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Original Research Article

A Hospital Based Comparative Clinical Assessment of Intravenous Magnesium Sulphate and Lignocaine in Attenuation of Pressor Response to Laryngoscopy and Endotracheal Intubation

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Conflict of interest: Nil

Abstract

Aim: This study was undertaken with the objective of comparing the effectiveness of magnesium sulphate and lignocaine for attenuation of pressor response during laryngoscopy and intubation at dosages of 30 mg/kg and 1.5 mg/kg respectively.

Methods: This study was undertaken in Department of Anesthesiology, Sri Krishna Medical College & Hospital, Muzaffarpur, Bihar and the duration of study was 1 years. Institutional Ethical committee clearance was obtained for the study. Informed written consent was obtained from all patients. 100 ASA 1 patients in age group of 18-40 years of either gender scheduled for elective surgeries under general anaesthesia with endotracheal intubation.

Results: There was no statistically significant difference between the groups with regard to demographic parameters. There was no significant change in HR, immediately after intubation, in both MgSO4 and lignocaine groups. In lignocaine group, at intubation, there was a rise in SBP from baseline which was not significant. Rise in DBP was seen at intubation with greater rise in group M but it was neither clinically nor statistically significant. There was an increase in MAP immediately after intubation, which wasn't significant.

Conclusion: MgSO4 30 mg/kg given intravenously as infusion over 10 minutes prior to induction and lignocaine 1.5 mg/kg given 90 seconds before intubation were comparable in attenuating pressor response to laryngoscopy and intubation with no clinically significant prolongation in time taken to extubate in MgSO4 group.

Keywords: Magnesium sulphate, lignocaine, intubation response, laryngoscopy.

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 block peripheral sensory receptors 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, anti-hypertensive drugs such phentolamine, sodium nitroprusside, nitro-glycerine, 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]

Lignocaine has been used both as surface anaesthetic and also by intravenous route to depress haemodynamic response to intubation. [4] Lignocaine, when used systemically, has antagonistic action on sodium channels and NMDA receptors, reduces the release of substance P, has glycinergic action, which decreases the airway reactivity. [5] Magnesium attenuates hemodynamic response by inhibition of catecholamine release from the adrenal medulla and also by reduction of the increased circulating norepinephrine when compared to that of a control group. [6] It also has a systemic and coronary vasodilation effect by antagonizing calcium ion in vascular smooth muscle. [7]

This study was undertaken with the objective of comparing the effectiveness of magnesium sulphate and lignocaine for attenuation of pressor response during laryngoscopy and intubation at dosages of 30 mg/kg and 1.5 mg/kg respectively.

Materials and Methods

This study was undertaken in Department of Anesthesiology, Sri Krishna Medical College & Hospital, Muzaffarpur, Bihar, India and the duration of study was 1 year. Institutional Ethical committee clearance was obtained for the study. Informed written consent was obtained from all patients.

100 ASA 1 patients in age group of 18-40 years of either gender scheduled for elective surgeries under general anaesthesia with endotracheal intubation. Patients were randomly allocated by computer generated randomisation table into two groups with sample size of 30 each. Group L (n =50) received preservative free 2% lignocaine 1.5 mg/kg

intravenously 90 seconds before intubation. Group M (n=50) received 30 mg/kg of magnesium sulphate in 100ml normal saline (NS), intravenously over 10 minutes before induction.

Patients allergic to or having any contraindications to the study drugs, anticipated difficult airway and emergency surgical procedures requiring rapid sequence induction were excluded from the study. On the day prior to surgery, adetailed pre-anaesthetic evaluation and routine pre-operative investigations were done.

Demographic (age, gender, weight) and vital parameters were recorded. All patients were pre-medicated with Tab. Alprazolam 0.25 mg and Tab. Ranitidine 150 mg on the night before the day of surgery and morning of surgery. On arrival in the operating room, basal parameters-heart rate(HR), systolic blood pressure(SBP), diastolic blood pressure(DBP), mean arterial pressure(MAP), baseline ECG and oxygen saturation were recorded. These parameters were recorded at induction and every minute thereafter till 10 mins post intubation.

