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Original Research Article

Comparative Study of Intravenous Lignocaine and Intravenous Magnesium Sulphate in Attenuating Stress Response to Laryngoscopy and Intubation

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ABSTRACT

Background and Aims: Laryngoscopy and tracheal intubation are associated with an increase in heart rate, systemic blood pressure, pulmonary arterial pressure and capillary wedge pressures. These hemodynamic changes are well tolerated in normotensive individuals, but are of greater significance in patients with coronary artery disease and cerebrovascular disorders, and have been recognized as a potential source of a number of complications.
Methods: This randomized study was done to compare and evaluate the efficacy of Lignocaine and Magnesium sulphate to attenuate the cardiovascular response to endotracheal intubation in ASA I patients. This study included 60 patients of ASA grade I aged between 15 and 45 years, scheduled for elective surgery under general anesthesia. Patients were randomized into two groups of 30 each. There was no significant difference in demographic or baseline hemodynamic variables between the two groups. Group L received i.v. Lignocaine in the dose of 1.5mg/kg 3 minutes before induction and Group M received i.v. Magnesium sulphate 40mg/kg over 1 minute just before induction.
Results: We found a significant rise in heart rate in both the groups but the rise was significant in Lignocaine group as compared to Magnesium group. (p value < 0.01) There was rise in systolic blood pressure immediately after drug administration in both the groups (p value <0.05). It remained significant at 1 minute after intubation but returned to baseline at 3 minutes, which dropped below baseline at 5 minutes post intubation in group M. In group L significant rise in SBP at 1 and 3 minutes was observed which returned to baseline at 5 minutes post intubation. The rise in SBP was significantly higher in group L as compared to group M (p value <0.05). The rise in DBP was observed in both the groups upto 3 minutes after intubation (p value <0.01) which further dropped below baseline in either groups (p value < 0.05). The progressive rise was significant in group L as compared to group M (p value < 0.01)
Conclusion: Magnesium sulphate provides fairly good and sustained control over rise in HR, SBP and DBP during laryngoscopy and tracheal intubation in ASA grade I patients as compared to Lignocaine.

Key Words: Intravenous Lignocaine, Intravenous Magnesium Sulphate, Stress Response, Laryngoscopy, Intubation.

INTRODUCTION

Laryngoscopy and endotracheal intubation are part of induction of general anesthesia. The occurrence of hemodynamic responses during laryngoscopy and endotracheal intubation are well known. Laryngoscopy results in stimulation of larynx, pharynx and trachea, which are

extensively innervated by the autonomic nervous system, activation of which leading to various cardiovascular changes like increase in heart rate, blood pressure, dysrrhythmias, cardiac asystole and even sudden death. [1-5]

These changes may prove to be detrimental especially in patients with ischemic heart disease, cerebrovascular disease, hypertension, old age and diabetes mellitus. Several techniques have been studied to attenuate this stress response but none of them are completely satisfactory. Hence there is constant search to attenuate the hemodynamic response to laryngoscopy and endotracheal intubation.

In the present study we have tried to assess and compare the efficacy of Magnesium sulphate and lignocaine in attenuating the untoward hemodynamic response to laryngoscopy and tracheal intubation.

METHODS

This prospective, randomised study was conducted after obtaining approval from the Ethical Committee and written informed consent from the patients. We included 60 patients belonging to American Society of Anesthesiologists (ASA) physical status I of either sex, aged between 15–45 years undergoing elective surgical procedures. The exclusion criteria included patients belonging to ASA II, III and IV, pregnant patients, patients for emergency procedure, those allergic to study drugs and patients with difficult airway, requiring duration for laryngoscopy more than 30 seconds and multiple attempts.

These patients were randomly assigned into two groups (30 in each group). Group L received 1.5 mg/kg of inj. Lignocaine and group M received 40 mg/kg of inj. Magnesium sulphate 50%.

Prior to surgery, the patients underwent through pre-anesthetic check up and required investigations. Patients were kept NPO 6 hours before surgery. All patients were pre-medicated with inj. Glycopyrolate 0.2 mg intramuscularly 30 min before induction of general anesthesia.

