	B	35	100.42	9.19	3.20	-0.82	7.22	1.58	68	0.11
	A	35	97.22	7.60						
Post	B	35	91.17	8.13	2.05	-1.55	5.66	1.13	68	0.25
	A	35	89.11	6.94						
1 min	B	35	104.66	11.82	-6.02	-11.47	-0.56	-2.20	68	0.03*
	A	35	110.68	11.01						
3 min	B	35	97.86	8.82	-3.28	-7.73	1.15	-1.47	68	0.14
	A	35	101.15	9.77						
5 min	B	35	93.16	7.74	-0.30	-4.16	3.54	-0.15	68	0.87
	A	35	93.46	8.40						
10 min	B	35	90.67	7.76	0.76	-2.98	4.52	0.40	68	0.68
	A	35	89.90	7.95						

statistically significant (P=1.00 and P=0.44 respectively). Our findings are consistent with K. Montazeri et al,[8] who found that there was statistically significant increase in heart rate from baseline in lignocaine group at 1 and 3 minutes followed by statistically non-significant increase at 5 minute. Similar results were seen in study done by Navid nooraei et al,[4] with 1.5mg/kg of lignocaine. They found statistically significant increase in mean heart rate after 1 min (P=0.001) and at 2 min (P=0.027) followed by statistically non-significant increase at 3, 4 and 5 minutes (P= 0.68, P=0.51, P=0.579 respectively). Feng CK et al,[10]also in their study found that when 2mg/kg lidocaine was used to attenuate the hemodynamic response to laryngoscopy and intubation, heart rate values at 1 minute and 3-minute interval were statistically significantly above baseline (P<0.05).

In magnesium group the mean heart rate decreased from the baseline to post induction and it was statistically not significant (P=0.35). After intubation the mean heart rate increased at 1 and 3 minutes, however the increase in heart rate from baseline was statistically significant only at 1 min (P<0.001). It gradually reached the baseline by 5 minutes and was statistically significantly below baseline by 10 min (P=0.009) but this was not clinically significant. Our results correlated with the study done by K. Montazeri et al,[8] also noted a significant increase in heart rate from base line at 1 min followed by non-significant increase at 3 min and fall below baseline at 5 minute both of which were not statistically significant. In our study, heart rates at baseline and post induction were comparable in both lignocaine and magnesium group with P values of 0.41 and 0.46 respectively. At 1-minute heart rate increased in both lignocaine and magnesium group from baseline but, there was a greater increase in heart rate in the lignocaine group than magnesium group which was statistically significant with P=0.03. At 3-minute heart rate in both group were higher compared to baseline but it was not statistically significant (P=0.26). At 5 and 10 min interval heart rate in magnesium group was below baseline whereas heart rate in lignocaine group reached below baseline only at 10 minute. However this difference was not statistically or clinically significant (P=0.63 and P= 0.54 respectively). This was comparable to study done in 2005 by K. Montazeri et al,[8] who found that there was statistically significant difference in mean heart rates between magnesium and lignocaine groups at 1 minute after laryngoscopy (P<0.05). However in our study the difference in mean heart rates at 3 and 5 minutes in the lignocaine and magnesium groups was not statistically significant contrary to that reported by K. Montazeri et al,[8] who found statistically significant difference even at 3 and 5 minutes. This can be attributed to the fact that the above mentioned authors have evaluated the difference in heart rate as % change from baseline whereas we have calculated the p values taking into consideration the difference in heart rate between both the groups at each given time interval.

In lignocaine group, mean systolic blood pressure decreased from baseline after induction, which was statistically significant (P=0.004) but clinically not significant. There was statistically significant increase following intubation at 1 min P<0.001 followed by non-statistically significant increase at 3 min with P=1.00. At 5 minute and 10-minute systolic blood pressures were below baseline but statistically significant only at 10-minute P=0.002. Our results were consistent with study done by Navid nooraei et al,[4] who found a similar statistically significant increase in systolic blood pressure in lignocaine group at 1 min (P=0.0001), 2 min (P=0.001) from base-

line after intubation. Though the mean systolic blood pressure was above baseline at 5 min it was not statistically significant (P=0.582) which is in accordance with our study.

