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Attenuating the Haemodynamic Stress To Laryngoscopy And Intubation With I.V. Magnesium Sulphate And Lignocaine-Comparative Study

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Abstract: *Direct laryngoscopy and endotracheal intubation frequently induces a cardiovascular stress response characterized by hypertension and tachycardia due to reflex sympathetic simulation. The response is transient occurring 30 seconds after intubation and lasting for less than 10 minutes[2].Although the haemodynamic stress response is transient, and of little consequence in healthy individuals, it is hazardous to those with systemic hypertension, coronary artery disease, and cerebro vascular disease. Complications like myocardial ischaemia, infarction, left ventricular failure, arrhythmias, intracranial hemorrhage can occur due to this response.The major cause of the haemodynamic stress response is due to the stimulation of supraglottic area by the laryngoscope blade followed by additional stimulation contributed by tracheal tube placement.Till date the mainstay of attenuation of the haemodynamic stress response was done by using various drugs like local anesthetics, beta-blockers, calcium channel blockers, opiods and vasodilators.[3,4] All of these techniques which are suggested have some disadvantages related to either cardiovascular or respiratory depression, but none of them directly inhibits the release of catecholamines. Magnesium sulphate blocks the release of catecholamines from the adrenergic nerve terminals and adrenal glands. In our study, which was carried out in the Department of Anaesthesiology at Narayana medical college hospital we compared intravenous Magnesium sulphate and Lignocaine in attenuating the haemodynamic stress response (increase in heart rate and an increase in the mean arterial pressure) to laryngoscopy and intubation.*

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I. Introduction

Haemodynamic stress response to laryngoscopy and intubation occurs as increase in the heart rate and the mean arterial pressure due to reflex sympathetic discharge in response to laryngo - tracheal stimulation.

Tracheal intubation alters respiratory and cardiovascular physiology by reflex response and also by the physical presence of endotracheal tube. Although these circulatory responses are transient and of little consequence in patients with normal circulatory system, they may be exaggerated in patients with coronary artery disease, reactive airways and intracranial pathology. This transitory variable and unpredictable response is mediated by both sympathetic and parasympathetic nervous systems. Usually bradycardia seen in neonates and infants during laryngoscopy and intubation is the autonomic equivalent of Laryngospasm response in adults. This reflex is mediated by an increase in vagal tone at the SA node and is virtually a monosynaptic response to a noxious stimuli in the airway.[2,4] The more common response to tracheal intubation is hypertension and tachycardia mediated by sympathetic efferents via the cardio accelarator nerves and sympathetic chain ganglia .The polysynaptic nature of pathways from the IX and X nerve afferents to the sympathetic nervous system via the brain stem and spinal cord results in a diffuse autonomic response which includes widespread release of norepinephrine from the adrenergic terminals and release of epinephrine from the adrenal medulla. One other reason for the hypertensive response is due to activation of renin-angiotensin system with release of renin from the renal juxtaglomerular apparatus, an end organ innervated by adrenergic nerve terminals.Magnesium sulphate blocks the release of catecholamines from the adrenergic nerve terminals and adrenal glands.

II. Material And Methods

This study was done with the following intentions:

1. To compare the efficacy of Magnesium sulphate and Lignocaine in attenuating the cardiovascular responses toLaryngoscopy andIntubation.
2. To observe any adverse effects of Magnesium sulphate in the specified dosage.
3. To observe any prolongation of neuromuscular blockade in this specified dose

After approval of the study by our institutional Ethics Committee, a total of 60 patients of both sexes

in the age group between 15-50years, belonging to ASA grade I and II undergoing elective surgery under general anaesthesia during the period of January 2014 to july 2015 were included in this double blinded, randomized, clinical study.Informed written consent was obtained from all the patients. Patients were assessed by detailed history and physical examination, supported by routine investigations (Haemoglobin percentage, Blood glucose, urea, creatinine, ECG, X - ray chest PA view).

