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ORIGINAL ARTICLE

Comparison of Effects of Intravenous Lignocaine and Magnesium Sulphate on the Cardiovascular Response to Laryngoscopy and Intubation

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ABSTRACT

Background and Objective: The patient's heart rate, blood pressure, pulmonary arterial pressure, and capillary wedge pressure will all rise during laryngoscopy and tracheal intubation. These hemodynamic alterations are less noticeable in those with normal blood pressure but have been linked to several diseases. They are well tolerated by those with normal blood pressure. The study's goal is to compare the cardiovascular effects of lignocaine and magnesium sulfate during laryngoscopy and intubation.

Methodology: The objective of this randomized controlled trial was to examine the effects of lignocaine and magnesium sulfate on cardiovascular and respiratory responses after endotracheal intubation in ASA I patients. The 60 participants' ages ranged from 15 to 45, and they were all classed as "grade I" by the ASA. Each of these patients underwent elective surgery under general anesthesia voluntarily. The sixty people were divided into two groups of thirty using a random selection method. Neither group was significantly different from the other in terms of demographics or blood-flow parameters. Group L was administered 1.5 mg/kg intravenously of lidocaine three minutes before to induction. Group M received an intravenous injection of magnesium sulfate at a rate of 40 mg/kg over the course of one minute.

Results: In both the Magnesium and Lignocaine groups, heart rates increased, but the Lignocaine group's increase was far more dramatic (p value 0.01). Both groups experienced an increase in systolic blood pressure following medication administration (p value 0.05). The results of Group M remained statistically significant for the first minute after the insertion of the tube, but by the fifth minute, they had returned to pre-insertion levels. In Group L, blood pressure increased significantly at 1 and 3 minutes after tube insertion, but by 5 minutes, blood pressure had returned to pre-insertion levels. The SBP of group L individuals increased significantly greater than that of group M participants. (p 0.05). Immediately following intubation, there was a statistically significant (p 0.01) increase in DBP in both groups; however, three minutes later, DBP had returned to pre-intubation levels in both groups (p 0.01). It is five percent as important. (p 0.05). The results revealed that the growth rate of group L members was significantly higher than that of group M members. In the presented situation, p equals 0.01

Conclusion: When it comes to preventing a rise in heart rate, blood pressure, and blood pressure during laryngoscopy and tracheal intubation for patients with an ASA grade I, magnesium sulfate is more beneficial than lidocaine.

INTRODUCTION

One of the steps in administering general anesthesia is performing an endotracheal intubation and laryngoscopy. It is common knowledge among doctors that laryngoscopy and endotracheal intubation trigger hemodynamic effects. When doing a laryngoscopy, the autonomic nervous system is aroused, causing the larynx, pharynx, and trachea to respond. These modifications may have unfavorable effects on people, especially those who are already vulnerable due to preexisting diseases including ischemic heart disease, cerebrovascular sickness, high blood pressure, old age, or type 2 diabetes. Many studies have been conducted to find ways to stop this stress reaction, but none of them have been wholly successful. Magnesium sulphate and lignocaine were examined in this study for their abilities to reduce the detrimental effects of laryngoscopy and tracheal intubation on blood flow. Individuals who had undergone these treatments were studied for this study.

METHODOLOGY

This prospective, randomized trial was approved by the Ethical Committee, and all participants supplied written informed permission. Sixty people, ages 15 to 45, all with ASA physical status I, agreed to be surgical subjects for the study. Patients with an ASA respiratory status of 2, 3, or 4, women who are pregnant, patients undergoing emergency surgery, patients who are allergic to the study drugs, and patients whose airways are difficult to open and require more than 30 seconds of laryngoscopy and more than one attempt were excluded from the study. The study also did not include patients with an ASA respiratory status of 2, 3, or 4.

These people were arbitrarily separated into two categories (30 in each group). Participants in Group M received a dose of 40 mg/kg of magnesium sulphate 50%, while those in Group L received a dose of 1.5 mg/kg of lidocaine. Patients received complete physicals and any additional diagnostic testing that was considered essential before undergoing any type of surgical operation. Before surgical treatments, patients were instructed to fast for at least six hours. Each subject had 0.2 milligrams of glycopyrrolate injected into a muscle prior to receiving the general anesthesia. It was necessary to do this before the operation.

To administer Ringer lactate intravenously, a cannula was placed in a vein in the patient's non-dominant hand. The patient's health was evaluated with the aid of a pulsoximeter, an electrocardiogram (ECG), and a noninvasive blood pressure monitor (NIBP).