In magnesium group the mean systolic blood pressure decreased from the baseline to post induction and it was statistically significant (P<0.001) but not clinically significant. After intubation the systolic blood pressure increased at 1 and 3 minutes, this increase from baseline was statistically significant only at 1 min (P=0.008). It gradually reached the below baseline by 5 minutes and was statistically significantly below baseline by 5 min and 10 min (P=0.001 and P<0.001 respectively) however it was not clinically significant. Our results were consistent with study done by Navid nooraei et al in 2013,[4] who noted a similar statistically significant increase in systolic blood pressure in magnesium group only at 1 min (P=0.011) followed by a stable systolic blood pressure as compared to baseline. Other similar studies done by Yi-Ming et aland Stanley Tam et alfound similar significant increase of SBP only at 1 minute post intubation followed by stable SBP.[10,11] In our study, mean systolic blood pressure at baseline and post induction were comparable in both lignocaine and magnesium group with P values of 0.29 and 0.39 respectively. At 1-minute systolic blood pressure increased in both lignocaine and magnesium group from baseline but, there was a greater increase of systolic blood pressure in the lignocaine group than magnesium group which was statistically significant with P=0.002. At 3-minute systolic blood pressure in both group were higher compared to baseline but it was not statistically significant (P=0.10). At 5 and 10 min interval systolic blood pressure in both groups were statistically significantly below baseline (P=0.001 and P=0.001 respectively). However, this difference was not clinically significant. Our results were consistent with study done by Navid Nooraei et al in 2013,[4] who noted a similar statistically significant increase in blood pressure in lignocaine group when compared to magnesium group at 1 min (P=0.001) and 2 min (P=0.033) followed by non-significant difference between two groups at 3, 4 and 5 minutes.

In lignocaine group, mean diastolic blood pressure decreased from baseline after induction, which was not significant both statistically (P=0.036) and clinically. There was statistically significant increase following intubation at 1 min P<0.001 followed by statistically non-significant increase at 3 min and 5 min with P=0.08 and P=1.00 respectively. At 10 minute diastolic blood pressures were below baseline but statistically not significant P=1.00. Our results were consistent with study done by Navid nooraei et al in 2013,[4] who found similar statistically significant increase in diastolic blood pressure at 1 minute (P=0.046) followed by stable DBP.

In magnesium group the mean diastolic blood pressure decreased from the baseline to post induction and it was statistically significant (P=0.02) but not clinically significant. After intubation there was a statistically significant increase in the diastolic blood pressure at 1 minute with P=0.03. It gradually reached the below baseline by 3 minutes and was statistically significantly below baseline by 5 min and 10 min (P=0.02 and P=0.001 respectively) however it was not clinically significant. On the contrary, in the study done by Navid nooraei et al,[4] in 2013, they found no statistically significant increase in DBP from baseline at 1, 2, 3, 4 and 5 minutes. This may be attributed to the fact that they used 60 mg/kg of magnesium sulphate whereas we have used 30 mg/kg of magnesium sulphate. Moreover though statistically significant this increase in mean DBP from baseline was not clinically significant in our study. In our study, mean diastolic blood pressure at baseline and post induction were comparable in both lignocaine and magnesium group with P values of 0.21 and 0.92 respectively. At 1 minute and 3minute also there was no significant difference between two groups in diastolic blood pressure (P=0.08, P=0.09). The diastolic blood pressure reached baseline value by 3 minute in magnesium group whereas in lignocaine group it touched baseline only at 10 minute. At 5 and 10 min interval there was statistically significant difference in the mean diastolic blood pressure between lignocaine and magnesium group (P=0.02 and P=0.002 respectively). However this difference was not clinically significant. Our results were consistent with study done by Navid nooraei et al in 2013,[4] who also found no statistically significant difference in diastolic blood pressure at 1 and 3 minute following intubation among the two groups but contrary to our finding they also reported no statistically significant difference in the mean DBP between both groups even at 5 minute. In another similar study by Mehrdad Mesbah Kiaee et al,[12]who conducted study in 150 patients undergoing CABG receiving either magnesium sulphate 50mg/kg or lignocaine 1.5mg/kg randomly also showed statistically significant difference between lignocaine and magnesium groups in diastolic blood pressure only at 1 minute after intubation (P=0.018).

