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Comparison of magnesium sulphate with lignocaine for blunting response to laryngoscopy andintubation

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Abstract

Background: Haemodynamic changes occurring during direct laryngoscopy and endotracheal intubation are well tolerated by healthy individuals but can be fatal in patients with hypertension, heart disease and intracranial hypertension. Many methods have been tried to obtund these responses.
Methods: 60 consenting patients were randomised to receive 30 mg/kg of magnesium sulphate (MgSO4) in 100 ml saline over 10 min before induction or preservative free 2% lignocaine 1.5 mg/kg diluted to 5 ml with saline 90 secs before intubation. Heart rate, systolic, diastolic & mean blood pressures and time taken to extubate were monitored.
Results: Hemodynamic parameters showed no significant rise at intubation in both the groups. Time taken to extubate was similar in both the groups.
Conclusion: $MgSO_4$ 30 mg/kg given intravenously as infusionover 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 $MgSO_4$ group.

Keyword: Magnesium sulphate, lignocaine, intubation response, laryngoscopy

Introduction

One of the commonest modes of securing airway in general anaesthesia is endotracheal intubation. Direct laryngoscopy and endotracheal intubation are noxious stimuli and almost always associated with haemodynamic changes due to reflex sympathetic stimulation caused by laryngo-pharyngeal stimulation [1].Though these changes are usually well tolerated by healthy individuals they can be fatal in patients with hypertension, coronary artery disease and intracranial hypertension. Many pharmacologicalmeasures have been used to obtund these responses.

Lignocaine has been used both as surface anaesthetic and also by intravenous route to depress haemodynamic response to intubation[2].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 [3].

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[4]. It also has a systemic and coronary vasodilation effect by antagonizing calcium ion in vascular smoothmuscle [5].

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

Materials and Methods

This study was undertaken in ESIC-MC & PGIMSR hospital, Rajajinagar, Bengaluru during 1st January 2019 to 30th June 2020. Institutional Ethical committee clearance was obtained for the study. Informed written consent was obtained from all patients.

A sample size of 60 (with 30 in each group) was calculated taking previous study done byMendonca*et al*.[6] as reference with confidence interval 95%, power of the study 80% andvariance 10%, with 20% effect difference.

Sixty 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 =30) received preservative free 2% lignocaine 1.5 mg/kg intravenously 90 seconds before intubation. Group M (n=30) 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, anticipateddifficult airway and emergency surgical procedures requiring rapid sequence inductionwere 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 $MgSO_4$ in 100ml saline while those in group L received 100ml plain salineover 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/kgdiluted to 5ml with salineand 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 wererecorded 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 and66% 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 wasreversed with neostigmine 50 mcg/kg and glycopyrrolate 10 mcg/kg slow IV.Patients wereextubated 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)	30.8±6.4	32.7±7	0.295
Height (cm)	158.9±8.6	156.8±3.6	0.231
Weight (Kg)	59.3±9.1	59.1±9.6	0.912
Male to female ratio	1:1.5	1:0.6	0.071

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 comparisonp value
Basal	87.7±17.4	-	91.7±15.6	-	0.352
At Induction					
0 Minute	84.2±14	0.055	86.7±12.2	0.021*	0.463
1 Minute	83.8±13.4	0.056	82.6±12	<0.001*	0.709
2 Minute	83.2±13.6	0.026*	80±12.6	<0.001*	0.353
3 Minute	83.1±14.9	0.020*	80.9±13.8	<0.001*	0.554
After Intubation					
0 Minute	89.5±16.8	0.509	88.8±10.9	0.267	0.856
1 Minute	87±14.6	0.791	86.3±11.8	0.053	0.831
2 Minute	85.1±14.7	0.318	84.5±12.5	0.011*	0.873
3 Minute	83.5±16.4	0.140	84±11	0.008*	0.890
4 Minute	82.4±14.6	0.059	81.3±12.7	0.001*	0.756
5 Minute	81.9±15	0.033*	81.5±11.8	0.001*	0.909
6 Minute	81.4±15.5	0.039*	80.3±12.6	<0.001*	0.763
7 Minute	81±15.3	0.024*	79.2±12.5	<0.001*	0.620
8 Minute	79.6±15.3	0.009*	76.9±12	<0.001*	0.454
9 Minute	78.6±14.8	0.006*	75.9±12.2	<0.001*	0.450
10 Minute	79.4±14.6	0.012*	76.6±9.5	<0.001*	0.392

Note: * significant at 5% level of significance (p<0.05)