INCLUSION CRITERIA are patients belonging to ASA grade – I and II MPG grade – I and II, and laryngoscopy time less than 15 seconds.

EXCLUSION CRITERIA for the study includes patients belonging to ASA grade more than two., MPG > II , predicted difficult airway, systemic hypertension, coronary artery heart disease,obesity, diabetes mellitus, patients on antihypertensives and cardiac drugs, and valvular heart diseases.

Out of the 60 patients 30 were randomly included in the L group **(LIGNOCAINE GROUP**) and the other 30 were included in the M group (**MAGNESIUM SULPHATE GROUP**).

All the patients were premedicated with injection Glycopyrolate 0.01mg/kg intramuscularly 45minutes prior to surgery.Patients were shifted to the operation theatre and connected to the noninvasive multichannel monitor, and intravenous access secured using 18G cannula.

Baseline Heart rate, Blood pressure (Mean arterial pressure), SPO2 were recorded.

The patients were preoxygenated with 100% oxygen for 3 minutes. All the patients received injection fentanyl 1mcg/kg intravenously. Two minutes after the administration of fentanyl, patients in L group received injection Lignocaine 1.5mg/kg and the patients in the M group received injection Magnesium sulphate 30mg/kg intravenously. One minute after that the patients were induced with injection Thiopentone5mg/kg and injection vecuronium 0.08mg/kg intravenously. Then the patients were mask ventilated with 100% oxygen and after 3 minutes heart rate, blood pressure (mean arterial pressure) were noted and taken as the post induction value.Laryngoscopy and Intubation was done using appropriate size Macintosh blade and appropriate size endotracheal tube. Measurement of heart rate, blood pressure (Mean arterial pressure) and SPO2 was done immediately after laryngoscopy and at one, three and five minutes after placement of the endotracheal tube. Surgical incision was allowed after the last measurement. Syringes were prepared by a postgraduate who did not take part in the study and the injections were given by him. Intubation was performed by an experienced anaesthesiologist who was double blinded to the drugs given. Neuromuscular blockade was monitored using the nerve stimulator with train of four for every 5 minutes after intubation upto 45minutes.

The incidence of complications like hypotension, arrhythmias, nausea, flushing, sweating were recorded until the patient was discharged from the post anaesthesia care unit.

Statistical Analysis

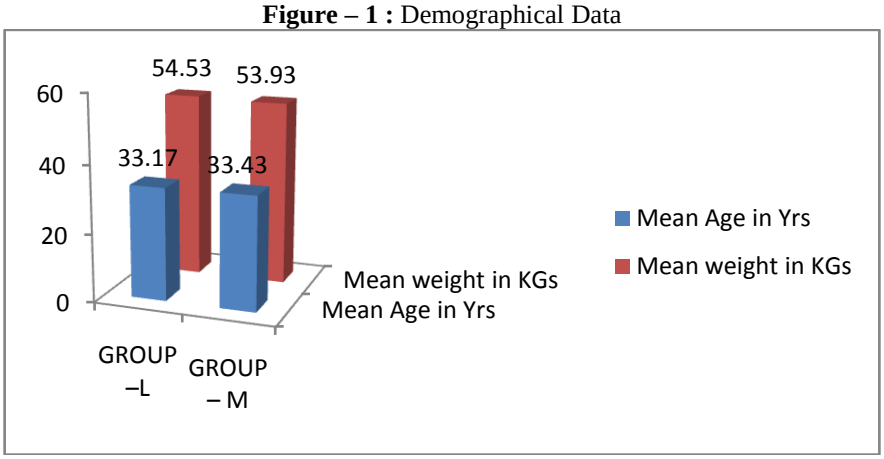
Statistical analysis was done using SPSS version 20.0. Students independent "t" test was used to compare various factors between the two groups. Results were expressed as mean and standard deviation (Mean $\pm$ SD) Chi-square test was done to compare proportions, and p values less than 0.05 was considered as statistically significant.