Each patient was given three minutes of pure oxygen before surgery. Group L patients were given lignocaine intravenously at a rate of 1.5 mg/kg three minutes before their scheduled general anesthetic procedure. It was done before the general anesthetic so that everything would go well. Prior to undergoing general anesthesia, those in Group M were given 40 milligrams per kilogram of body weight (mg/kg) of magnesium sulphate intravenously at a pace of one minute. Then, an intravenous dose of thiopentone (5 mg/kg) was given to induce unconsciousness. A dose of 1.5 mg/kg of succinylcholine was administered, and then the patient was given a 100 percent oxygen mask. After the fasciculations stopped, a laryngoscopy with a Macintosh laryngoscope blade was done, and an endotracheal tube was

placed. The patient was kept unconscious with a gas mixture consisting of 50% oxygen, 50% nitrous oxide, and 1% isoflurane.

Statistical analysis: The average clinical parameters of the two study groups were compared using the independent sample t-test to see if there was a statistically significant difference between the groups (such as HR, SBP, and DBP). Verification of the validity of the normality and variance assumptions allowed us to conclude that this was the case. We used the t-test, which does not need evenly paired samples, to compare the two groups' parameters. We used the paired t-test to compare each group before and after receiving the study medications (Lignocaine and Magnesium sulphate). A p-value of 0.05 or less is considered to be the cutoff for statistical significance. All testable hypotheses were crafted to be in agreement with or disagreement with the null hypothesis.

RESULTS

Table 1:

	Group L	Group M	p value
Age (yrs)	29. 2 ± 8.44	30.1 ± 9.15	> 0.05
Gender M/F	15/15	15/15	
Basal HR (bpm)	73.2 ± 3.94	72 ± 3.84	> 0.05
Basal SBP (mmHg)	123.43 ± 6.73	122.27 ± 6.03	> 0.05
Basal DBP (mmHg)	79.33 ± 5.31	77.27 ± 4.97	> 0.05

Table 2:

	Baseline	after Lignocaine	1 min	3 min	5 min
HR (bpm)	73.2 ± 3.73	70.99 ± 3.19	102.68± 18.85	96.10 ± 7.75	80 ± 6.14
p value		0.59	<0.01	<0.01	<0.01
SBP(mmHg)	114.51 ±5.63	127.19 ±5.48	139 ±7.26	141 ±7.21	124.58 ±5.01
p value		0.81	<0.01	<0.01	0.65
DBP(mmHg)	78.32 ±6.11	81.24 ± 7.28	96.83 ±7.73	99 ±8.69	74.25 ±8.02
p value		0.29	<0.01	<0.01	<0.05

Table 3:

	Baseline	after Magnesium sulphate	1 min	3 min	5 min
HR (bpm)	72 ±3.98	78.2 ±4.39	82.3 ±4.79	79.22 ±4.58	78.2 ±6.81
p value		<0.01	<0.01	<0.01	<0.01
SBP(mmHg)	119.19 ± 5.02	124.18 ± 5.39	125.57 ±8.33	123.12 ±6.44	117.24 ±5.32
p value		0.5	<0.01	0.19	<0.01
DBP(mmHg)	76.18 ±5.01	77.3 ±4.27	86.05 ±4.71	85.69 ±6.53	71.71 ±6.82
p value		0.18	<0.01	<0.01	<0.01

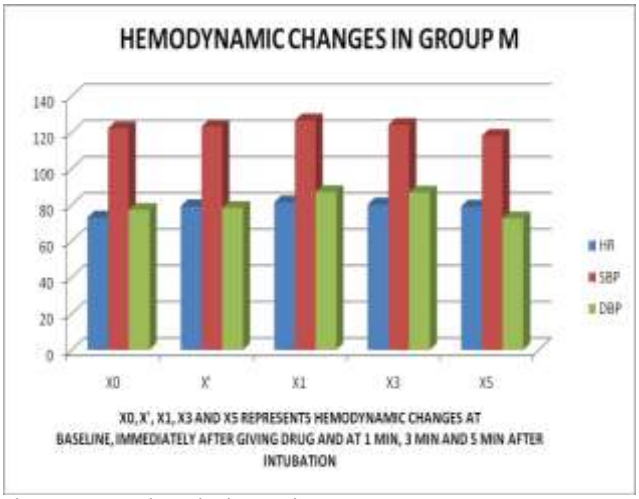
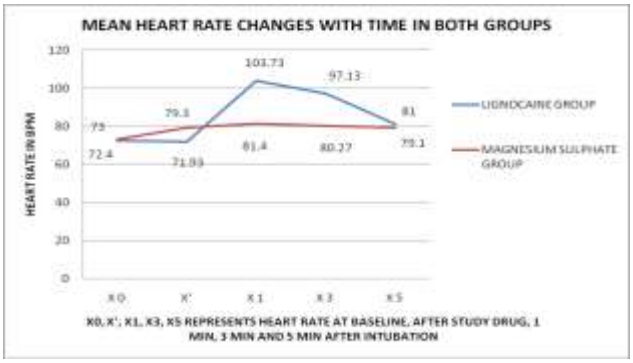