In the operation theatre, intravenous (IV) access was established with 18 G cannula in suitable vein on nondominant hand and Ringer lactate was started. Vital signs were measured by placing an electrocardiogram (ECG), a NIBP monitor and pulsoximeter on the patients.

All the patients were pre-oxygenated with 100% oxygen for 3 minutes. Patients belonging to Group L received inj. Lignocaine 1.5 mg/kg 3 minutes before induction of general anesthesia and Group M received inj. Magnesium sulphate (50%) 40 mg/kg over 1 minute just before induction of general anesthesia. This was followed by induction of general anesthesia with inj. Thiopentone 5 mg/kg followed by inj. Succinylcholine in the dose of 1.5 mg/kg was given and the patient was ventilated through face mask with 100% oxygen. Laryngoscopy was done after stoppage of fasciculations with Macintosh laryngoscope blade and endotracheal tube was passed. Anesthesia was maintained on Oxygen (50%) and Nitrous oxide (50%) and 1% Isoflurane.

Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and SpO_2 were recorded at before giving study drug, immediately after giving study drug and at the 1^{st}, 3^{rd} and 5^{th} minute after intubation.

Statistical analysis:

The statistical significance of difference of average clinical parameters (such as HR, SBP, DBP) between two study groups has been tested using independent sample ‘t’ test after confirming the underlying normality and equal variance assumptions.

Comparison of parameters between two groups was done using unpaired ‘t’ test.

Comparison of parameters before and after giving study drug (Lignocaine and Magnesium sulphate) were analysed using paired ‘t’ test among individual groups.

The p-value less than 0.05 was considered statistically significant. All the hypotheses

were formulated using two tailed alternatives against each null hypothesis.

RESULTS

Patient characteristics of the two groups were comparable and no statistically significant differences were noted. (Table I)

Hemodynamic changes in Group L after administration of i.v. Lignocaine were studied and compared to baseline values. (Table II)

Table I: Patient Characteristics:

	Group L	Group M	p value
Age (yrs)	30. 3 ± 9.43	31.3 ± 8.76	> 0.05*****
Sex	M - 15 F – 15	M - 15 F -15	
Basal HR (bpm)	72.4 ± 4.84	73 ± 4.95	> 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*****

*** nonsignificant**

Table II: Hemodynamic Changes in Group L Immediate, 1, 3 And 5 Min after Intubation

	Baseline	after Lignocaine	1 min	3 min	5 min
HR (bpm)	72.4 ± 4.84	71.93 ± 3.23	103.73±19.96	97.13 ± 8.69	81 ± 7.20
p value		0.66*****	< 0.01#	< 0.01#	< 0.01#
SBP(mmHg)	123.43 ± 6.73	123.27 ± 6.44	146 ± 8.15	140 ± 8.19	123.87 ± 4.03
p value		0.83*****	< 0.01#	< 0.01#	0.74*****
DBP(mmHg)	79.33 ± 5.31	80.13 ± 8.17	97.93 ± 7.73	98 ± 7.70	75.27 ± 9.04
p value		0.37*****	< 0.01#	< 0.01#	<0.05#

 # significant, * non significant

Immediately after giving Lignocaine, fall in heart rate was noticed but it was not significant. There was significant rise in heart rate at 1 minute, 3 minutes as well as 5 minutes following intubation in this group as compared to baseline values.

Systolic blood pressure remained unaffected immediately after giving Lignocaine but significant rise was noticed at 1 minute and 3 minutes after intubation as compared to baseline. There was no significant difference in SBP at 5 minutes following intubation.

Diastolic blood pressure also remained unaffected immediately after giving Lignocaine but significant rise was noticed at 1 minute and 3 minutes after intubation as compared to baseline. Surprisingly significant fall in DBP was observed 5 minutes following intubation in this group as compared to baseline.