III. Result

Table : 1 – DEMOGRAPHIC CHACTERS BETWEEN GROUPS

Parameters	**Group – L (n=30)**	**Group – M (n=30)**	**P value**
Mean Age in Years (+S.D)	33.17$\pm$8.05	33.43$\pm$7.82	0.8995
Mean weight in Kgs (+S.D)	54.53$\pm$3.33	53.93$\pm$4.19	0.5416
S.D – Standard Deviation	(P>0.05)		

Figure – 1 : Demographical Data



The groups were matched for demographic data, and there was no statistically significant difference found between the groups in age, sex, weight and surgical position.

With patient on table before giving Lignocaine or Magnesium sulphate, baseline heart rate and mean arterial pressure were recorded for the two groups (Table: 2) and (Table: 3) respectively. There was no significant difference in heart rate and MAP between the 2 groups.

Table: 2 – BASE LINE HEART RATE

Base Line H.R.	**Group – L**	**Group – M**
Mean ±S.D	87.17±6.56	85.83±6.35
HR – Heart Rate	(P=0.4268)	

Figure – 2 Base Line Heart Rate

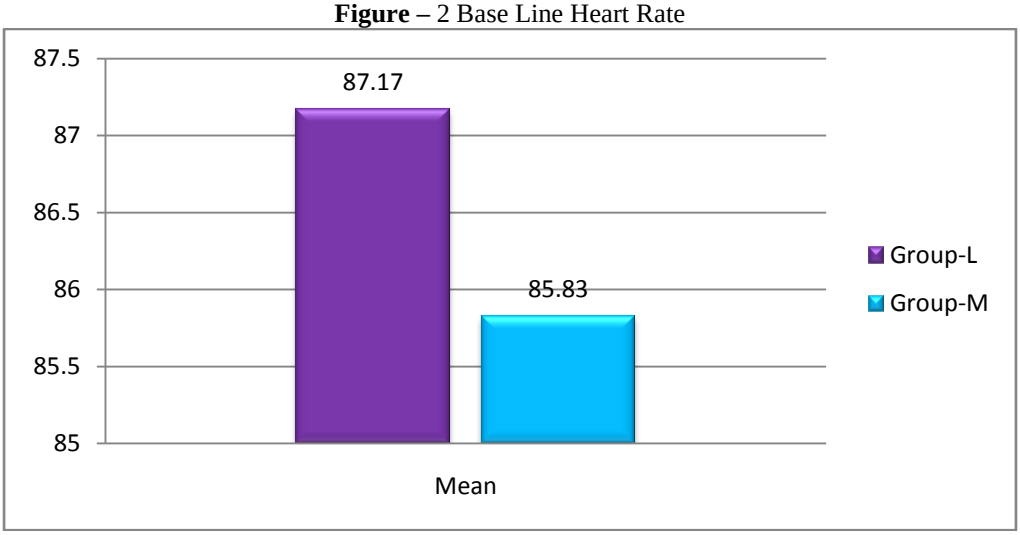
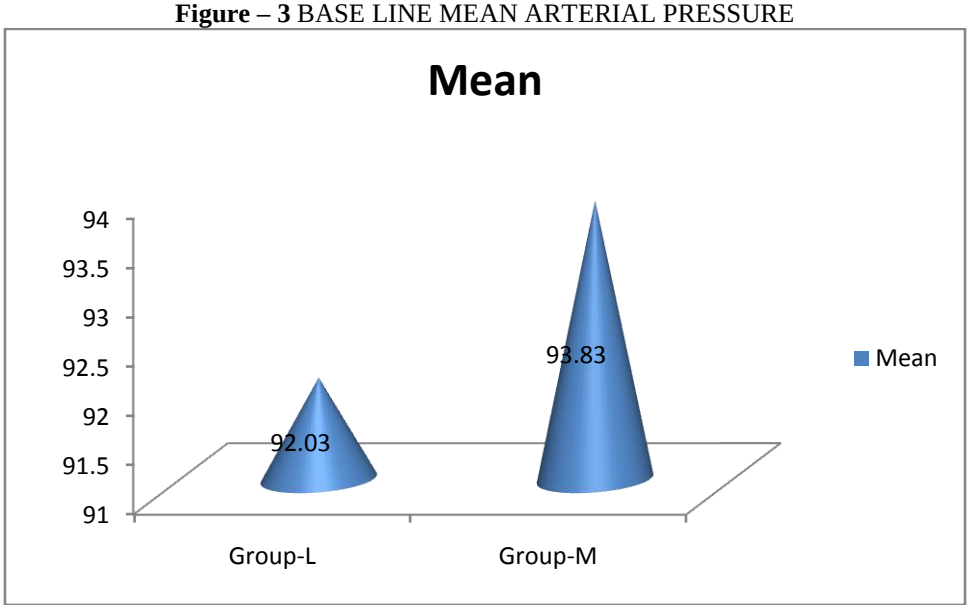