Figure 1: Haemodynamic changes in group M



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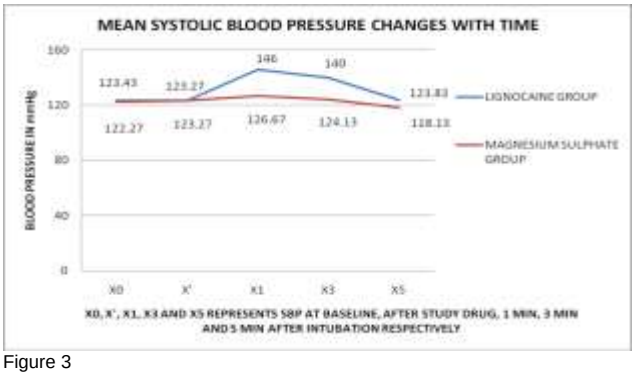

Figure 3

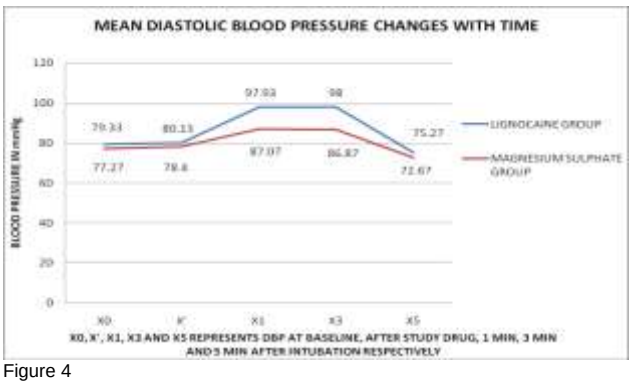

Figure 4

According to the data, neither group of patients was significantly different from the other. (Table 1). Changes in blood flow were measured in Group L after intravenous lidocaine was administered and compared to baseline values. As seen in Table II, lignocaine caused a transient and mild reduction in heart rate. Although the systolic blood pressure did not change immediately after lidocaine was injected, it did rise significantly one and three minutes after the tube was placed. For the first five minutes after the tube was put, there was little to no change in the subject's blood pressure. Diastolic blood pressure did not immediately change after intravenous lidocaine administration. One and three minutes after intubation, however, there was a notable increase compared to pre-intubation levels. The diastolic blood pressure of this group dramatically decreased after 5 minutes of intubation, relative to the pre-intubation value. Magnesium sulphate was given intravenously to Group M, and their blood flow was then monitored and compared to the initial levels. The heart rates of this group significantly increased when magnesium sulfate was given, and again one minute, three minutes, and five minutes following intubation, as compared to the values obtained at baseline (Table III). It remained high after three minutes had elapsed, but

statistically speaking it was no different than when it had initially started. After intubation, this group experienced a significant reduction in systolic blood pressure five minutes later. It was not immediately apparent after the magnesium sulfate injection, but it was noticeable one and three minutes later. After 5 minutes of intubation, this group's DBP dropped significantly compared to its baseline levels.

The hemodynamic responses of both groups were measured and compared immediately after the administration of the study medications. In Group L, the average heart rate was 71.93 beats per minute, while in Group M it was 79.3 beats per minute. It was shown that Group M saw a much larger increase in heart rate than Group L. (p value 0.01). The average heart rates of both groups are shown changing over time in Figure 3. The average blood pressure of the study's participants, immediately after receiving the study drug, was 123.27 6.44 mmHg. Group L had a mean systolic blood pressure of 80.13 8.17 mmHg, while Group M had a mean systolic blood pressure of 78.4 5.8 mmHg Treatment with the drug had no noticeable effect on DBP (p value was greater than 0.05). Blood pressure and pulse rate were monitored and compared before and after trachea implantation (Figure 3). The average heart rate in Group M was 81.4 5.68 beats per minute, while the average heart rate in Group L was 103.73 19.96 beats per minute. The heart rates of those in Group L increased noticeably greater than those in Group M after just one minute after intubation (p 0.05). SBP averaged 124 13.74 mmHg in Group M, compared to 140 8.19 mmHg in Groups L and M. SBP in Group L increased significantly more than in Group M three minutes after intubation. significance level of.05.