Hemodynamic changes in Group M after administration of i.v. Magnesium sulphate were studied and compared to baseline values. (Table III)

Table III: Hemodynamic Changes in Group M immediate, 1, 3 and 5 min after intubation

	Baseline	after Magnesium sulphate	1 min	3 min	5 min
HR (bpm)	73 ± 4.95	79.3 ± 5.48	81.4 ± 5.68	80.23±5.67	79.1 ± 7.92
p value		< 0.01#	< 0.01#	< 0.01#	< 0.01#
SBP(mmHg)	122.27 ± 6.03	123.27 ± 6.44	126.67±8.47	124.13±7.45	118.13±6.39
p value		0.5*****	< 0.01#	0.23*****	< 0.01#
DBP(mmHg)	77.27 ± 4.97	78.4 ± 5.16	87.07± 5.60	86.87± 5.55	72.67± 7.67
p value		0.18*****	< 0.01#	< 0.01#	< 0.01#

 # significant
*** non significant**

There was significant rise in heart rate immediately after administration of Magnesium sulphate as well as at 1 minute, 3 minutes and 5 minutes following intubation in this group as compared to baseline values.

Non-significant rise in systolic blood pressure was noticed immediately after giving Magnesium sulphate as compared to baseline but it became significant at the end of 1 minute after intubation. It remained high at the end of 3 minutes though not statistically significant as compared to

baseline. Significant decrease in SBP was found at the end of 5 minutes after intubation in this group.

Rise in diastolic blood pressure was observed. It was not significant immediately following administration of Magnesium sulphate but was definitely significant at 1 and 3 minutes after intubation. Significant decrease in DBP was found at the end of 5 minutes after intubation as compared to baseline in this group.

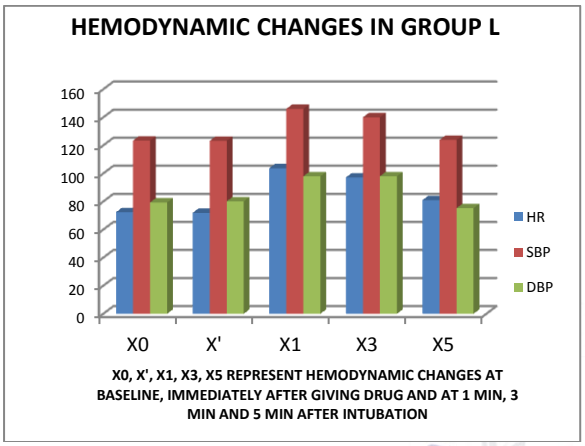

Figure1 : Haemodynamic changes in group L

The comparison of hemodynamic changes immediately after giving study drugs were done between two groups.

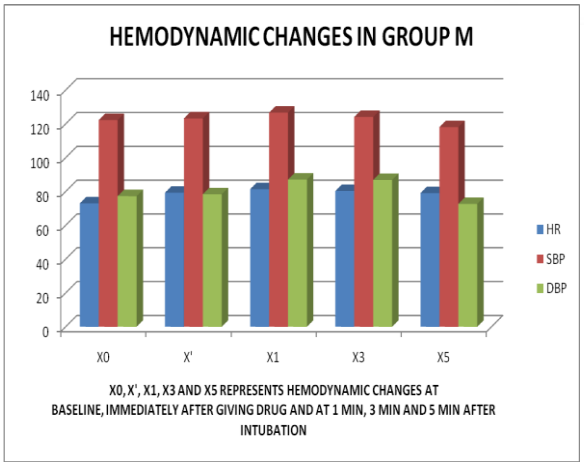

Figure 2: Haemodynamic changes in group M

The mean heart rate was 71.93 ± 3.23 bpm in Group L and 79.3 ± 5.48 bpm in Group M. There was significant rise in heart rate in Group M as compared to Group L (p value < 0.01).