The study drugs were administered by an anaesthesiologist (observer 1). Ten minutes before induction, patients in Group M received 30mg/kg of MgSO4 in 100ml saline while those in group L received 100ml plain saline over 10 mins. After pre-oxygenation for 3 min, all patients received IV midazolam 0.05 mg/kg, fentanyl 2mcg/kg and propofol 2mg/kg. After confirming adequacy of mask ventilation, vecuronium 0.1mg/kg was given IV. Ninety seconds before intubation, patients in Group L received preservative free2% lignocaine 1.5mg/kg diluted to 5ml with saline and patients in GroupM received 5ml plain saline. A consultant anaesthesiologist (Observer 2) did a gentle brief laryngoscopy and intubated the patient with appropriate sized cuffed endotracheal tube in all patients. The tube was secured after confirming proper placement. HR, SBP, DBP & MAP were recorded at induction and at every minute thereafter till 10 minutes after intubation. Any adverse effects were observed for and treated accordingly. Anaesthesia was maintained with 33% oxygen and 66% nitrous oxide (1:3) with sevoflurane titrating to maintain MAC of 1.3 and intermittent boluses of vecuronium (0.02 mg/kg). At the end of surgery, inhalational agents were turned off. Residual neuromuscular blockade was reversed with neostigmine 50 mcg/kg and glycopyrrolate 10 mcg/kg slow IV. Patients were extubated when awake and had adequate spontaneous respiratory efforts. The time taken to extubate, from turning off the inhalational agents to extubation, was recorded in both groups.

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean± standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries and diagrammatic presentation of the same was made. The difference in the means of analysis variables between two independent groups was tested by unpaired t test. If the p-value was < 0.05, then the results were considered to be statistically significant. Data were analyzed using SPSS software v.23.0 and Microsoft office 2007. There were no dropouts from the study or major complications.

Results

Table 1: Distribution of Demographic parameters between study groups

Parameters	Group L	Group M	p value
Age (yrs)	29.8±5.5	31.6±7	0.250
Height (cm)	157.3±8.9	157.3±3.7	0.235
Weight (Kg)	60.3±9.1	60.1±9.6	0.925

There was no statistically significant difference between the groups with regard to demographic parameters.

Table 2: Comparison of change in HR over time between study groups

HR	Group L	Intra group p value from basal	Group M	Intra group p value from basal	Between group Comparison p value
Basal	87.3±17.7	-	93.7±15.5	-	0.359
At Induction					
0 Minute	84.6±14	0.050	87.4±12.8	0.020	0.424
1 Minute	83.7±13.7	0.052	83.7±12	<0.001	0.745
2 Minute	83.1±13.3	0.025	80±12.8	<0.001	0.376
3 Minute	82.1±13.9	0.020	80.4±13.7	<0.001	0.525
After Intubation					
0 Minute	88.5±16.4	0.510	88.5±10.6	0.250	0.824
1 Minute	87±14.5	0.765	86.4±11.9	0.055	0.826
2 Minute	85.5±14.6	0.330	83.6±12.8	0.010	0.865
3 Minute	83.7±16.4	0.120	84±11	0.007	0.820
4 Minute	82.8±14.6	0.055	82.8±12.7	0.001	0.715
5 Minute	81.9±15	0.032	81.9±11.9	0.001	0.935
6 Minute	81.4±15.5	0.034	80.5±12.7	<0.001	0.756
7 Minute	81±15.5	0.022	79.7±12.6	<0.001	0.610
8 Minute	79.1±15.4	0.009	76.5±12	<0.001	0.450
9 Minute	78.2±14.6	0.006	75.9±12.2	<0.001	0.445
10 Minute	79.1±14.6	0.010	76.5±9.5	<0.001	0.389

There was no significant change in HR, immediately after intubation, in both MgSO4 and lignocaine groups. In lignocaine group, there was a decrease in HR after 5 mins post intubation as compared to 2 mins post intubation in MgSO4 group, which wasn't clinically significant though there was statistically significant difference.

