Original Article

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Comparitive Study of the Effect of IV Magnesium Sulfate and IV Lignocaine on Hemodynamic Response to Laryngoscopy and Endotracheal Intubation

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Abstract

To compare the efficacy of IV Lignocaine (preservative free) (1.5mg/kg body weight) and IV Magnesium sulphate (20 mg/kg body weight) in elective surgery under general anesthesia for controlling the hemodynamic response to laryngoscopy and endotracheal intubation. It can be observed from the present study, the two groups were comparable for age, sex, weight and ASA status of the patients. Magnesium sulphate at 20 mg/kg bodyweight produced significant decrease in heart rate and blood pressure than lignocaine group. The mean heart rate was 92.16 + 10.98 and 123.84 + 8.78 per minute(p=0.0001) at one minute post intubation in Magnesium sulphate group and Lignocaine group respectively. The mean systolic pressure was 130.92 + 8.17 mm of Hg and 158.20 + 4.76 mm of Hg (p=0.0001) and the mean diastolic pressure was 87.64 + 5.7 mm Hg and 99.28 + 4.3 mm of Hg (p=0.0001) at one minute post intubation in Magnesium sulphate group and Lignocaine group respectively. It can be observed from the above result that Magnesium sulphate (in a dose of 20 mg/kg body weight) 3 minutes prior to laryngoscopy when compared to lignocaine (in a dose of 1.5 mg/kg body weight) 3 minutes prior to laryngoscopy causes greater attenuation of stress response to laryngoscopy and endotracheal intubation resulting in greater reduction of heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and rate pressure product thus causing better hemodynamic stability and thus proving beneficial in patients with hypertension or history or coronary artery disease making it a desirable choice for such patients.

Keywords: Magnesium sulphate; Lignocaine; Intubating and Anaesthesia.

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Introduction

Airway management is the most important thing during delivery of general anaesthesia. Patients who have been anaesthetized are unable to maintain an adequate airway on their own and artificial airway maintenance devices are employed.[1] Traditionally, endotracheal intubation has been the mainstay in providing adequate airway management and delivering anaesthesia.

Reid and Brace (1940) were the first to report the circulatory response to laryngeal and tracheal stimulation in anaesthetized man; those circulatory responses were tachycardia and rise in arterial blood pressure.[2] Takeshima et al[3] (1967) found rise in mean arterial blood pressure of 20 mmHg at the time of laryngoscopy and tracheal intubation, and they concluded that laryngoscopy was a more potent stimulus to hypertension than intubation.

Increase in blood pressure and heart rate occurs most commonly from reflex sympathetic discharge in response to laryngotracheal stimulation, which in turn leads to increased plasma norepinephrine concentration.[4]

Direct laryngoscopy and intubation usually induces a cardiovascular stress response characterized by hypertension, tachycardia & arrhythmias due to reflex sympathetic stimulation and thereby increases the myocardial oxygen demand. The response in transient occurring 30 seconds after intubation and lasting for less than 10 minutes.[5]

Stress response may be well tolerated in healthy patients but may be dangerous in patients with coronary artery disease, hypertension, myocardial infarction, cerebrovascular disease and thyrotoxicosis.[6]

There are various techniques by which this intubation-related stress response can be attenuated, all of which depend on reduction in input stimuli or the blockade of adrenergic responses.

So far, various techniques like topical and intravenous Lignocaine, opioids[7-9], deep inhalational anaesthesia, Adrenergic blockers (β blockers)[10-16], α2 agonists, Calcium channel blockers (nifedipine), vasodilators like nitroglycerine[17], Magnesium Suphate have been used. Local anaesthetics such as lignocaine and chloroprocaine were tried in both forms topical as well as intravenous, where intravenous lidocaine and chloroprocaine showed promising results. The postulated mechanism of IV local anaesthetics in inhibiting sympathetic response associated with intubation appears to result from an increased threshold for airway stimulation, central inhibition of sympathetic transmission and direct depression of cardiovascular response. In addition to its local anaesthetic action, lignocaine reduces the anaesthetic requirement by 30% with a 1.5mg/kg body weight IV bolus that is minimally depressive to the cardiovascular system.[18-22] Magnesium Sulphate has been described as the physiological calcium antagonist[23] as it competes with Ca for membrane channels and can modify many calcium - mediated responses. Magnesium ions can inhibit the release of catecholamine from adrenal glands and peripheral adrenergic nerves terminals[26] and has direct vasodilating properties.