Table: 3 – BASE LINE MEAN ARTERIAL PRESSURE

Base Line MAP	**Group – L**	**Group – M**
Mean ±S.D	92.03±9.75	83.83±8.78
MAP – Mean Arterial Pressure	(P=0.455)	

Figure – 3 BASE LINE MEAN ARTERIAL PRESSURE

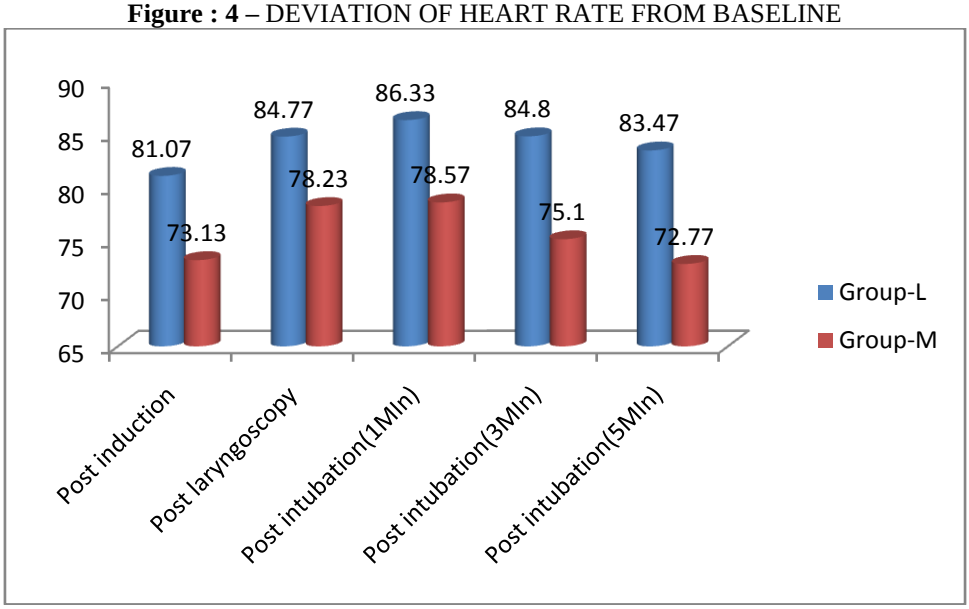


After the drug was administered intravenously any deviation from the baseline values were recorded following induction, laryngoscopy, and intubation. (1 minute, 3 minutes, and 5minutes) (Table: 4, Table: 5)

Table : 4 – DEVIATION OF HEART RATE FROM BASELINE

Heart Rate	**Group – L** Mean ±S.D	**Group – M** Mean ±S.D	**P values**
Post Induction	81.07±6.79	73.13±14.25	0.0079
Post Laryngscopy	84.77±6.06	78.23±5.08	0.0001
Post Intubation (1 Min)	86.3±36.32	78.57±4.45	0.0001
Post Intubation (3 Min)	84.80±5.97	75.10±4.29	0.0001
Post Intubation (5 Min)	83.47±6.27	72.77±3.98	0.0001
			P<0.05

