

# A randomized double-blind comparative study between efficacy of magnesium sulphate 30 mg/kg and 2% lignocaine 1.5 mg/kg in attenuating cardiovascular response to laryngoscopy and endotracheal intubation

<sup>1</sup>Dr. B Sunitha, <sup>2</sup>Dr. T Tejaswini, <sup>3</sup>Dr. G Alekhya, <sup>4</sup>Dr. P Sateesh

<sup>1,2,3</sup>Assistant Professor, Department of Anesthesiology, Government Medical College, Mahabubnagar, Telangana, India

<sup>4</sup>Assistant Professor, Department of Anesthesiology, Viswabharathi Medical College, Kurnool, Andhra Pradesh, India

## Corresponding Author:

Dr. T Tejaswini

## Abstract

The standard technique of laryngoscopy and endotracheal intubation involves the stimulation of Larynx, Pharynx, Epipharynx and trachea, which are extensively innervated by Autonomic nervous system, namely the parasympathetic innervation via vagus and glossopharyngeal nerves and sympathetic via superior cervical ganglion. All patients were explained in detail about the study and informed consent was taken. Patients were randomly allocated to receive intravenous Magnesium sulphate infusion 30 mg/kg, 15 minutes before induction of anaesthesia or injection Lignocaine 1.5mg/kg intravenously 90 seconds before intubation. All patients had an peripheral intravenous line secured in the pre-operative holding area. Randomization was done by picking lots. The Anaesthesiologist who prepared and administered the drug was not involved with the intra and post-operative management of the patient. We conclude that Magnesium sulphate and Lignocaine are effective in blunting the hemodynamic response to intubation, but Magnesium sulphate is superior to Lignocaine in blunting the hemodynamic response to laryngoscopy and endotracheal intubation without any significant side effects.

**Keywords:** Magnesium sulphate, lignocaine, laryngoscopy and endotracheal intubation

## Introduction

Laryngoscopy and Endotracheal intubation are the frequently performed procedures in the practice of anaesthesia. These are the most stressful conditions to which the patient is subjected.

The standard technique of laryngoscopy and endotracheal intubation involves the stimulation of Larynx, Pharynx, Epipharynx and trachea, which are extensively innervated by Autonomic nervous system, namely the parasympathetic innervation via vagus and glossopharyngeal nerves and sympathetic via superior cervical ganglion.

Direct laryngoscopy and tracheal intubation following induction of anaesthesia is almost always associated with hemodynamic changes due to reflex sympathetic discharge caused by

epipharyngeal and laryngopharyngeal stimulation [1].

The circulatory response to laryngoscopy and intubation include [2, 3]

1. Increase in Heart rate.
2. Increase in Arterial pressure.
3. Increase in intracranial tension.
4. Increase in intraocular pressure.
5. Cardiac dysrhythmias.
6. Cardiac asystole.
7. Coronary and cerebral infarction and haemorrhage.

These adverse responses which occur in a normal sequence of induction and intubation and are further aggravated by

1. Light planes of Anaesthesia.
2. Hypoxia.
3. Hypercarbia.
4. Anxiety.
5. Reflex baroreceptor effect following induction agents like Thiopentone Sodium.

Hypertensive patients are more prone to have significant increases in blood pressure whether they have been treated before or not [4]. The haemodynamic changes are not serious enough in a normal individual but it may be hazardous to the patients with compromised circulatory system and cerebrovascular disorders [5, 6, 7].

Reid and Brace in 1940 were first to report the circulatory responses to laryngeal and tracheal stimulation in anaesthetized man [8].

## Methodology

A total number of 70 patients, 35 in each group with inclusion and exclusion criteria were selected for study, patients were allocated randomly to each group by lottery method.

## Inclusion criteria

1. ASA grade I and II physical status.
2. Patient requiring general anaesthesia and not requiring elective ventilation.
3. Age between 15-50yrs, belonging to both sexes.
4. Cormack Lehane grade I and II.
5. Laryngoscopy and intubation time <15 second.
6. Single attempt at intubation.
7. Patients willing for study.

## Exclusion criteria

1. Patients with ASA Grade  $\geq$ III.
2. Patient allergic to Magnesium sulphate or Lignocaine.
3. Patients age <15 or > 50
4. Cormack Lehane III and IV.
5. Laryngoscopy and intubation lasting for >15 seconds.
6. More than one attempt at intubation.
7. Patients with suspected airway anomalies or anticipated difficult airway.
8. Patient not willing for study.