I.V. Magnesium Sulphate has been extensively tried with reasonable margin of safety in management of Pregnancy induced hypertension.[24]

Catecholamine release inhibition[25] and vasodilatation properties of $MgSO_4$ prompted us to study its effect on pressor response to Laryngoscopy and intubation.

Not many clinical literatures are available comparing Lignocaine and Magnesium sulphate for attenuation of hemodynamic stress response to Laryngoscopy and endotracheal intubation.

Pharmacology of Lignocaine[28-30]

Lignocaine is the most commonly used local anaesthetic agent. It is a tertiary amide which is effective in regional anaesthesia and as cardiac antidysrhythmic.

Chemistry

Lignocaine is diethylamino - 2, 6 - acet - oxylidide. It is an amide obtained from reaction of diethylamino acetic acid and xylene.

Structure: It consists of liphophilic aromatic group, hydrophilic tertiary amine and intermediate amide bond.

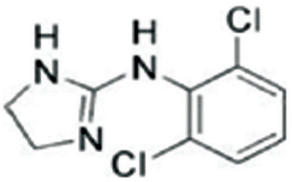


Fig. 2:

Molecular weight Base - 234.

Hydrochloride salt – 270

Physiochemical and Pharmacological Properties:

Freely soluble in water. Very stable, can be boiled for 8hrs in 30% HCL. Can be autoclaved for 6 hours or multiple times without loss of potency. p Ka - 7.9.

Volume of distribution - 91 Liters. Plasma clearance - 0.95 liters/min. Elimination half life - 96 minutes.

Mechanism of Action: Sodium channel is a specific receptor for the action of lignocaine. It acts by preventing transmission of impulses through neural or myocardial cell membrane.

Selective binding of sodium channels in inactivated closed states and their stabilization prevents conversion of these channels to activated open states resulting in inhibition of conduction of action potential. Site of binding is to H' gates present at inner portion of sodium channel.

In the heart: Phase 4 depolarization is delayed in ventricular cells. It fails to delay phase 4 depolarization in atrial cardiac cells. It can hyperpolarize purkinje fibers. Threshold for excitability is altered. Rate of depolarization of action potential is slowed such that it fails to reach threshold potential

Distribution

After intravenous administration plasma concentration is decreased due to initial high uptake into lungs and its distribution to highly perfused tissues like brain, heart and kidneys.

Pharmacodynamics

Lignocaine blocks the conduction of impulses in the nerve fibres at the site of injection by closing sodium channels. Sensory and motor fibres are inherently equally sensitive to lignocaine. Smaller fibres and nonmyelinated nerve fibres are blocked more easily than longer and myelinated fibres.

Autonomic fibres are more susceptible than somatic fibres. Among somatic fibres order of blockade is, pain temperature touch deep pressure sense. Addition of vasoconstrictor eg. Adrenaline (1:50,000 to 1:200000) can prolong the duration of action of lignocaine by decreasing the rate of removal from the local site of injection in to the circulation and reduces the systemic toxicity; of lignocaine by decreasing the rate of absorption and keeping the plasma concentration lower. It is very effective surface analgesic causing rapid absorption from mucosal surface. The peak blood concentration is achieved within 4 to 15 minutes after instillation. Given intravenously, peak blood levels are achieved immediately.

Pharmacology of Magnesium[31-33]

It is a sulphuric acid Magnesium salt (1:1) heptahydrate.

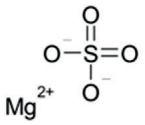


Fig. 3:

Magnesium is the fourth abundant cation in the body and the second most plentiful intracellular cation after potassium. Because of its numerous physiologic activities, magnesium is called "nature's physiological calcium channel blocker"

Mechanism of Action

Magnesium Sulphate acts as a non competitive inhibitor of the IP3 - gated calcium channel and of IP3 binding and decreases intracellular calcium.