Figure : 4 – DEVIATION OF HEART RATE FROM BASELINE

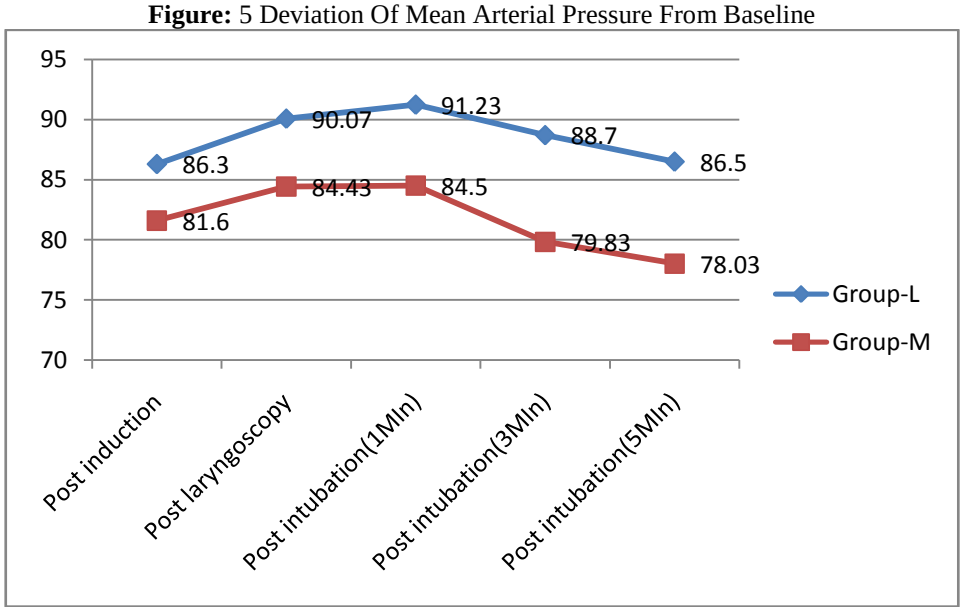


There was fall in heart rate ($P < 0.05$) noted following induction, laryngoscopy, and intubation, in Magnesium group and Lignocaine group, but the fall in heart rate was more significant in the DEVIATON Magnesium OF HEART group when RATE compared with the Lignocaine group.Fig.4,5

Table: 5 DEVIATION OF MEAN ARTERIAL PRESSURE FROM BASELINE

MAP	**Group – L** Mean ±S.D	**Group – M** Mean ±S.D	**P values**
Post Induction	86.3±8.47	81.60±8.14	0.0313
Post Laryngscopy	90.07±8.91	84.43±7.82	0.0117
Post Intubation (1 Min)	88.70±8.95	79.83±7.34	0.0001
Post Intubation (3 Min)	88.70±8.95	79.83±7.34	0.0001
Post Intubation (5 Min)	86.50±8.37	78.03±7.10	0.0001
(MAP – Mean Arterial Pressure)			P<0.05