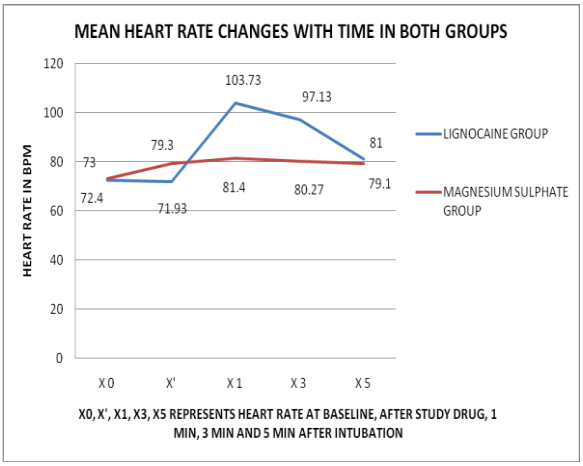

Figure 3: mean heart rate changes with time in both groups

Immediately after study drug the mean SBP of both the groups was 123.27 ± 6.44 mmHg.

Similarly mean DBP in Group L and Group M were 80.13 ± 8.17mmHg and 78.4 ± 5.16 mmHg respectively. There was no significant change in the DBP following the drug treatment (p value > 0.05).

The hemodynamic changes 1 minute after tracheal intubation were studied and compared. (Figure 3)

Mean heart rate was 103.73 ± 19.96 bpm in Group L and 81.4 ± 5.68 bpm in Group M. There was significant rise in heart rate 1 minute following intubation in Group L as compared to Group M (p value <0.01).

Mean SBP were 146 ± 8.15 mmHg and 126.67 ± 8.47 mmHg in Group L and Group M respectively. There was significant rise in SBP 1 minute following intubation in Group L as compared to Group M (p value <0.01).

The hemodynamic changes 3 minutes after tracheal intubation were taken and the two groups were compared. (Figure 3)

Mean heart rate was 97.13 ± 8.69 bpm in Group L and 80.23 ± 5.67 bpm in Group M. There was significant rise in heart rate 3 minutes following intubation in Group L as compared to Group M (p value <0.01).

Mean SBP were 140 ± 8.19 mmHg and 124.13 ± 7.45 mmHg in Group L and

Group M respectively. There was significant rise in SBP 3 minutes following intubation in Group L as compared to Group M (p value <0.01).

Mean DBP were 98 ± 7.70mmHg and 86.87 ± 5.55 mmHg in Group L and Group M respectively. There was significant rise in DBP 3 minutes following intubation in Group L as compared to Group M (p value <0.01).

The hemodynamic changes 5 minutes after tracheal intubation were studied and evaluated. (Figure 3)

Mean heart rate was 81 ± 7.2 bpm in Group L and 79.1 ± 7.92 bpm in Group M. There was no significant difference in heart rate (p value 0.17).

Mean SBP readings were 123.87 ± 4.03 mmHg and 118.13 ± 6.39 mmHg in Group L and Group M respectively. There was significant rise in SBP in Group L as compared to Group M (p value <0.01). (Figure 4)

Mean DBP readings were 75.27 ± 9.04 mmHg and 72.67 ± 7.67 mmHg in Group L and Group M respectively. There was no significant difference in DBP in both groups (p value 0.23). (Figure 5)

DISCUSSION

Laryngoscopy and intubation of trachea often evoke cardiovascular response in the form of increase in arterial blood pressure, heart rate and changes in cardiac rhythm. These responses are believed to be due to the reflex sympathetico adrenal discharge, which may put the patient at risk for cerebral haemorrhage, left ventricular failure etc. Magnesium inhibits catecholamine release both from the adrenergic nerve terminals and the adrenal medulla in vitro. The mechanism of action of Lignocaine in attenuation of this reflex hemodynamic response is due to direct cardiac depression and peripheral vasodilatation. Both these drugs have been studied in attenuating the response to intubation and have shown promising results. There have been no such studies on these drugs in our set up and these drugs being less expensive and easily available has been chosen for the study.