## Methodology

All patients were explained in detail about the study and informed consent was taken. Patients were randomly allocated to receive intravenous Magnesium sulphate infusion 30 mg/kg, 15 minutes before induction of anaesthesia or injection Lignocaine 1.5mg/kg intravenously 90 seconds before intubation. All patients had an peripheral intravenous line secured in the pre-operative holding area. Randomization was done by picking lots. The Anaesthesiologist who prepared and administered the drug was not involved with the intra and post-operative management of the patient.

Monitors connected to patient include electrocardiogram, pulse oximeter and non-invasive blood pressure. Baseline systolic blood pressure, diastolic blood pressure, mean arterial blood pressure and heart rate were recorded. After randomization, patients of Group M, received injection magnesium sulphate 30mg/kg diluted to 50 ml in normal saline and given as an infusion over 15 minutes and then 10ml normal saline 90 seconds before intubation. Group L received infusion of plain normal saline in a 50ml syringe over 15 minutes and 1.5mg/kg preservative free I.V Lignocaine diluted to 10ml with normal saline 90 seconds before intubation.

Intraoperative monitoring included electrocardiogram, non-invasive blood pressure and pulse oximetry. Patient were premedicated with inj. Glycopyrrolate 0.005mg/kg, Inj. Fentanyl 1mcg/kg, induced with Inj. Thiopentone 5mg/kg, they were intubated with an appropriate size oral cuffed endotracheal tube after giving Inj. Vecuronium 0.1mg/kg. Anaesthesia was maintained intra-operatively with Oxygen, Nitrous Oxide and Sevoflurane at 33%, 66% and 1.5-2% respectively. Neuromuscular blockade was maintained with incremental doses of Vecuronium.

The parameters recorded and analysed for the study are heart rate, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure and spo2.