Table 3: Comparison of change in SBP over time between study groups

SBP	Group L	Intra group p value from basal	Group M	Intra group p value from basal	Between group comparison p value
Basal	127.3±11.9	-	127.5±10.5	-	0.910
At Induction					
0 Minute	110.6±9.8	<0.001	110.5±10.9	<0.001	0.885
1 Minute	103.9±12.2	<0.001	105.5±12.8	<0.001	0.555
2 Minute	101.6±10.8	<0.001	101.9±11.9	<0.001	0.865
3 Minute	101.9±10.4	<0.001	100.9±12.8	<0.001	0.767
After Intubation					
0 Minute	127.1±14.9	0.510	127±16.4	0.620	0.975
1 Minute	116±13.7	<0.001	116.4±12.8	<0.001	0.940
2 Minute	108.2±12.8	<0.001	111.9±10.0	<0.001	0.354
3 Minute	106.4±12.8	<0.001	107.3±11.9	<0.001	0.689
4 Minute	103.7±12.1	<0.001	105.5±12.8	<0.001	0.420

5 Minute	101±11.9	<0.001	104.6±13.7	<0.001	0.230
6 Minute	101.4±12	<0.001	104.4±12.8	<0.001	0.330
7 Minute	101.9±13.7	<0.001	105.5±11.9	<0.001	0.240
8 Minute	102.8±11.5	<0.001	104.6±13.7	<0.001	0.600
9 Minute	103.7±11.1	<0.001	102.8±13.7	<0.001	0.860
10 Minute	106.4±10.6	<0.001	105.5±12.8	<0.001	0.872

In lignocaine group, at intubation, there was a rise in SBP from baseline which was not significant. From One min post intubation, statistically significant decline in SBP was seen in both the groups, which was within the acceptable clinical limits.

Table 4: Comparison of change in DBP over time between study groups

DBP	Group L	Intra group p value from basal	Group M	Intra group p value from basal	Between group comparison p value
Basal	84.6±9.1	-	84.1±10.1	-	0.735
At Induction					
0 Minute	72.8±11	<0.001	71.7±11	<0.001	0.856
1 Minute	68.2±12.8	<0.001	67.2±10.8	<0.001	0.609
2 Minute	65.7±9.5	<0.001	62.7±11.4	<0.001	0.267
3 Minute	66.9±12.5	<0.001	66.6±14.6	<0.001	0.924
After Intubation					
0 Minute	82.2±14.3	0.320	85.5±12.8	0.45	0.309
1 Minute	75.5±13.4	0.002	76.6±11.4	0.001	0.734
2 Minute	70.5±11.3	<0.001	74.6±10.9	<0.001	0.165
3 Minute	68.2±14.6	<0.001	71.9±9.5	<0.001	0.478
4 Minute	66.4±11.9	<0.001	68.9±11.9	<0.001	0.376
5 Minute	65.5±11.8	<0.001	68.2±13	<0.001	0.346
6 Minute	65.5±12.1	<0.001	67.3±11.9	<0.001	0.546
7 Minute	67±11.7	<0.001	70.5±13.7	<0.001	0.235
8 Minute	67.3±9.2	<0.001	68.1±13.9	<0.001	0.820
9 Minute	67.9±9.1	<0.001	67.3±13.7	<0.001	0.965
10 Minute	69.1±9.7	<0.001	68.2±11.9	<0.001	0.935

Rise in DBP was seen at intubation with greater rise in group M but it was neither clinically nor statistically significant. One min post intubation, there was a decrease in DBP, in both the groups (within the group) upto 10th minute while it was not significant between the groups.