There was no significant change in HR, immediately after intubation, in both $MgSO_4$ 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	128.8±11.2	-	128.5±10.7	-	0.916
At Induction					
0 Minute	110.6±9.8	<0.001*	110.9±10.1	<0.001*	0.897
1 Minute	103.9±12.2	<0.001*	105.9±12.4	<0.001*	0.539
2 Minute	101.6±10.8	<0.001*	101.1±11.6	<0.001*	0.872
3 Minute	101.9±10.4	<0.001*	100.9±12.3	<0.001*	0.752
After Intubation					
0 Minute	127.1±14.9	0.524	127±16.7	0.608	0.981
1 Minute	116±13.6	<0.001*	116.3±12.9	<0.001*	0.938
2 Minute	108.7±12.7	<0.001*	111.6±10.7	<0.001*	0.333
3 Minute	106.4±12.8	<0.001*	107.7±11.1	<0.001*	0.675
4 Minute	103.1±12.1	<0.001*	105.8±12.3	<0.001*	0.407
5 Minute	101±11.7	<0.001*	104.8±13.4	<0.001*	0.246
6 Minute	101.4±12	<0.001*	104.4±12.6	<0.001*	0.349
7 Minute	101.6±13.4	<0.001*	105.3±11.2	<0.001*	0.241
8 Minute	102.6±11.5	<0.001*	104.4±13.8	<0.001*	0.600
9 Minute	103.4±11.1	<0.001*	102.9±13.6	<0.001*	0.876
10 Minute	106.1±10.6	<0.001*	105.6±12.6	<0.001*	0.877

Note: * significant at 5% level of significance ($p<0.05$)

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 comparisonp value
Basal	84.9±9.6	-	84.1±10.1	-	0.755
At Induction					
0 Minute	72.1±11	<0.001*	71.7±11	<0.001*	0.870
1 Minute	68.8±12.2	<0.001*	67.2±10.8	<0.001*	0.602
2 Minute	65.7±9.5	<0.001*	62.7±11.4	<0.001*	0.273
3 Minute	66.9±12.5	<0.001*	66.6±14.6	<0.001*	0.925
After Intubation					
0 Minute	82.2±14.3	0.314	85.8±12.3	0.41	0.304
1 Minute	75.5±13.4	0.002*	76.6±11.4	0.001*	0.749
2 Minute	70.5±11.3	<0.001*	74.8±10.9	<0.001*	0.139
3 Minute	68.9±14.2	<0.001*	71.2±9.5	<0.001*	0.471
4 Minute	66.4±11.4	<0.001*	68.9±11.2	<0.001*	0.388
5 Minute	65.3±11.8	<0.001*	68.1±13	<0.001*	0.375
6 Minute	65.9±12.1	<0.001*	67.9±11.8	<0.001*	0.521
7 Minute	67±11.7	<0.001*	70.8±13.5	<0.001*	0.242
8 Minute	67.5±9.2	<0.001*	68.1±13.9	<0.001*	0.835
9 Minute	67.9±9.1	<0.001*	67.9±13.2	<0.001*	0.991
10 Minute	69.1±9.7	<0.001*	68.9±11.1	<0.001*	0.951

Note: * significant at 5% level of significance ($p<0.05$)

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 10^{th} 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	-	0.522
At Induction					
0 Minute	86.1±9.4	<0.001*	84.4±9.5	<0.001*	0.498
1 Minute	81.7±11	<0.001*	80±11.2	<0.001*	0.571
2 Minute	78.6±8.4	<0.001*	75.6±11.2	<0.001*	0.234
3 Minute	80.3±11.3	<0.001*	77.8±13.3	<0.001*	0.424
After Intubation					
0 Minute	98.7±13	0.612	99.3±13.4	0.576	0.861
1 Minute	90.6±12.5	<0.001*	89.5±11.6	0.002*	0.732
2 Minute	83.8±11.1	<0.001*	87±9.8	<0.001*	0.241
3 Minute	83.2±11.8	<0.001*	84.1±10.2	<0.001*	0.735
4 Minute	79.5±10.8	<0.001*	81.9±11.6	<0.001*	0.417
5 Minute	78.4±11.6	<0.001*	81.6±12.4	<0.001*	0.306
6 Minute	79.3±11.9	<0.001*	80.8±11.8	<0.001*	0.626
7 Minute	79.9±11.5	<0.001*	83.5±12.7	<0.001*	0.254
8 Minute	80.8±9.9	<0.001*	81.1±14.1	<0.001*	0.924
9 Minute	82.2±10.2	<0.001*	80.1±13.6	<0.001*	0.515
10 Minute	83.3±10	<0.001*	82.1±11	<0.001*	0.651

Note: * significant at 5% level of significance (p<0.05)

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.

Table 6: Time taken to extubate between Study Groups

Parameters	Group L	Group M	p value
Time taken to extubate in min	6.9±1.2	8.3±2.3	0.006*

Note: * significant at 5% level of significance (p<0.05)

Time taken to extubate in MgSO4 group was longer than lignocaine group by 1.4 minutes, which was statistically significant (p value 0.006) but clinically insignificant.