Abou Madi, Kieszler and Yacoub [6] showed that 1.5mg/kg of Lignocaine given i.v. before laryngoscopy was the optimal dose for attenuating this response. Using a similar anesthetic technique, Tam Chung and Campbell [7] observed complete attenuation of the response when this dose of lignocaine was given 3 minutes before laryngoscopy. C.D Miller and S.J Warren [8] showed that Lignocaine 1.5mg/kg given i.v. within 3 minutes of laryngoscopy failed to attenuate the cardiovascular response to laryngoscopy and intubation. Similarly Chraemmer-Jorgensen and co-authors [9] gave i.v. Lignocaine 1.5mg/kg beginning 2 minutes before laryngoscopy. These studies showed that Lignocaine had no significant effects on cardiovascular effects of laryngoscopy and intubation. Madi and

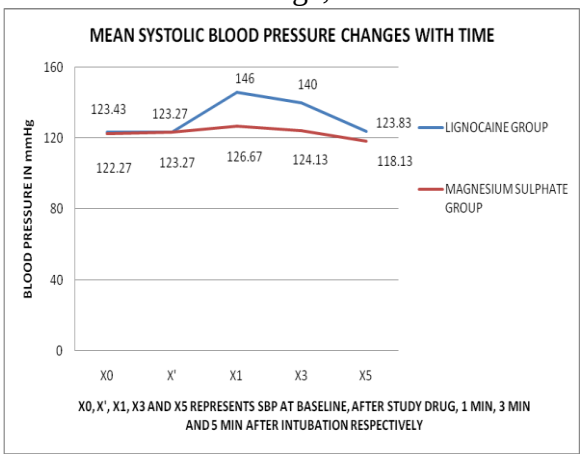

Figure 4: mean systolic blood pressure changes with time in both groups

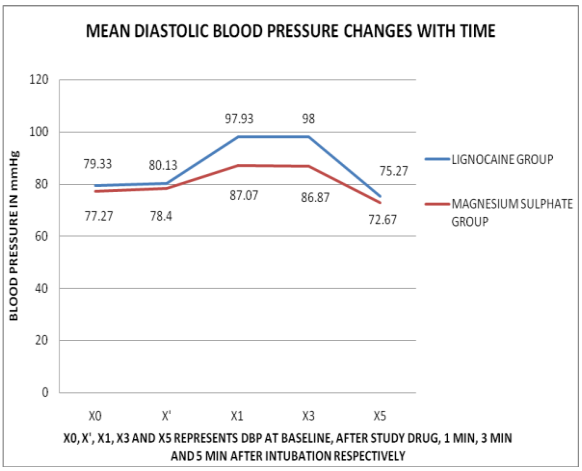

Figure 5: mean diastolic blood pressure changes with time in both groups

coauthor [6] found 3 minutes as a "borderline statistical protection" against the response to laryngoscopy and intubation, therefore Lignocaine 1.5mg /kg has been given 3 minutes prior to intubation in this study.

In the present study we have taken 60 normotensive patients of ASA grade I and randomly allocated them to receive either Magnesium sulphate (40mg/kg) over 1 minute just before induction or Lignocaine (1.5mg/kg) 3 minutes before induction and have tried to compare the efficacy of both these drugs in attenuating the response to the same. The groups were comparable with respect to their demographic and hemodynamic variables. (Observations, Table 1)