Changes in blood flow were tracked and analyzed for five minutes after a tracheal tube was placed in the patient's windpipe. The average heart rate in Group L was 81,7.2 beats per minute, which was significantly higher than Group M's average heart rate of 79,1.79 beats per minute. Nothing unusual happened in terms of heart rate (p value 0.16). The average systolic blood pressure was 123.87 4.03 mm Hg in Group L and 118.13 6.39 mm Hg in Group M. Group L's SBP was significantly higher than Group M's (p 0.01). (Table 4).

Group L had mean DBP of 75.27 9.04 mmHg, while Group M had mean DBP of 72.67 7.67 mmHg. Concentrations of DBP did not differ significantly between the two groups (p = 0.23). (Fig. 5).

DISCUSSION

The patient may be at danger for a cerebral hemorrhage, left ventricular failure, and other complications as a result of the reflex sympathetico adrenal discharge because of these responses. Lignocaine acts in this way to immediately slow down the heart rate and enlarge the blood vessels that are surrounding the heart. These two medications have been investigated by researchers to see whether or not they can minimize the response to intubation, and the results have been encouraging. The fact that comparable research has never been conducted on these medications in our environment, along with the fact that they are less costly and more readily available, led to their selection for the clinical trial.

Before performing a laryngoscopy, the researchers Abou Madi, Kieszler, and Yacoub [6] determined that the optimal dose of lidocaine to inhibit this response was 1.5 mg/kg administered intravenously. C.D. Miller and S.J. After performing a laryngoscopy and intubation, Warren [8] observed that administering lidocaine intravenously at a rate of 1.5 mg/kg three minutes later had no impact on the way the heart and lungs reacted to the procedures. Two minutes before to the laryngoscopy, Chraemmer-Jorgensen and his colleagues [9] administered lignocaine at a rate of 1.5 mg/kg through intravenous injection. In light of this fact, lignocaine at a dose of 1.5 milligrams per kilogram was administered three minutes before intubation in this trial. We evaluated the two medications to see whether one was more effective at lowering the reactivity to the induction. Both in terms of their demographic make-up and their hemodynamics, the groups were comparable. (Details can be found in Table 1)

Initially, the magnesium group experienced an increase of 13.39 beats per minute in their heart rate after receiving an injection of magnesium sulphate. In the group that served as the control, there was not much of a difference in the heart rate from the beginning. Two minutes after the intubation, the heart rate of the control group increased by 30.9 beats per minute, whereas the heart rate of the magnesium group remained nearly the same as the data before magnesium was administered. There were significant variations between the groups two minutes after the intubation was performed. In 1998, G. According to D. Puri and colleagues' [11] findings, once magnesium sulphate was administered, HR had a tendency to increase, albeit not by a significant amount. After endotracheal intubation, the HR went up by a significant amount for both groups.

According to the findings of Fallah et al. [12], which were published in 2005, there were not statistically significant changes identified between the magnesium groups' mean heart rates. The magnesium and lignocaine groups, on the other hand, showed statistically significant differences from one another. Following a laryngoscopy, the magnesium (40 mg/kg) group experienced an increase in the average heart rate of up to 10% at 1 and 3 minutes following the procedure. After five minutes, the usual rate of the heartbeat had returned. In the group that received lignocaine, the average heart rate decreased by up to 10% after the drug was administered, but it increased by up to 25% after the patient was intubated, and it didn't return to normal for at least 5 minutes after that change.

According to studies published in 2006, the heart rate increased after magnesium (40 mg/kg) was given, increased further after intubation, and didn't return to normal until 5 minutes after intubation. After magnesium administration, the heart rate increased. So far, magnesium. After taking the investigational drug, Group M participants' heart rates increased significantly, while Group L participants' heart rates remained unchanged. Both groups' heart rates were higher 1, 3, and 5 minutes after intubation (Observations, Table 2 and 3). In the first and third minutes after intubation, Group L had a higher heart rate than Group M. The groups differed statistically within five minutes of the trial.