Table 5: Comparison of change in MAP over time between study groups

MAP	Group L	Intra group p value from basal	Group M	Intra group p value from basal	Between group comparisonp value
Basal	99.7±9.4	-	97.9±12	-	0.515
At Induction					
0 Minute	86.4±9.1	<0.001	84.4±9.5	<0.001	0.489
1 Minute	81.9±11	<0.001	80±11.2	<0.001	0.517
2 Minute	78.2±8.4	<0.001	75.6±11.2	<0.001	0.245

3 Minute	80.5±11.3	<0.001	77.8±13.3	<0.001	0.428
After Intubation					
0 Minute	98.2±13	0.629	99.1±13.4	0.525	0.868
1 Minute	90.6±12.5	<0.001	89.1±11.6	0.002	0.739
2 Minute	83.7±11.9	<0.001	87±9.8	<0.001	0.240
3 Minute	83.7±11.8	<0.001	84.6±10.0	<0.001	0.732
4 Minute	79.1±10.8	<0.001	81.9±11.9	<0.001	0.412
5 Minute	78.2±11.6	<0.001	81.9±12.4	<0.001	0.310
6 Minute	79.1±11.9	<0.001	80.8±11.8	<0.001	0.630
7 Minute	79.9±11.5	<0.001	83.7±12.8	<0.001	0.254
8 Minute	80.8±9.9	<0.001	81.9±14.1	<0.001	0.924
9 Minute	82.8±10.2	<0.001	80.1±13.6	<0.001	0.520
10 Minute	83.7±10	<0.001	82.1±11	<0.001	0.645

MAP in both the groups followed similar trend. There was an increase in MAP immediately after intubation, which wasn't significant. After 1 min post intubation, statistically significant fall in MAP was seen in both the groups which was clinically acceptable.

Discussion

Endotracheal intubation is an essential component of general anesthesia. It serves in the maintenance of patency of upper airway, proper ventilation, reduction in the risk of aspiration, and delivery of the inhalational anesthetic agents to the patients through breathing circuits. [8] Laryngoscopy and tracheal intubation are considered the most critical events during induction of general anesthesia which stimulate somatic and visceral nociceptive afferents fibers which induce reflex sympato-adrenal responses associated with enhanced neuronal activity in the cervical sympathetic efferent fibers. [9] Sympathetic stimulation from laryngoscopy and endotracheal intubation causes a significant increase in the plasma concentration of catecholamines (adrenaline and noradrenaline) [10,11] that can provoke left ventricular failure, renal failure, surgical bleeding, cerebral hemorrhage and myocardial ischemia in anesthetized patients.

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 laryngotracheal 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. [12]

In our study, in lignocaine group, there was rise in HR after an initial fall post induction, nearing baseline value at intubation but it was neither statistically nor clinically significant. From 5th minute post intubation decrease in HR continued throughout till 10th minute which was statistically significant but clinically insignificant. Thus, in our study, we noted that lignocaine attenuated HR response

post laryngoscopy and intubation. In a study done by Panda et al [13] where lignocaine 1.5 mg /kg was used 90 seconds before intubation, similar trend of HR was seen. HR was seen reaching the baseline value at intubation followed by a decreasing trend till 10th minute post intubation. In a study by Nooraei et al. [7] where lignocaine 1.5 mg/kg used 3 minutes prior to intubation, HR showed a statistically significant rise following intubation in initial 3 minutes and reached basal value in 4th and 5th minute which was statistically insignificant.

In a study by Mahajan et al. [14] where 30 mg/kg of MgSO4 was used 15 minutes prior to induction, HR showed highly significant ($p<0.001$) fall compared to predrug value and remained significantly lower for 30 minutes post intubation. Mendonca et al. [15] used 30 mg/kg of MgSO4 10 minutes prior to induction and HR had increasing trend post drug infusion which tended to decrease at induction and rise at laryngoscopy followed by falling trend till 6th min post 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. [16] 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. [17] 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 demographic parameters.

In the lignocaine group of our study, at intubation, SBP was near the baseline value. Statistically significant decrease in SBP was seen from 1st minute till 10th minute post intubation. DBP and MAP also showed similar trend, in that, values were near baseline at the time of intubation followed by statistically significant decrease till 10th minute post intubation, though all were within clinically acceptable limits. In the study by Panda et al.13 lignocaine 1.5 mg/ kg was given 90 seconds before intubation. They found that SBP, DBP and MAP decreased after induction of anesthesia with an increase towards baseline immediately after intubation. MAP showed a significant decrease as compared with baseline from 3rd minute post intubation which continued till 10th minute. Trend of response was similar to the one in our study.