Physiological Actions of Magnesium

It is an important calcium regulator at both intracellular and extracellular sites. Magnesium (Mg) is essential for the release and action of Parathormone and Mg deficiency impairs the mobilization of calcium from bone.

Mg is an essential cofactor for the Na+K+ATPase system and thus is an important regulator of transmembrane electrical potential. It is necessary for normal energy metabolism and in its absence weakness occurs. It is also essential for the regulation of calcium in the myocardium. It is important in the release of neurotransmitters and certain hormones, notably thyroid stimulating hormone and insulin, and in the degranulation of mast cells.

Pharmacokinetics

Distribution of Mg to the intracellular space is partly determined by the initial level of magnesium stores, which can vary widely. Volume of distribution rapidly increases with a constant infusion, suggesting that active uptake of Mg into the intracellular space is an important component of the redistribution process. Thus a loading dose of Mg is required to raise the plasma concentration. Absorption of Mg following deep intramuscular injection is relatively slow, with a peak plasma concentration being achieved at around 60 min. The sole route of elimination of Mg is by renal clearance. Urinary excretion of Mg is rapid and is a linear of plasma concentration

Pharmacodynamics

Cardiovascular system Mg reduces systemic vascular resistance with a compensatory increase in cardiac output. At higher plasma concentration

it produces bradycardia. Mg has a major role in management of cardiac rhythm disturbances like ventricular tachycardia and fibrillation, torsades pointes, multifocal atrial tachycardia, dysrhythmias associated with digitalis, hypokalemia, alcoholism, myocardial infarction, cardiac surgery and catecholamine release. The vasodilatory and antidysrhythmic effects of Mg have been presumed to protect against hypertensive responses to direct laryngoscopy and tracheal intubation, management of patients undergoing resection of pheochromocytoma, and cross clamping of the abdominal aorta.

Mg may act as cardioprotective drug by attenuating the increase in intracellular calcium ion flux that accompanies myocardial ischaemia followed by reperfusion.

Central nervous system Neurophysiological studies have demonstrated that Mg is a physiological and pharmacological blocker of NMDA receptors in neuronal tissue. As the role of the NMDA receptor in pain perception has become apparent, there is increasing use of Mg for the management of both acute and chronic pain.

Mg inhibits calcium entry into neuronal cells at a variety of calcium channels including acting as an NMDA antagonist. Mg penetrates the blood brain barrier poorly. Parenterally administered drug has little CNS depressant effects. Its anticonvulsant action in management of preeclampsia may be due to cerebral vasodilator action.

Peripheral nervous system Mg may potentiate the action of local anesthetics. Autonomic nerve terminals Mg progressively inhibits release of catecholamines from both adrenergic nerve terminals and adrenal medulla.

Respiratory system Because of its smooth muscle relaxant properties Mg is a bronchodilator. Genitourinary system It is used as a tocolytic for the management of premature labour. It is also effective renal vasodilator, and exerts a significant diuretic effect

Musculoskeletal system Mg produces dose dependant, presynaptic inhibition of neuromuscular release in peripheral nerves by competing with calcium for membrane channels on the presynaptic terminal. It potentiates the action and prolongs the duration of all nondepolarizing muscle relaxants.

Plasma magnesium levels

1.5 – 2.2 mEq/l	-	normal plasma concentration
4 – 6 mEq/l	-	Therapeutic levels.
6 – 10 mEq/l	-	Prolonged P-Q interval. Widened QRS complex
10 mEq / l	-	loss of deep tendon reflexes
15 mEq / l	-	Sino atrial, AV nodal block Respiratory paralysis.
25 Eq/l	-	Cardiac arrest

Contraindications: In patients with Renal Failure, Heart Block, Extensive myocardial damage.

Interaction/Toxicity: Potentiates depolarizing and non - depolarizing muscle relaxants.

Potentiates central nervous system depressant effect of sedatives, narcotics and volatile anaesthetics.