In 1989 James M F M et al [10] studied the effects of pretreatment with 60 mg/kg body weight Magnesium sulphate intravenous on the catecholamine release and cardiovascular response associated with tracheal intubation. Induction of anesthesia produced no significant changes in heart rate and blood pressure in either group. In the magnesium group, the injection of Magnesium sulphate produced an initial increase in heart rate of 13 ± 3.9 bpm; in the control group, there was no significant change in heart rate from the baseline level. Heart rate increased by 30.9 bpm 2 minutes after intubation in the control group, whereas in the magnesium group, heart rate remained virtually unchanged from post-magnesium values. The difference between groups at 2 minutes after intubation was significant. In 1998 G. D. Puri et al [11] found that HR tended to increase after Magnesium sulphate administration, but not significantly. It decreased toward the baseline after the induction of anesthesia in the magnesium group, whereas it increased significantly in the control group ($P < 0.001$). HR further increased after endotracheal intubation in both the groups. It was significantly more rapid immediately after and 3 minutes after endotracheal intubation from the baseline. In 2005 Fallah et al [12] observed no significant differences as percent base values of mean heart rate between different magnesium groups but between magnesium group and lignocaine group the differences were significant. In magnesium group (40mg/kg) upto 10% of rise in mean pulse rate was noticed at 1 and 3 minutes after laryngoscopy which dropped below baseline after 5 minutes. Whereas in lignocaine group, mean pulse rate dropped down upto 10% immediately after giving drug, it raised upto 25% immediately after intubation and did not come to baseline even at 5 minutes. In 2006 Sharma J et al [13] and in 2009 Trivedi V et al [14] found that in magnesium group (40 mg/kg) immediately after giving drug there was rise in heart rate which increased further immediately after intubation and did not come to baseline till 5 minutes post intubation. In present study, immediately after giving study drug significant rise in heart rate was seen in Group M whereas no such change was found in Group L as compared to baseline. Significant rise in heart rate was seen at 1, 3 and 5 minutes after intubation as compared to baseline in both the groups (Observations, Table 2 and 3). Rise in heart rate was significant in Group L as compared to Group M, 1 and 3 minutes after intubation, but the difference was not significant at 5 minutes. (Observations, Table 4, 5, 6 and 7).

James M F M et al [10] noticed that induction of anesthesia produced a small decrease in systolic and diastolic blood pressures in both groups. Immediately after intubation, systolic blood pressure increased in the control group but not in the magnesium group, the difference between the groups at this point being significant. 2 minutes after intubation, systolic blood pressure increased slightly in the magnesium group toward baseline levels, but in the control group systolic blood pressure increased above baseline levels ($P < 0.01$ for differences between groups). Diastolic blood pressures showed similar changes. Puri et al [11] found that MAP decreased ($P < 0.001$) after Magnesium administration alone compared with the control group (Normal Saline), ($P < 0.05$) at

preinduction stage. It decreased after the induction of anesthesia, but the decrease was significant (P < 0.01) only in the control group and there was no significant difference between the two groups immediately before intubation. MAP increased after intubation and was significantly higher in control group (lignocaine) compared with the magnesium group immediately after and 3 minutes after intubation (P < 0.01). Compared with baseline, the postintubation MAP values were significantly higher only in the control group (P < 0.01). Fallah et al [12] found that in magnesium group (40mg/kg) upto 20% rise in mean arterial pressure was noticed at 1 and 3 minutes after laryngoscopy which dropped below baseline values after 5 minutes. Whereas in lignocaine group, mean arterial pressure dropped down upto 10% of baseline immediately after giving drug, it raised upto 30% immediately after intubation and did not come to baseline till 5 minutes. Sharma J et al [13] also noticed drop in SBP and DBP immediately after giving Magnesium which remained below baseline till 5 minutes. Vandana T et al [14] found decrease in MAP immediately after giving magnesium which remained below baseline till 5 minutes.

In 2013 Navid Nooraei et al [15] found magnesium sulphate as more effective than lignocaine in controlling hemodynamics although it may increase the HR.

In 2016 Narmatha et al [16] found that using 30 mg/kg of magnesium sulphate there was a reduction in the HR and MAP (P<0.05) which was statistically significant when compared with lignocaine group and concluded that magnesium sulphate in the dose of 30 mg/kg given IV 1 min prior to induction, attenuates the cardiovascular response to laryngoscopy and intubation in a better manner than lignocaine.