Discussion

During direct laryngoscopy, proprioceptors are stimulated by the pressure exerted at the base of tongue which results in increase in heart rate, blood pressure and increase in plasma catecholamine concentration.[1] Passage of endotracheal tube was found to further exaggerate this response by somato-visceral reflex followed by rapid regression of SBP and HR while plasma catecholamine concentration regress more slowly. Reflex sympathetic response produced during laryngoscopy and intubation are usually well tolerated in healthy individuals but can be fatal in patients with ischemic heart disease, hypertension, raised intracranial pressure. Therefore it is important to take measures to attenuate the pressor response during laryngoscopy and intubation.

We used lignocaine 2% and magnesium sulphate 50% in our study.

Lignocaine has been used in various routes such as gargle, spray, intravenous, nebulization.[7,8,9] Lignocaine acts by NMDA receptor antagonism, sodium channel antagonism and suppression of atrioventricular nodal activity.[3] Intravenous lignocaine has its onset in 1-2 minutes with duration of action for 1 hour. It has proved ideal for control of short lived haemodynamic sequelae, associated with laryngoscopy and intubation.[10] There are various studies with lignocaine being used intravenously to study its effect on pressor response during intubation.

In a study conducted by Abou madi *et al*.[11]intravenous lignocaine was used in doses of 0.75 mg and 1.5 mg per kg and cardiovascular response pre and post intubation were compared. It was found that in group where 0.75 mg per kg lignocaine was used DBP and HR showed significant rise. In group where 1.5 mg per kg was used, changes in BP & HR were statistically not significant. Stoelting RK[7] confirmed that 1.5 mg per kg of lignocaine given 90 seconds prior to intubation was safe. In our study we used 2% lignocaine 1.5 mg per kg intravenously, 90 seconds prior to intubation.

Magnesium is a non-competitive inhibitor of inositol triphosphate gated calcium channels and thus functions as an endogenous calcium antagonist by affecting its uptake and distribution. At neuromuscular junction, it inhibits calcium mediated release of acetyl choline from the presynaptic nerve terminal. In central nervous system, it exerts its depressant effect byinhibition ofN-methyl-D-aspartate glutamate receptor and inhibition of catecholamine release. It also shows modulatory effects on sodium and potassium currents, thus influencing membrane potential. It stabilizeshaemodynamics by inhibition of catecholamine release from the adrenal medulla and peripheral adrenergic nerve endings and direct blockade of catecholamine receptors. It has antiarrhythmic properties related to L-type calcium channel antagonism.[12] MgSO4 has onset of 2-3 minutes and acts for a duration of 30 min by IV route.

In a study conducted by Mahajan *et al*.[13] 30 mg per kg of MgSO4 was used to study pressor response to laryngoscopy and intubation. It was found to attenuate pressor response well with better haemodynamic stability than dexmedetomidine 1mcg per kg. Panda *et al*.[14]conducted a study to ascertain minimal effective dose of MgSO4 for attenuation of intubation response in hypertensive patients. They compared 30 mg/kg, 40 mg/kg and 50 mg/kg of MgSO4 with lignocaine 1.5 mg/kg (control group). HR was in decreasing trend post induction with a brief increase after intubation in all groups. There was no statistical significance in HR between the groups throughout the study period. MAP was well maintained in 30 mg/kg group throughout the study period where as in 40 mg/kg group it showed significant decrease from 3rd minute post intubationtill the end of study period and in 50 mg/kg group, it decreased after induction and remained lower. 40 mg/kg and 50 mg/kg groups required significantly more interventions to manage hypotensive episodes than 30 mg/kg group.It was concluded that 30 mg /kg was the optimum dose of MgSO4 to prevent pressor response for laryngoscopy and intubation in hypertensive patients, with better cardiac stability.

From the inferences of literature search, we chose to use 30mg/kg of magnesium sulphate in our study.

Heart rate

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 thatlignocaine attenuated HR response post laryngoscopy and intubation.

In a study done by Panda *et al*.[14], 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*.[5] 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.

Response on HR was similar in our study and that by Panda *et al*.[14], possible reason being similar timing of administration, dosage and the inducing agent, propofol.

In MgSO4 group of our study, HR at intubation, was near baseline value. From 2nd minute post intubation, HR showed a statistically significant decrease till 10th minute post intubation

but was not clinically significant.