Materials and Methods

The study was conducted in a tertiary care hospital and was approved by the hospital ethics committee and conducted as a Prospective Randomized Double Blind Placebo Controlled Study on 50 adult patients. Sample size is selected based on the calculation according to EPI into 6 versions of guidelines issued by certificate for disease control (CDC) Atlanta, Georgia, United States with population size of 1000 and with high expectation of 10% and with low expectation of 0.05%.

Inclusion criteria: Patients with Mallampatti airway grade I and II of ASA I & II undergoing elective surgery requiring general anesthesia and not on elective ventilation of Age 15-50 years belonging to both sexes and weighing between 50-100 kgs.

Exclusion criteria: Non consenting patients/relative refusal for procedure. Patients with medical comorbidities like hypertension, ischemic heart diseases arrhythmias & uncontrolled diabetes. Patients with Mallampatti III and IV. Expected difficult intubation .If patient is allergic to any of these drugs. Pregnant and lactating women. Patients with acute and chronic renal failure, compromised cardiovascular function, severely deranged liver function, endocrine disorders or on long term drug therapy with antipsychotics, antidepressants, anxiolytics. Hemodynamically compromised patients and Cases taken on an emergency.

Technique

A detailed pre-anesthetic evaluation including history of previous medical illness, previous surgeries, general examination and appropriate baseline investigations was carried out. An informed written consent was obtained. Nil by mouth status

for 6 hrs preceding surgery was confirmed. Patients were re-examined on table, baseline values of pulse, blood pressure were recorded with the help of Non invasive blood pressure (NIBP) monitoring system and pulse oximeter. Examination of the cardiovascular and respiratory system was done. Intravenous access was obtained with 18G venous cannula.

These fifty patients were randomly allocated to two groups of 25 each using lottery method as described below (group M and L). Group M (n=25) - received magnesium sulphate 20mg/kg diluted over 10cc normal saline intravenously 1 minute prior to the IV injection of Vecuronium.

Group L (n=25) - received lignocaine 1.5mg/kg diluted over 10cc normal saline intravenously 1 min, prior to the IV injection of Vecuronium.

Bias removal: Both the study drugs were prepared as clear colourless 10 ml solutions containing either magnesium sulfate 20mg/kg or lignocaine 1.5mg/kg. Airway management and tracheal intubation was carried out by anesthetist who was unaware of group allocation. Double Blind procedure is where not only the patient, but also the investigator is unaware which group of patient or patients are given drug or drugs and which group is on placebo. Both placebo and drug are labeled accordingly by the principle investigator.

Premedication: inj Glycopyrrolate 5 mcg/kg body weight, Ondansetron 0.1mg/kg body weight, Midazolam 0.02mg/kg body weight, Fentanyl 1 mcg / kg body weight given 15 mins prior to induction.

Induction: Patients were preoxygenated with 100% oxygen for 3 minutes, pre-induction heart rate, blood pressure, oxygen saturation(SpO_2) were recorded and considered as baseline, followed by Intravenous Propofol 2 mg/kg till the eyelash reflex is absent and mask ventilation was done, study drug [Group M- inj Magnesium Sulphate 20mg/kg; Group L – inj Lignocaine 1.5mg/kg; diluted over 10cc normal saline intravenously] injected over 1 minute, followed by intravenous Vecuronium 0.01 mg/kg as neuromuscular blocking agent, anaesthesia was maintained with 50% N2O, 50% O2 and isoflurane (0.6% - 1%) for 3 minutes.

Soon after induction, SBP, DBP, MAP & RPP, heart rate, and SpO_2 were recorded and after 3 minutes of the study drug, endotracheal intubation was done. Laryngoscopy was performed with a standard Mac-intosh curved laryngoscope blade and smooth single attempt oro-tracheal intubation with an appropriate size cuffed endotracheal tube was performed (no: 7 for Female patients and no: 8 for Male patients. Adverse effects during laryngoscopy like hypotension, bradycardia, desaturation & ECG changes if any were recorded. SBP, DBP, MAP & RPP, heart rate and SpO_2 were again recorded at intubation followed by 1, 3, 5, 10 and 15 minutes post intubation. Intraoperatively anaesthesia was maintained with isoflurane (0.5 to 1.2%), N_2O (50%), O_2 (50%) and intermittent intravenous Vecuronium by using closed circuit on mechanical ventilation.