In present study, In Group L, systolic blood pressure remained unaffected immediately after giving Lignocaine but significant rise was noticed at 1 minute and 3 minutes after intubation as compared to baseline. However there was no significant rise in SBP at 5 minutes in this group. In Group M nonsignificant rise in systolic blood pressure was noticed immediately after giving Magnesium sulphate as compared to baseline but it became significant at the end of 1 minute after intubation. It tends to return towards baseline at 3 minutes. SBP dropped below baseline significantly at the end of 5 minutes after intubation in Group M. Immediately after study drugs, SBP remained unaffected. Significantly higher SBP was recorded in Group L as compared to Group M at 1, 3 and 5 minutes after intubation. Diastolic blood pressure also remained unaffected immediately after giving Lignocaine but significant rise was noticed at 1 and 3 minutes after intubation as compared to baseline in Group L. In Group M non significant rise in DBP was observed immediately following administration of Magnesium sulphate but the rise was definitely significant at 1 and 3 minutes after intubation as compared to baseline. There was no significant difference in DBP in both groups after giving study drug. The rise in DBP was significantly higher in Group L as compared to Group M at 1 and 3 minutes after intubation. However there was fall in DBP from baseline after 5 minutes in both the groups but there was no significant difference between the groups. (Observations, Table 4, 5, 6 and 7).

Calcium exerts a major role in stimulus-response coupling, including the release of catecholamines from the adrenal gland and adrenergic nerve terminals in response to sympathetic stimulation. Magnesium, because it competes with calcium for membrane channels, has been described as the physiological calcium antagonist [17] and can modify many calcium-mediated responses. The present study shows that magnesium can significantly attenuate the output of catecholamines at the time of tracheal intubation and thus reduces the severity of cardiovascular disturbances. The

cardiovascular effects observed in this study were particularly interesting. It might be expected that magnesium would slow the atrial rate by inhibiting the calcium mediated depolarizing current in pacemaker tissue, an effect that has been demonstrated in isolated animal hearts. [18] However, in the intact animal the ability of Magnesium to inhibit the release of acetylcholine from the vagus nerve predominates [19] and, therefore, the overall effect is the increase in heart rate seen in this study. Although heart rate in the lignocaine group was considerably less than that in the magnesium group prior to intubation, the heart rate after intubation in this group was significantly higher than in the magnesium group. This was presumably due to the fact that in the lignocaine group there was a significant increase in epinephrine levels as compared to magnesium group. In the present study, increases in both systolic and diastolic blood pressure were less in the magnesium group than in the lignocaine group. The improved control of blood pressure in the magnesium group was probably, therefore, due to a combination of vasodilatory effects of the ion and inhibition of catecholamine release. It might be argued that magnesium is producing its effect by a central sedative mechanism, but this is unlikely as magnesium crosses the blood-brain barrier with difficulty and has little or no central sedative effect even at much higher serum levels. [20] The actions of magnesium in protecting against the potentially harmful cardiovascular effects of tracheal intubation are probably not superior to the actions of the potent short-acting opiate agents, Fentanyl and Alfentanil. Alfentanil in particular shows considerable promise in this regard. [21] However, the use of opiates has been associated with muscle rigidity, bradycardia, hypotension, and respiratory depression. In circumstances in which these complications may be undesirable, magnesium could be a useful alternative. A serum magnesium level of 2-4 mmol/L at the time of endotracheal intubation may be particularly valuable in the hypertensive pregnant patients. Magnesium has also been shown to reduce fasciculations [22] and potassium release [23] after succinylcholine, and these actions combined with the cardiovascular control that can be achieved by the use of magnesium may be of value. Magnesium does not appear to prolong the duration of action of Succinylcholine, [13,24] but the interaction between magnesium and the nondepolarizing relaxants must be borne in mind if this technique is to be used in combination with these latter drugs. If this combination is to be used, then the dosage of relaxant should be reduced. [23,24] There is currently no data available as to the effect that Magnesium sulphate might have on the onset, time or intensity of block achieved when non-depolarizing relaxants are used to facilitate tracheal intubation. [23,24]

CONCLUSION

So, it is concluded that (1) Magnesium sulphate provides fairly good and sustained control over rise in systolic and diastolic blood pressure during tracheal intubation but rise in heart rate is no significantly mitigated. (2) Lignocaine poorly controls rise in systolic and diastolic blood pressure and does not prevent rise in heart rate. (3) Magnesium sulphate provides better control over rise in HR, SBP and DBP during laryngoscopy and tracheal intubation in ASA grade I patients as compared to Lignocaine.

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