## Results

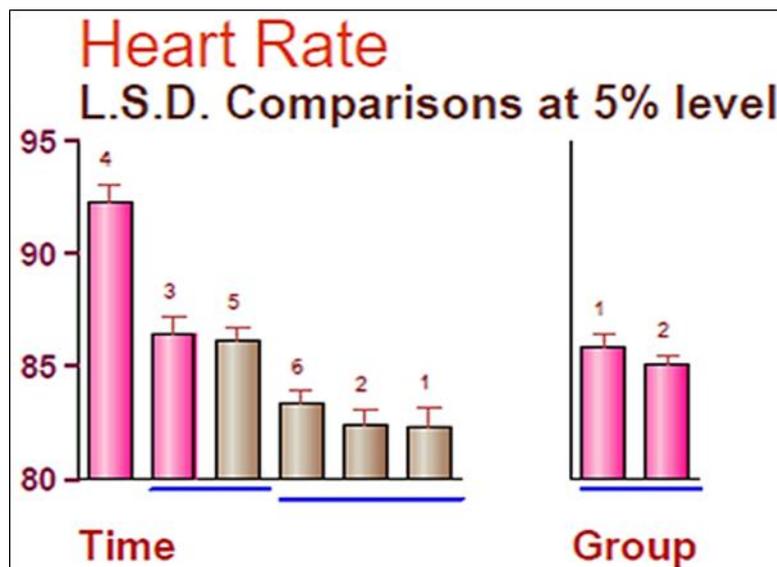


Fig 1: Heart Rate

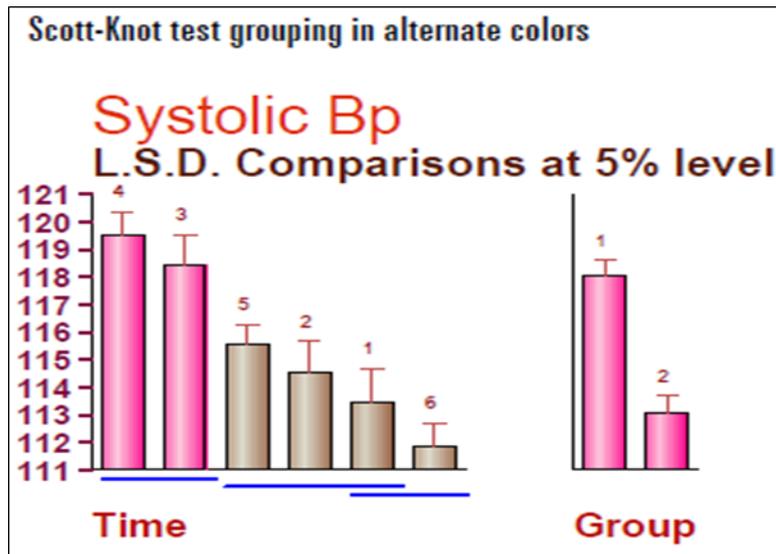


Fig 2: Systolic BP

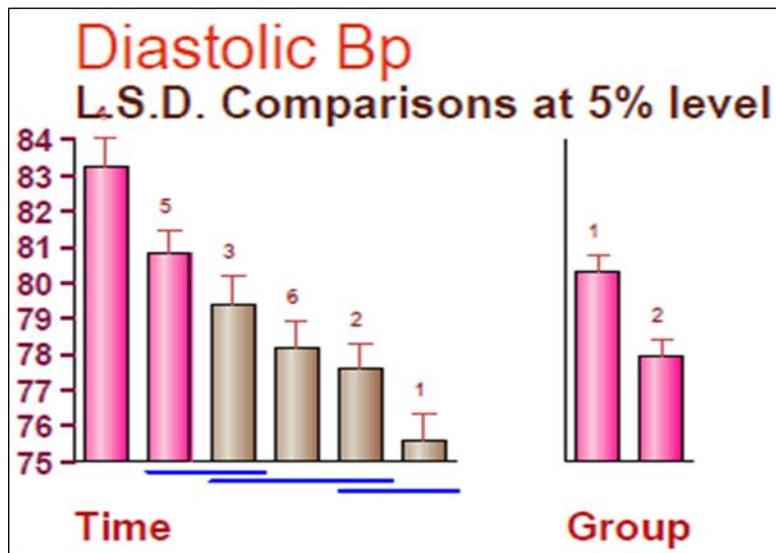


Fig 3: Diastolic BP

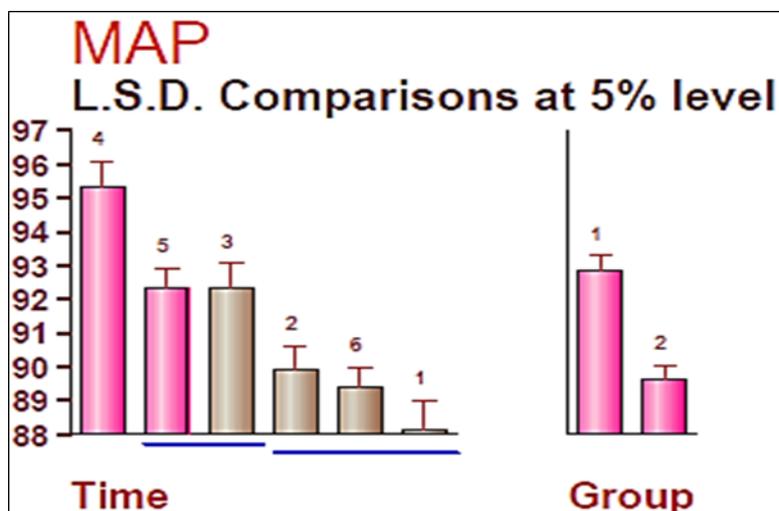


Fig 4: Map

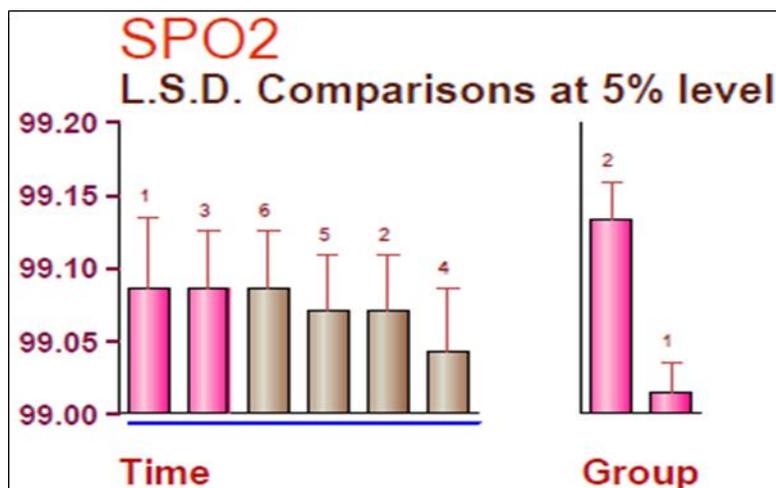


Fig 5: SPo2

## Discussion

General anaesthesia has almost become synonymous with endotracheal anaesthesia. Laryngoscopy and endotracheal intubation are considered as the most critical events during general anaesthesia. They provoke a transient, but marked sympathetic and sympathoadrenal response. In our study we compared intravenous Lignocaine, a drug which has been successfully used to blunt the hemodynamic response to intubation with Magnesium sulphate [9].

All the baseline values Systolic blood pressure, Diastolic blood pressure, mean arterial pressure and Heart rate and spo2 are comparable between the two groups. There is statistically no significant difference in the baseline values between the two groups.