At the end of surgery, patient was reversed with Glycopyrrolate and Neostigmine 8 mcg/kg and 0.05mg/kg respectively followed by extubation after thorough oral suctioning. Post extubation patient was shifted to anaesthesia recovery room and vital parameters were monitored.

Following patients were eliminated from our study:

- Laryngoscopy duration>25 sec. Failiure to intubate in the first attempt.

Results

Statistical Analysis

Data was compiled and continuous data are presented as Mean & SD and categorised data as percentages. Demographic characterstics, perioperative vitals were compared using students't' test and nominal data were compared with Chi Square test. Repeated measures of analysis of variance (RMANOVA) were used to compare continuous variables. Statistical analysis was performed using GRAPH.PAD INSTAT Software Package. A 'p' value of <0.05 was considered statistically significant.

Observation and Result

In our study, 50 patients of ASA grades I and II of age group between 18–60 years posted for various surgical procedures requiring general anaesthesia were included in the study to compare the effects of intravenous magnesium sulphate and intravenous lignocaine on hemodynamic response to laryngoscopy and endotracheal intubation.

Group M (n=25) - received magnesium sulphate 20mg/kg body weight intravenously 1 min, prior to the IV injection of Vecuronium.

Group L (n=25) - received lignocaine 1.5mg/kg body weight intravenously 1 min, prior to the IV injection of Vecuronium.

There is no significant difference between age and weight of patients of both the groups (L & M) since p value > 0.05. (Table 1).

Table 1: Age & Weight distribution of amongst study population.

Age & weight	Group				P value
	Lignocaine		$MgSO_4$		
	Mean	SD	Mean	SD	
Age	40.36	11.940	36.76	10.465	0.263
Weight	53.80	6.225	55.40	7.461	0.414

28 females accounting for 56% of total sample size as compared to 22 males (44%). Since p value is greater than 0.05 there is no significant difference between sexes in both groups. (Table 2).

Table 2: Gender distribution of patients amongst study population.

			Group		Total
			Lignocaine	$MgSO_4$	
Sex	Female	Count	15	13	28
		% within Group	60.0%	52.0%	56.0%
	Male	Count	10	12	22
		% within Group	40.0%	48.0%	44.0%
Total		Count	25	25	50
		% within Group	100.0%	100.0%	100.0%

Table 3: Distribution of ASA grade in different study groups.

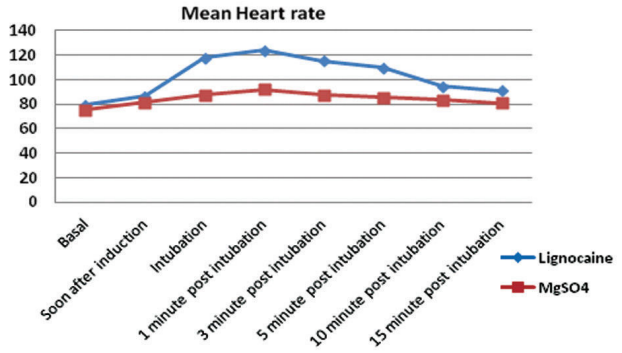
			Group		Total
			Lignocaine	$MgSO_4$	
ASA	I	Count	12	16	28
		% within Group	48.0%	64.0%	56.0%
	II	Count	13	9	22
		% within Group	52.0%	36.0%	44.0%
Total		Count	25	25	50
		% within Group	100.0%	100.0%	100.0%

In group L (Lignocaine),12 patients out of 25 belongs to ASA I and 13 out of 25 patients belongs to ASA II (Table 3) In ASA II, 10 patients were hypertensive, well controlled with medications and 5 patients were Diabetic on oral hypoglycaemic drugs. In group M ($MgSO_4$), 16 patients out of 25 belongs to ASA I and 9 out of 25 patients belongs to ASA II (Table 3) In ASA II, 10 patients were hypertensive, well controlled with medications and 2 patients were diabetic on oral hypoglycaemic drugs.