Statistically significant increase in SBP was seen from 1 to 3 mins post intubation. At 4th and 5th minute, SBP was higher than baseline but statistically insignificant. Increase in DBP was seen but not statistically significant. Increase in MAP showed an increase in 1st and 2ndminutes which were statistically significant but in 3rd, 4th and 5th minutes this difference was not statistically significant despite higher values of MAP. In the study by Padmawar et al. [18] lignocaine 1.5mg/kg was administered prior to induction of anesthesia. SBP increased at 1st minute post intubation. DBP and MAP also increased post intubation. The rise in SBP post intubation was statistically significant and remained so till 5th minute post intubation. In the study by Bhalerao et al. [19] where 1.5 mg/kg of lignocaine was administered 90 seconds prior to intubation, MAP, SBP and DBP showed increase towards baseline at intubation which was not statistically significant. There was a statistically significant decrease in MAP from 4th minute till 10th minute post intubation.

Thus the effect on SBP, DBP and MAP in our study was similar to the studies by Panda et al. [13], Mendonca et al. [15] and

Bhalerao et al. [19] But lignocaine did not attenuate BP responses well in studies by Nooraei et al. [7] and Padmawar et al. [18] This difference of result in studies by Nooraei et al. [7] and Padmawar et al. [18] in comparison to our study could be attributed to timing of lignocaine administration where lignocaine was administered 3 minutes prior or at induction of anesthesia. In MgSO4 group of our study, at intubation, SBP neared the baseline value which gradually decreased and remained low till 10th minute post intubation. This decrease at all points remained statistically significant but was not clinically significant requiring intervention. DBP and MAP also decreased at induction which continued for 3 minutes post induction with rise nearing basal value at intubation followed by a decrease from 1st minute till 10th minute post intubation which was clinically insignificant but statistically significant.

In study by Panda et al. [13] SBP, DBP and MAP of group using 30 mg/kgof magnesium sulphate in 100ml NS over 10 minutes showed decrease after induction, with an increase towards baseline immediately after intubation. But this increase was not significant when compared to baseline. MAP was well maintained after intubation throughout the study period. No patient required any intervention to manage hypotensive episodes when compared to groups using 40 mg/kg and 50 mg/kg. MAP remained low till 10th minute post intubation. In study by Mahajan et al. [14] 30 mg/kg was given as infusion 15 min prior to induction. SBP showed a significant reduction in mean value. SBP continued to remain low even at intubation and 30 minute post intubation which were statistically significant at all points. DBP also followed a similar trend.

In our study Magnesium sulphate 30 mg/kg and lignocaine 1.5 mg/kg had comparable attenuation of response on HR, SBP, DBP and MAP with none of them needing any intervention for management of hypotension or any adverse effects. In study by Mendonca et al. [15] where 30 mg/kg MgSO4 was compared with 2 mg/kg of lignocaine they concluded that lower dose of MgSO4 was sufficient to attenuate haemodynamic response with results similar to lignocaine. In the study by Panda et al.13 where 3 different doses 30 mg/kg, 40 mg/kg and 50 mg/kg of MgSO4 were compared with lignocaine 1.5 mg/kg observed that 30 mg/kg was optimal dose maintaining better haemodynamic stability than lignocaine with higher doses of MgSO4 causing fall in blood pressure requiring intervention. In the study by Bhalerao et al. [19] 50 mg/kg of MgSO4 was better than lignocaine 2 mg/kg but required interventions to manage hypotension which was not seen with lignocaine. [20]

Conclusion

Magnesium sulphate 30 mg/kg given IV as infusion over 10 minutes prior to induction of anaesthesia was comparable to lignocaine 1.5 mg/kg given IV 90 seconds before intubation in attenuating haemodynamic response to laryngoscopy and intubation with no adverse effects. Though time taken to extubate was statistically significant in MgSO4 group, it was clinically insignificant. Thus Magnesium sulphate 30 mg/kg IV is a safe alternative to lignocaine with no adverse effects.

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