Figure: 5 Deviation Of Mean Arterial Pressure From Baseline

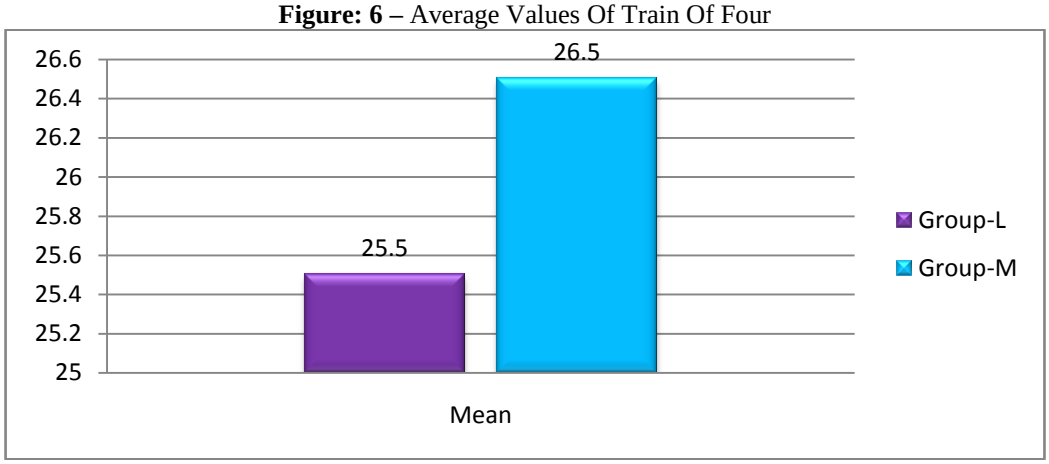


Like heart rate there was a significant fall in mean arterial pressure in both the groups, but there was statistically significant fall (P < 0.05) in the Magnesium group when compared with the Lignocainegroup .

Table: 6 – Average Values Of Train Of Four

TOF in Minutes	**Group – L**	**Group – M**	**P Value**
Mean ±S.D	25.5±4.84	26.5±4.89	0.52
	(P>0.05)		

Figure: 6 – Average Values Of Train Of Four



The train of four values were recorded every 5 minutes upto 45 minutes following drug administration and the duration needed for the return of third twitch using neuromuscular monitoring was recorded. This showed no statistically significant difference between the 2 groups.(P>0.05). (Table: 6)

The incidence of complications for magnesium like hypotension, sweating, arrhythmia, nausea,

flushing, and hot sense were watched for until the patients were discharged from post anaesthesia care unit. There were no such complications observed in this study.

IV. Discussion

Laryngoscopy and endotracheal intubation can cause striking changes in haemodynamic and intracranial pressure probably as a result of intense sympathetic nervous system stimulation[36]. In patients who are at risk of developing increased intracranial pressure, arterial hypertension, myocardial ischemia and these changes may be life threatening. They may lead to cerebral haemorrhage, left ventricular failure and life threatening cardiac arrhythmias. Various techniques were tried to attenuate these cardiovascular responses , one of them being deep inhalational anaesthesia which may cause intracranial hypertension. The other technique being the administration of a large dose of thiopental sodium which can effectively prevent arterial and intracranial hypertension, but in these cases there are risks of cardiac depression. Potent vasodilator drugs need larger doses to attenuate arterial blood pressure and fail to prevent tachycardia caused by laryngoscopy and endotracheal intubation. Vasodilator drugs cause cerebral hypertension. Some of them cause hypertension with reflex tachycardia and others depress the myocardium severely in patients with preexisting left ventricular dysfunction or those receiving beta adrenergic antagonist. These effects are not desirable and limit their usefulness.Various studies have shown that intravenous lignocaine is effective in preventing or attenuating the arterial hypertension and tachycardia in response to endotracheal intubation[37]. some publications have shown the attenuated haemodynamic responses on intubation with intravenous lignocaine [38,39] .

Kim et al [40] and Fuji et al [41] carried out several randomized open studies on adult surgical patients to assess the effect of intravenous lignocaine. They also found reduced haemodynamic stimulation during intubation.