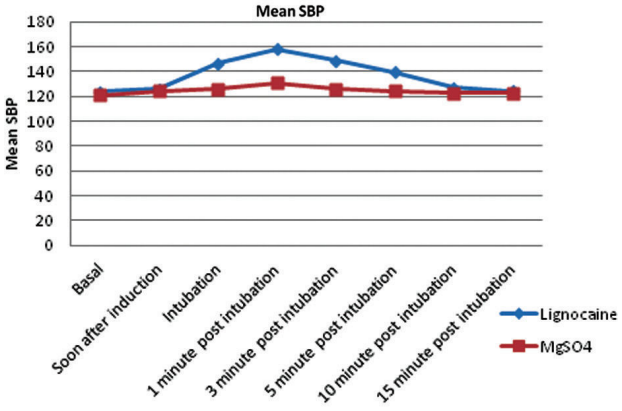
Using chi square test, p = 0.436, it shows that there is no statistical significance (P >0.005) for ASA grading between group L & M.

There was no statistically significant difference (p=0.544) between the baseline mean heart rate of both the groups. The mean baseline heart rate in Group L was 79.21 ± 22.67 beats/min, whereas that in group M was 75.36 ± 21.44 beats/min. (Graph 4).

Graph 4: Comparison of HR (beats/min) in different study groups at various time interval.



Graph 5: Comparison of SBP (in mm of Hg) in different study groups at various time interval.



In Group L, the heart rate increased during intubation (T3) and remained high till 5 minute post intubation (T6) and became near normal at 15 minute post intubation. (Graph 4) In Group M, the heart rate didn't rise significantly. Marginal rise was observed during intubation (T3) and 1 minute post intubation (T4). During Intubation (T3), heart rate increased by 38.79 % & 12.40 % above the baseline in Group L and Group M respectively which is statistically significant (p=0.0001).

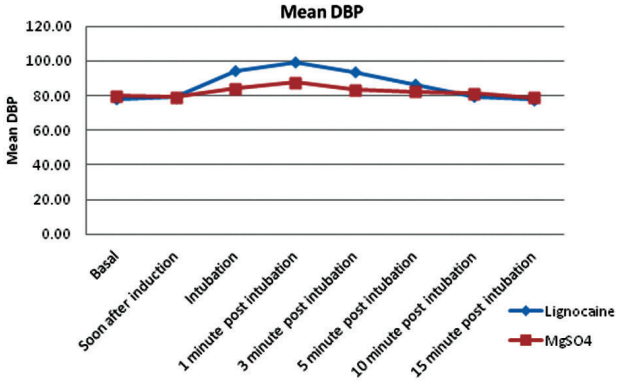
At 1 minute post intubation, there was 44.63 % rise in heart rate from baseline in Group L as compared to 16.80 % rise in Group M which is statistically significant (P=0.001). After 1 minute post intubation, both groups showed significant fall in heart rate till T8 where it became near normal to the baseline. (Graph 4)

At Intubation (T3), SBP increased by 22.96 % and 4.56 % above the baseline in Group L & Group M respectively which is statistically significant

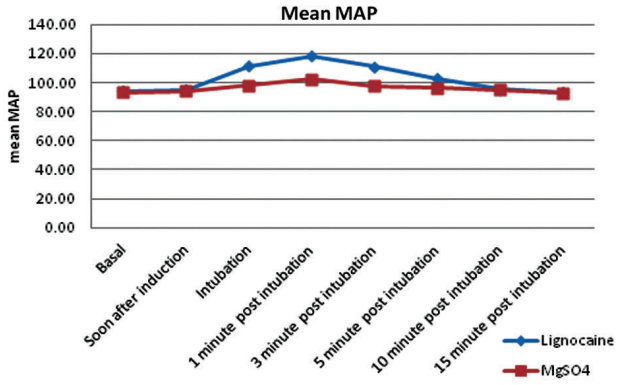
(p=0.001). At one minute post intubation(T4), both group L & M showed rise in SBP by 34.64% & 9.76 % respectively which is statistically significant (p=0.0001). At 15 minute post intubation (T8), SBP didn't rise significantly and remained relatively stable around the baseline in both the groups. (Graph 5).