Anesthesia lowered both groups' systolic and diastolic blood pressures. This held true whether group got injection first. Magnesium-treated patients did not experience an increase in systolic blood pressure after tube implantation. Even the "control" group experienced this. Both groups' worldviews were different. Diastolic blood pressure also changed. Puri et al. [11] found that magnesium alone lowered pre-induction mean arterial pressure (P 0.001). 0.05 Only the control group changed following anesthetic injection (P 0.01). Before intubation, the two groups couldn't be statistically differentiated. Both pre- and post-intubation (3 minutes apart) lignocaine revealed greater MAP than magnesium (P 0.01). In two minutes, air pressure normalized. Compared to baseline, lignocaine reduced mean arterial pressure by 10%. After the operation, something happened. After intubation, the treatment group's mean arterial pressure rose 30% and stayed high for 5 minutes. Systolic and diastolic blood pressure reduced fast after magnesium injection [13] (Sharma J., et al.]. Navid Nooraei et al. [15] found that magnesium sulfate regulates hemodynamics better than lignocaine. Despite increasing HR, this was true. Compared to the lignocaine group, 30 mg/kg of magnesium sulphate significantly reduced HR and MAP (P 0.05). Lidocaine isn't as effective as magnesium sulfate at reducing heart rate during laryngoscopy and intubation. Intravenous magnesium sulfate is given one minute before induction. Five minutes in, there were no evidence of a rising SBP. Group M's systolic blood pressure increased after taking magnesium sulfate, however the increase was not statistically significant. The significance of this increase became obvious once the patient was given a breathing tube. Usually, the level normalizes within three minutes. Five minutes into intubation, Group M's blood pressure dropped significantly. SBP's blood pressure didn't change after taking experimental medications. Group L participants showed greater blood pressure

one, three, and five minutes after the tube was introduced. Group L's diastolic blood pressure did not change after lidocaine administration, but it increased one and three minutes after intubation compared to pre-intubation values. Although there was no immediate difference, this was the result. Group M's DBP rose after taking magnesium sulfate, although not much.

When calcium is present, sympathetic activation of adrenergic nerve terminals stimulates adrenal catecholamine release. This rivalry disrupts calcium-dependent processes. Magnesium prevents intubation-induced catecholamine release. This reduces heart problems. This study indicated that the heart was most affected. Magnesium reduces calcium-driven depolarizing current in pacemaker tissue, delaying animal atrial beats. This affects pacemaker tissue. We got ideas from an animal. In healthy animals, magnesium can prevent vagus nerve acetylcholine release [18]. This raised heart rate. Before tubes were implanted, the lignocaine group's heart rate was lower than the magnesium group's. After intubation, lignocaine-treated patients had higher heart rates than magnesium-treated patients. Compared to magnesium, lignocaine raised epinephrine levels higher. The magnesium group presumably had better blood pressure regulation due to vasodilatory effects and reduced catecholamine production. Because magnesium inhibits catecholamine release. Studies demonstrate that magnesium affects these processes. Even at high blood levels, magnesium has no neurological effects. Magnesium probably doesn't work this way. The blood-brain barrier prevents magnesium from entering the brain. High dosages of magnesium have a modest effect on the CNS. [20]

Magnesium may help in this situation. Magnesium levels between 2 and 4 mmol/L may increase a pregnant woman's survival after endotracheal intubation. Magnesium reduces succinylcholine-induced fasciculations [22] and potassium release [23]. This could be useful because magnesium affects heart pumping. Magnesium doesn't seem to prolong succinylcholine's effects. [13,24]. Magnesium and other relaxants that don't lower heart rate should be used with these medicines. This combination requires a lower dose of the relaxant. [23,24] Magnesium sulfate's effects on non-depolarizing relaxant block duration and strength are unknown. Magnesium sulfate aids intubation. [23,24]

CONCLUSION

Magnesium sulfate reduces systolic and diastolic blood pressure following tracheal intubation but not heart rate. Lignocaine doesn't impact heart rate or systolic blood pressure. Magnesium sulphate is superior to lidocaine in ASA grade I patients during laryngoscopy and tracheal intubation.

REFERENCES

1 Burstein CL, Lopinto FJ and Newman W. Electrocardiographic studies during endotracheal intubation during usual routine techniques. Anesthesiology. 1950; 11: 224.

2 Miller Forbes A, Dally F. Acute hypertension during induction of anesthesia and endotracheal intubation in normotensive patients. British Journal of Anesthesia. 1970; 42: 618.

3 Prys Roberts C, Greene LT, Meloche R, Foex P. Studies of anesthesia in relation to hypertension, hemodynamic consequences of induction and endotracheal intubation. British Journal of Anesthesia. 1971; 43: 531-547.