Calcium exerts a major role in stimulus-response relationship, including the release of catecholamines from the adrenal gland and adrenergic nerve terminals in response to sympathetic stimulation. Because Magnesium competes with calcium for membrane channels, it has been described as the physiological calcium antagonist and can modify many calcium mediated responses.[42]

The ability of magnesium ion in inhibiting the release of catecholamines has long been recognized, hence it is considered for use in laryngoscopy and intubation to minimize unwanted cardiovascular responses. [43,44]

Fawcett WJ, Haxby EJ, Male DA [43] showed that in those using magnesium , an increase in systolic blood pressure compared to baseline value occurred in the first minute of intubation; where as in the lidocaine group this increase was significant for the first two minutes. Clearly, intubation does cause an increase in systolic pressure, but variables gradually return to normal. Although diastolic blood pressure after intubation increased in both groups, this increase was not significant ineither group compared to baseline value. These results may be explained by the fact that magnesium causes vasodilatation directly and also indirectly by sympathetic ganglia blockage and inhibition of release of catecholamines. The difference in mean systolic blood pressure between the two groups can be explained by the effect of magnesium on transient reduction of Systemic Vascular Resistance (SVR) in combination with reduced arterial pressure. Findings of this study regarding heart rate in the two groups indicate that, in addition to a reduction in SVR after administration of magnesium, increased heart rate may be due to inhibition of release of acetylcholine from vagal nerves.

On comparison with our study, in which we used Magnesium sulphate in the dose of 30mg/kg intravenously to assess the cardiovascular response to laryngoscopy and intubation showed a decrease in heart rate ($P < 0.005$) and the mean arterial pressure also showed a decrease ($P < 0.05$) on comparison with the Lignocaine group.

G.D. Puri et.al. [30]in their study on the effect of Magnesium sulphate on haemodynamics and its efficiency in attenuating the response to endotracheal intubation in patients with coronary artery disease, studied 36 patients, of which one group received 50mg/kg of Magnesium sulphate intravenously and another group received 1mg/kg Lignocaine intravenously.

Their results showed a decrease in mean arterial pressure from basal values of 91 ± 14.5 to 86.6 ± 14.5 mm of Hg after intubation ($P<0.05$). The heart rate showed a mild increase from 65.2 ± 12.7 to 69.7 ± 13.7 beats/ minute ($P < 0.001$), and the cardiac index is also increased ($P< 0.01$) in the Magnesium sulphate group when compared with control group.

Three patients in the magnesium group and two patients in the control group had severe hypotension that needed pharmacological treatment. This fall was noted in the patients who were taking ACE inhibitors or beta-blockers for coronary artery disease and this potentiated the myocardial depressing effect of Magnesium sulphate. In this study all the patients were ventilated post operatively, so the enhancing effect of neuromuscular blockade was not studied.

On comparison with our study, in which we used Magnesium sulphate in the dose of 30mg/kg

intravenously to assess the cardiovascular response to laryngoscopy and intubation showed a decrease in heart rate ($P<0.005$) and the mean arterial pressure also showed a decrease ($P<0.05$) on comparison with the Lignocaine group.

The increase in the heart rate noted in this study by G.D.Puri et.al.[30] may be due to the ability of Magnesium sulphate in higher doses to inhibit the release of acetylcholine from the vagal nerve predominantly.

This theory was supported by Michael F.M. James, FFARCS, R. Eryk Beer, FFA(SA), and Jan D. Esser, MMED[29] in their study, "Intravenous Magnesium sulphate inhibits catecholamine release associated with tracheal intubation" in which they used Magnesium sulphate in the dose of 60mg/kg intravenously and their results showed an initial increase in heart rate by 13$\pm$ 3.9 beats/ minute.

This effect was not observed in our study as we used a dose of 30mg/kg, which was a lesser dose on comparison with their study.

The mean arterial pressure in both studies showed a declining trend, but three patients in the Magnesium sulphate group in their study showed hypotension which required treatment, which was not observed in our study since we included majority ASA grade I patients, who were not on any drugs acting on cardiovascular system preoperatively. However we should be aware of the adverse haemodynamic interactions of Magnesium sulphate in patients receiving cardiovascular drugs like ACE inhibitors, betablockers and calcium channel blockers.