At Intubation (T3), DBP increased by 16.56 % and 4.32 % above the baseline in Group L & Group M respectively which is statistically significant (p=0.001). At one minute post intubation(T4), both group L & M showed rise in DBP by 21.36 % & 7.88 % respectively which is statistically significant (p=0.001).

Graph 6: Comparison of DBP (in mm of Hg) in different study groups at various time interval.



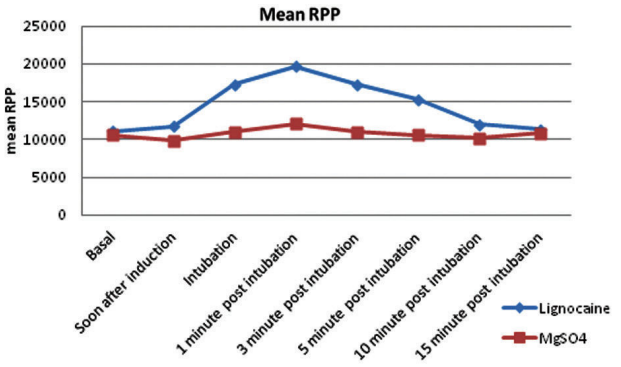
Graph 7: Comparison of MAP (in mm of Hg) in in different study groups at various time interval.



At 15 minute post intubation (T8), DBP didn't rise significantly and remained relatively stable around the baseline in Group L while in Group M, DBP showed marginal drop of 0.92%. This is not statistically significant.(p value = 0.492). (Graph 6)

During intubation (T3), both Group L & Group M showed rise of MAP of 17.52 % & 4.88% respectively from baseline which is significant (p value=0.001). The MAP continued to rise till 1 minute post intubation in both Group L & Group M BY 24.32 % & 9 % respectively from baseline which is significant(p=0.0001). The difference between the two groups was significant at time points (T5, T6) with p value< 0.05.The MAP became near normal i.e. 93.28 ± 5.2 mm Hg & 92.88 ± 3.3 mm Hg in Group L & Group M respectively by 15 minute post intubation (T8). (Graph 7).

Graph 8: Comparison of RPP in in different study groups at various time interval.



The RPP was calculated by the product of mean systolic blood pressure and mean heart rate.

The mean pre operative baseline Rate Pressure Product (RPP) in Group L was 11072 ± 1588.348 and that in Group M was 10567.48 ± 1236.985 which were comparable and the difference was not statistically significant (p=0.2156). When RPP of both the groups was compared the following observations were made:

During intubation (T3), both Group L & Group M showed rise of RPP from baseline which is significant. (p value=0.0001). The MAP continued to rise till 1 minute post intubation (T4) in both Group L & Group M respectively from baseline which is significant. (p=0.001). The difference between the two groups was significant at time points (T5, T6, and T7) with p value< 0.05. (Graph 8).

The RPP became near normal in Group L & Group M respectively by 15 minute post intubation.

Discussion

Laryngoscopy and endotracheal intubation is associated with rise in heart rate, blood pressure and incidence of cardiac arrhythmias.[47] It also results in increased myocardial oxygen demand. An optimal balance of myocardial oxygen demand and supply is of great importance and it is important to monitor parameters such as Rate Pressure Product which generally is an indicator of myocardial oxygen demand. Several drugs belonging to different pharmacological classes may be effective in either attenuating or obliterating the pressor response to laryngoscopy and intubation

like topical and intravenous Lignocaine, opioids[7,8,9] deep inhalational anesthesia, Adrenergic blockers (β blockers) 10-16, α 2 agonists, Calcium channel blockers, vasodilators like Nitroglycerine30 & Magnesium Suphate. In our study we used lignocaine in a dose of 1.5mg/kg, 3 minutes prior to laryngoscopy and endotracheal intubation. Similar dose was used by Robert K Stoelting et al57who suggested that, Intravenous Lignocaine given in the doses of 1.5 mg/kg 3 minutes before laryngoscopy and intubation, sufficiently attenuate the laryngoscopic responses.