In lignocaine group, mean blood pressure decreased from baseline after induction, which was statistically significant (P=0.001) but clinically not significant. There was statistically significant increase following intubation at 1 min P<0.001 followed by statistically non-significant increase at 3 min with P=0.40. At 5 minute and 10-minute mean blood pressures were below baseline but statistically significant only at 10 minute P<0.001, however it was not clinically significant. Similarly in the study done by Navid nooraei et al in 2013,[4] they found statistically significant difference in mean blood pressure at 1 after intubation followed by no statistically significant difference in the mean blood pressure at 3, 4 and 5 minute. Our findings are consistent with K. Montazeri et al,[8] who found that there was statistically significant increase in mean blood pressure from baseline in lignocaine group at 1 minute followed by statistically non-significant increase at 5

minute.

In magnesium group the mean blood pressure decreased from the baseline to post induction and it was statistically significant (P<0.001) but not clinically significant. After intubation the mean blood pressure increased at 1 minute, this increase from baseline was neither statistically significant (P=0.43) nor clinically. It gradually reached the below baseline by 3 minutes and was statistically significantly below baseline by 5 min and 10 min (P=0.003 and P<0.001 respectively) however it was not clinically significant. Our results correlated with the study done by K. Montazeri et alin 2005,[8] who noted a statistically significant increase in mean blood pressure from base line only immediately after intubation at 1 min followed by non-significant increase at 3 min and fall below baseline at 5 minute both of which were not statistically significant. Similarly in the study done by Navid nooraei et al in 2013,[4] they found statistically significant difference in mean blood pressure only at 1 after intubation followed by no statistically significant difference in the mean blood pressure at 2, 3, 4 and 5 minute.

In our study, mean blood pressures at baseline and post induction were comparable in both lignocaine and magnesium group with P values of 0.11 and 0.25 respectively. At 1-minute mean blood pressure increased in both lignocaine and magnesium group from baseline but, there was a greater increase of mean blood pressure in the lignocaine group than magnesium group which was statistically significant with P=0.03. At 3 minute mean blood pressure in both group were higher compared to baseline but it was not statistically significant (P=0.14). At 5 and 10 min interval mean blood pressure in both groups were below baseline but not significant clinically or statistically (P=0.87 and P=0.68 respectively). This was comparable to study done by Navid nooraei et al in 2013,[4] where the Mean blood pressure in the first and second minutes after intubation was significantly lower in the magnesium group compared to lignocaine group with P values of 0.012 and 0.04 respectively. Followed by non-statistically significant increase at 3, 4 and 5minutes. Another similar study conducted in 2005 by K. Montazeri et al,[8] who found that there was statistically significant difference between magnesium and lignocaine groups in mean blood pressure at 1 and 3 minutes after laryngoscopy (P<0.05 and P<0.05 respectively) showing that mean blood pressure was better controlled in magnesium group. In another similar study by Mehrdad Mesbah Kiaee et al,[12]who conducted study in 150 patients undergoing CABG receiving either magnesium sulphate 50mg/kg or lignocaine 1.5mg/kg randomly also showed similar statistically siginificant difference between lignocaine and magnesium groups in mean blood pressure after intubation (P=0.049) only at 1 minute.

Percent rise in the HR, SBP, DBP, and MAP were significantly less in Group B(magnesium) compared to Group A(lignocaine). Thus overall, from the present study it was seen that when $MgSO_4$ 30mg/kg given intravenous 90 seconds prior to laryngoscopy and intubation for attenuation of pressor response results in significant reduction in HR, SBP, DBP and MAP at 1 min after laryngoscopy and intubation and no significant changes after 3 min when compared to intravenous lignocaine 1.5mg/kg.

Conclusion

Intravenous lignocaine and intravenous magnesium sulphate both significantly attenuates the sympathetic response to laryngoscopy and tracheal intubation. Intravenous magnesium sulphate at 30mg/kg is more efficient than lignocaine 1.5mg/kg in attenuating the sympathetic response to laryngoscopy and intubation. Hence we conclude that intravenous magnesium sulphate 30mg/kg 90 seconds prior to laryngoscopy and intubation is superior to lignocaine 1.5mg/kg prior to laryngoscopy and intubation.

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