In a dose response study of Magnesium sulphate in suppressing the cardiovascular responses to laryngoscopy and intubation done by K.Montazeri M.D., M.Fallah M.D.[33] studied 6 groups of patients of 20 each with varying doses of Magnesium sulphate (10,20,30,40,and 50mg/kg) and compared with the control using Lignocaine in the dose of 1.5mg/kg. and observed that there was significant reduction in heart rate on comparison between Magnesium sulphate and Lignocaine groups($P < 0.05$). But within the magnesium groups the difference in heart rate was not significant ($P > 0.05$).

The train of four at 45 minutes after induction of anaesthesia in all groups had no statistically significant differences.

Adverse effects of Magnesium sulphate like hypotension, arrhythmia, nausea, sweating and flushing, were observed in certain patients in higher doses.

Similarly in our study using 30mg/kg of Magnesium sulphate there was a reduction in the heart rate ($P <0.05$) and the mean arterial pressure ($P<0.05$), which was statistically significant when compared with the lignocaine group lending support to our study. We did not observe any side effects in any of the patients. Similarly neuromuscular blockade was not significantly prolonged in our study as seen by the train of four values at 45 minutes.

Like heart rate there was significant fall in the mean arterial pressure when compared with the lignocaine group ($P < 0.05$), but the fall is not significant within the magnesium groups ($P > 0.05$) .

In a study by G.M. Saunders FRCA, K.M. Sim M.Md (Anaes),[32]" Is it Feasible to use Magnesium sulphate as hypotensive agent in oral and maxillo facial surgery?

Studied 16 patients using Magnesium sulphate infusion at 40g/hr until the mean arterial pressure reached to 55$\pm$ 5 mm Hg and followed by maintenance of 5g/hr until 30 min. prior to the end of surgery, their results showed decrease in mean arterial pressure and heart rate from a baseline during the surgery and which returned to baseline 26 minutes after the surgery.

Train of four monitoring during surgery showed no response in all the patients on Magnesium sulphate infusion. On terminating the Magnesium sulphate infusion the first tetanic contraction appeared with no fade, followed by train of four pattern similar to that seen with depolarizing neuromuscular blockers. There were no complications like reflex tachycardia or arrhythmias observed.

This study corroborates with our study in the reduction of mean arterial pressure and heart rate significantly in intravenous use of Magnesium sulphate, but we altered the dose to 30mg/kg bolus, as they have used higher doses and observed few side effects like prolonged sedation postoperatively.

In another study by R.W. Allen,M.B., CH.B., F.F.A.R.C.S.I, M.F.M. James, CH.B., P.C. Uys, CH.b. FFA (SA)[27] in " Attenuation of the pressor response to tracheal intubation in hypertensive proteinuric pregnant patients by Lignocaine, Alfentanil, and Magnesium sulphate". They used Magnesium sulphate 40mg/kg intravenously and observed an increase in the systolic, diastolic blood pressure and mean arterial pressure following intubation, in the lignocaine group when compared to the Magnesium group. Similarly the heart rate was decreased in the Magnesium sulphate group significantly when compared to the other two groups. Alfentanil caused least change in heart rate but caused significant fetal depression. Although Magnesium sulphate and Alfentanil provide adequate control of cardiovascular response in hypertensive patients, Alfentanil is less reliable in controlling severe hypertension.

This study corroborates with the evidences in our study that Magnesium sulphate intravenously given prior to induction can have adequate control of cardiovascular response to laryngoscopy and intubation.

Our present study lends support to previous studies that the use of Magnesium sulphate 30mg/kg

intravenously one minute prior to induction reduces the heart rate and mean arterial pressure in response to laryngoscopy and intubation in a favourable manner without any side effects.

V. Conclusion

We conclude that, Magnesium sulphate in the dose of 30mg/kg given intravenously one minute prior to induction,

- ❖ Attenuates the cardiovascular responses to laryngoscopy and intubation in a better manner than lignocaine.
- ❖ Does not cause any adverse effects in any of the patients.
- ❖ Does not cause prolongation of the neuromuscular blockade

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