4 Reid LC, Brace DE. Irritation of respiratory tract and its reflex effects upon the heart. The Journal of surgery, gynecology and obstetrics. 1940; 70:157.

5 Takeshima K, Noda K, Higaki M. Cardiovascular response to rapid anesthesia induction and endotracheal intubation. Anesthesia & Analgesia: March/April 1964 - Volume 43 - Issue 2 - ppg 201-208

6 Abou-Madi MN, Keszler H, Yacoub JM. Cardiovascular response to laryngoscopy and tracheal intubation following small and large intravenous doses of Lidocaine. Can Anaes Soc J. 1977 Jan; 24(1):12-19.

7 Tam S Chung F, Cambell J M Attenuation of circulatory response to endotracheal intubation using i.v lidocaine :a determination of the optimal time of Injection. Canadian Journal of Anaesthesia 1985:32:565.

8 CD Miller, SJ Warren. i.v. Lignocaine fails to attenuate the cardiovascular response to laryngoscopy and tracheal intubation. B.J.A. 1990;65:216-219.

9 Chraemmer -Jorgensen B, Hoilund –Carlsen PF, Marving J, Christensen V. Lack of effect of intravenous Lidocaine on hemodynamic responses to rapid sequence induction of general anesthesia. Anesth Analg. 1986 Oct; 65(10):1037-1041.

10 Michael FM James, R Eryk Beer, Jan D Esser. Magnesium sulphate inhibits catecholamine release associated with tracheal intubation. Anesthesia and Analgesia. 1989; 68:772-776.

11 Puri GD, Marudhachalem KS, Pramila Chari, Suri RK. The effects of Magnesium sulphate on hemodynamic and its efficacy in attenuating the response to endotracheal intubation in patients with coronary artery disease. Anesth Analg. 1998; 87:808-11

12 K Montazeri, M Fallah. A dose response study of Magnesium sulphate in suppressing cardiovascular responses to laryngoscopy and endotracheal intubation. Journal of Research in Medical Sciences. 2005; 10(2):82-86

13 Juhi Sharma, Vikas Sharma, Ranbhushan, Satyadev Gupta. Comparative study of Magnesium Sulphate and Esmolol in attenuating the pressor response to endotracheal intubation in controlled hypertensive patients. J Anesth. Clin Pharmacol. 2006; 22(3): 255-259

14 Vandana Trivedi, Rajesh Patel. Comparative study of efficacy of i.v. Magnesium sulphate versus Buprenorphine for attenuating the pressor response to laryngoscopy and intubation. J Anaesth Cli Pharmacol 2009; 25(4):459-462

15 Nooraei et al, Effects of intravenous magnesium sulphate and lidocaine on haemodynamic variables following direct laryngoscopy and intubation in elective surgery patients, Tanaffos. 2013;12(1):57-63 (Pubmed)

16 Narmatha et al, A comparative study on the efficacy of magnesium sulphate against lignocaine in attenuating the cardiovascular responses to laryngoscopy and endotracheal intubation, IOSR-JDMS, 2016;15(4):61-64

17 lseri LT, French JH. Magnesium: Nature's physiologic calcium blocker. Am Heart J. 1984; 108:188-93.

18 Turlapaty PDMV, Carrier O. Influence of Magnesium on calcium induced responses of atrial and vascular muscle. J Pharmacol Exy Ther 1973;187:86-98.

19 Somjen GG, Baskerville EN. Effect of excess magnesium and vagal inhibition and acetylcholine sensitivity of the mammalian heart in situ and in vitro. Nature 1968; 217: 679-80

20 Casati A, Albertin A, Deni F, Fanelli G. Small doses of Remifentanil or Sufentanil for blunting cardiovascular response induced by tracheal intubation a double blind comparison. Eur J Anaesth. 2001; 18(2): 108-112.

21 Crawford DC, Fell D. Effects of Alfentanil on the pressor and catecholamine responses to tracheal intubation. Br J Anaesth. 1987; 59:707-12

22 De Vore JS, Asrami R. Magnesium sulfate prevents Succinylcholine induced fasciculations in toxemic patients. Anesthesiology. 1980; 52:76-7.

23 James MFM, Cork RC, Dennett JE. Succinylcholine pretreatment with Magnesium sulfate. Anesth Analg. 1986; 65:373-6.

24 Ghoneim MM, Long JP. The interaction between Magnesium and other neuromuscular blocking agents. Anesthesiology. 1970 Jan; 32(1):23-7