In a study by Panda *et al*.[14] which compared various doses of MgSO4 (30,40 and 50 mg/kg), HR tended to decrease after induction, with a brief rise post intubation in all 3 groups. In a study by Mahajan *et al*.[13] 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*.[6]used 30 mg/kg of $MgSO_4$ 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. Effect of HR in our study was similar to observations made by Panda *et al*.[14] and Mahajan *et al*. [13]

In studies conducted by Allen *et al*.[15] and Puri *et al*.[16] (where 40 mg/kg and 50 mg/kg of $MgSO_4$ was used respectively) increase in heart rate was found at induction of anaesthesia in patients receiving $MgSO_4$ which could be attributed to dosage andmethod of administration where drug was given as bolus (compared to 30 mg/kg infusion over 15 minutes in our study) and inducing agent used was thiopentone, in both the studies (where as propofol was used in Mahajan *et al*., [13]Panda *et al*.[14] and our study).

Systolic blood pressure, diastolic blood pressure and mean arterial pressure

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 ofintubation followed by statistically significant decrease till 10th minute post intubation, though all were within clinically acceptable limits.

In the study by Panda *et al*.[14] 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.

In the study by Mendonca *et al*.[6] lignocaine 2 mg/kg was given before induction of anaesthesia.At intubation increase in SBP and DBP nearing basal value was seen, which was followed by a decrease in the values till 6th min post intubation. In the study by Nooraei *et al*.[5] lignocaine 1.5 mg/kg was given 3 minutes prior to intubation. 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*. [17] 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*. [18] 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 byPanda *et al*. [14], Mendonca *et al*.[6] and Bhalerao *et al*.[18] But lignocaine did not attenuate BP responses well instudies by Nooraei *et al*.[5] and Padmawar *et al*. [17] This difference of result in studies by Nooraei *et al*. [5] andPadmawar *et al*.[17] 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 the study by Mendonca *et al*. [6]drug was administered at induction but still attenuation was seen, it could be due to the dosage of2 mg/kg used.

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 were 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 were clinically insignificant but statistically significant.
In study by Panda *et al*.,[14]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*.,[13] 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 study by Mendonca *et al*.,[6] where 30 mg/kgof $MgSO_4$ given as infusion 10 minutes prior to intubation, SBP and DBP decreased post induction with statistically significant increase post intubation but not of any clinical value. There were higher pressures at 3rd and 6th minute post intubation without any statistical or clinical relevance.
Observations made in studies by Mahajan *et al*.[13] and Panda *et al*.[14]were similar to that in our study indicating consistent action of $MgSO_4$ with respect to SBP and DBP.

Comparison between magnesium sulphate and lignocaine groups

In our study Magnesium sulphate 30 mg/kg and lignocaine 1.5 mg/kg had comparable attenuation ofresponse on HR, SBP, DBP and MAP with none of the them needing any intervention for management of hypotension or any adverse effects.
In study by Mendonca *et al*.,[6] where 30 mg/kg $MgSO_4$ was compared with 2 mg/kg of lignocaine they concluded that lower dose of $MgSO_4$ was sufficient to attenuate haemodynamic response with results similar to lignocaine. In the study by Panda *et al*.,[14]where 3 different doses 30 mg/kg, 40 mg/kg and 50 mg/kg of $MgSO_4$ 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*.,[18] 50 mg/kg of $MgSO_4$ was better than lignocaine 2 mg/kg but required interventions to manage hypotension which was not seen with lignocaine. In study by Padmawar *et al*.,[17] where 40 mg/kg of magnesium was used, it was observed that $MgSO_4$ had sustained control over haemodynamic response to intubation than lignocaine 1.5 mg/kg. In the study by Nooraei *et al*.,[5] 60 mg/kg of $MgSO_4$ was observed to have better control of BP than 1.5 mg of lignocaine but with poor control of HR. In the study by Sunil *et al*.,[19] 50 mg/kg of $MgSO_4$ was regarded as better in attenuating both HR and BP responses to intubation in comparison to 1.5 mg/kg of lignocaine. But study by Tajne *et al*.,[20] using same dose of $MgSO_4$ and lignocaine and same inducing agent thiopentone observed that $MgSO_4$ was effective in attenuating only BP responses well while lignocaine attenuated both tachycardia and hypertensive response associated with intubation.
In line with the above studies by Mendonca *et al*.[6] and Panda *et al*.,[14] our study toohada similar course. While other studies by Nooraei *et al*.,[5] Padmawar *et al*.,[17]Bhalerao *et al*.,[18] Sunil *et al*.[19] and Tajne *et al*.[20]have a result deviating from our study due to usage of higher dose of MgSO4. In few patients, they had adverse effects of tachycardia and hypotension needing intervention. Hence 30 mg/kg of magnesium sulphate and 1.5 mg/kg of lignocaine prove to be good agents for attenuation of intubation response with usage of propofol 2 mg/kg as inducing agent.

Conclusion

Magnesium sulphate 30 mg/kg given IV as infusion over 10 minutes prior to induction of anaesthesiais 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 $MgSO_4$ 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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