In Group M (Magnesium sulphate group) the systolic, diastolic, mean arterial pressure, heart rate and spo2, compare to Lignocaine group less increased from baseline at immediately and first minute and then decreased at third, and fifth minute post intubation. The change in systolic, diastolic, mean arterial pressure, heart rate and spo2 from base line in group M is statistically significant 38-39.

Coming to Group L (Lignocaine group), the systolic, diastolic, mean arterial pressure, heart rate and spo2 increased from baseline at immediately and first minute and then decreased at third, and fifth minute post intubation. This increase in systolic, diastolic, mean arterial pressure and heart rate at the 0 and first minute was statistically significant. The decrease in systolic, diastolic, mean arterial pressure, heart rate and spo2 at third, and fifth minute was statistically significant. These findings correlated with the findings of Anila d malde *et al.*

Thus, in group L, the systolic, diastolic, mean arterial pressure and heart rate and spo2 increased from the baseline value at first and second minute after intubation but it is not significant. Anila d malde *et al.* compared the efficacy of Fentanyl versus Lignocaine in attenuating the pressure response to intubation. They have used glycopyrrolate 0.2 mg as premedication prior to surgery. They had used halothane as the maintenance agent. They had similar observation in the lignocaine group.

Bruder *et al.* in a review article wrote that in clinical practice, lignocaine is particularly effective in preventing the pressure response to tracheal intubation, whatever is the route of administration, but not the increase in heart rate which is similar to our observation [10].

Wilson *et al.* showed that irrespective of timing of administration of injection of lignocaine at second, third or fourth minute before tracheal intubation, there was a significant increase in heart rate in all the groups.

When comparing the hemodynamic parameters between the Magnesium sulphate Group (Group M) and Lignocaine Group (Group L), systolic, diastolic, mean arterial pressure, heart

rate and spo<sub>2</sub> decreased from the baseline value after intubation in magnesium sulphate group, whereas in the Lignocaine group there was an increase in the 0 and first minute, it was a significant change and decreased at third and fifth minute after intubation. All the hemodynamic parameters showed significant difference when comparing between the two groups. There are no significant side effects like severe haemodynamic variability like hypotension and bradycardia.

## Conclusion

We conclude that Magnesium sulphate and Lignocaine are effective in blunting the hemodynamic response to intubation, but Magnesium sulphate is superior to Lignocaine in blunting the hemodynamic response to laryngoscopy and endotracheal intubation without any significant side effects.

## References

1. Reema Gel, Raka Rani, Singh OP, Deepak Malviya, Arya SK. Attenuation of cardiovascular response to laryngoscopy and intubation by various drugs in normotensive patients, Hospital today, 2000, 9.
2. Robert K, Stoelting MD. Blood pressure and heart rate changes during short duration laryngoscopy for tracheal intubation. Influence of viscous or intravenous lidocaine. Anaesthesia analgesia. 1978;57:197-199.
3. Prys Roberts, Greene Lt, Meloche R, Foex P. Studies of anaesthesia in relation to hypertension-ii. Haemodynamic consequences of induction and endotracheal intubation. British journal of anaesthesia. 1971;43:541-547.
4. Yoshitaka Fuji MD, Yuji Saitoh MD, Shinji Takahashi MD, Hidenori Toyooka MD, Hidenori Toyooka MD. Diltiazem-Lidocaine combination for the attenuation of cardiovascular responses to tracheal intubation in hypertensive patients. Canadian journal of anaesthesia. 1998;45:935-937.
5. Elizabeth J Fox, Garry S Sklar, Constance H Hill, Raymond Villanueva, Benton D King. Complication related to the pressor response to endotracheal intubation, Anaesthesiology. 1977;47:524-525.
6. Dalton B, Guiney T, *et al.* Myocardial ischaemia from tachycardia and hypertension in coronary heart disease patients undergoing anaesthesia. Ann. Mtg. American society of anesthesiologist, Boston, 1972, 201-202.
7. Donegan MF, Bedford RF. Intravenously administered lignocaine prevents intracranial hypertension during endotracheal suctioning. 1980;52:516-518.
8. Reid LC, Brace De. Intubation of the respiratory tract and its reflex upon heart. Surgery, gynaecology, obstetrics. 1970;11:157-162.
9. Stanley Tam, Frances Chung, Michael Campbell. Intravenous lignocaine. Optimal time for injection before tracheal intubation. Anaesthesia, analgesia. 1987;66:1036-1038.
10. Dr. Manorama Singh. Stress response and anaesthesia altering the peri and post-operative management. Indian journal of anaesthesia. 2003;37:427-429.

Accepted on 18/05/2022