K. Montazeri et al studied a dose response study of magnesium sulphate in suppressing cardiovascular responses to laryngoscopy & endotracheal Intubation and concluded that, the dose of 20mg/Kg of magnesium sulphate was the most effective with the less unexpected effects. So in our study IV magnesium sulphate 20mg/kg 3 minutes before laryngoscopy and endotracheal intubation was used.

We have included patient having ASA class 1 & 2 as it is safe to study the effect of a new drug in these patients and the drug effect to be studied will not be biased by the pre-existing disease or medication. At 1 minute post intubation (T4), there was 56% rise in mean heart rate from baseline in Group L as compared to 22% rise in Group M which is statistically significant (P=0.0001).

After 1 minute, both groups showed significant fall in heart rate till T8 where it became near normal to the baseline in group M but not in group L. (Graph 4) In Aug 2014, Mehrdad Mesbah Kiaee et al36 studied the effect of intravenous magnesium sulphate and lidocaine in hemodynamic responses to endotracheal intubation in elective coronary artery bypass grafting and concluded that both Lidocaine and Magnesium Sulphate lead to similar reduction in HR after endotracheal intubation (9 bpm). In group L (Lignocaine) the basal value of mean Systolic blood pressure, Diastolic blood pressure, and Mean arterial pressure was 123.56 mm Hg, mm 77.92 Hg, and 94.04 mm Hg respectively.

Following laryngoscopy and intubation, the maximal rise in mean Systolic blood pressure (SBP) was found to be 34.64 mm Hg (28%), that of Diastolic blood pressure (DBP) was 21.36 mm Hg (27%) and that of Mean arterial pressure (MAP) was 24.32 mm Hg (26%). (Graph 5,6 & 7 respectively)

Mounir Abou- Madi et al.[20] noticed the change in SBP by 30 mm Hg, change in the DBP was 22 mm Hg while CD Miller noticed an increase in SBP to be 33 mm Hg, DBP to be 37 mm Hg & Splinter et al. noticed a change in SBP to be 26 mm Hg, DBP to be 41 mm Hg and change in MAP to be 44 mmHg. In group M ($MgSO_4$) the basal value of Systolic blood pressure (SBP), Diastolic blood pressure (DBP), and Mean arterial pressure (MAP) was 121.16 mm Hg, mm 79.76 Hg, and 93.12 mm Hg respectively. Following laryngoscopy and intubation, the maximal rise in Systolic blood pressure (SBP) was found to be 9.76 mm Hg, that of Diastolic blood pressure (DBP) was 7.88 mm Hg and that of Mean arterial pressure (MAP) was 9 mm Hg. These elevated pressure readings started coming down by 3 minutes. However they remained above the baseline value even at the end of 5 minutes and reached baseline at 15 mins.

In the study of Dr. Santosh Kumar et al using $MgSO_4$ i.v 30mg/kg 3 minutes before laryngoscopy and intubation to blunt the pressor responses, found out the maximal increase in SBP was 5 mm Hg, DBP was 4 mm Hg and MAP to be 4mm Hg post intubation. In our study highest mean rate pressure product was observed at 1 minute post intubation in Lignocaine group (19652.44±1781.67) as compared to Magnesium sulphate group (12058.36±1545.54). The difference being statistically significant (p = 0.0001). (Graph 8).

Conclusion

Thus to conclude Magnesium Sulphate (in dose of 20mg/kg IV bolus, 3 minutes prior to laryngoscopy) when compared to Lignocaine (in dose of 1.5mg/kg IV bolus, 3 minutes prior laryngoscopy) causes greater attenuation of stress response to laryngoscopy and endotracheal intubation resulting in greater reduction of HR, SBP, DBP, MAP & RPP thus causing better hemodynamic stability. In patients with hypertension and history of coronary artery disease, keeping Rate Pressure Product (RPP) low is beneficial. Hence intravenous Magnesium Sulphate in dose of 20mg/kg, 3 minutes prior laryngoscopy and endotracheal intubation may be recommended to blunt the hemodynamic response to laryngoscopy and intubation in normal patients and also in patients with hypertension and coronary artery disease.

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