COMPARATIVE STUDY BETWEEN INTRAVENOUS 50% MAGNESIUM SULPHATE AND DEXMEDETOMIDINE FOR ATTENUATION OF CARDIOVASCULAR STRESS RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

Dissertation submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the regulations for

The award of the degree of

ANAESTHESIOLOGY

M.D. BRANCH - X

THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004.

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY

CHENNAI - 600 032.

OCTOBER -2016

CERTIFICATE

This is to certify that this dissertation entitled **"COMPARATIVE STUDY BETWEEN INTRAVENOUS 50% MAGNESIUM SULPHATE AND DEXMEDETOMIDINE FOR ATTENUATION OF CARDIOVASCULAR STRESS RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION"** is a bonafide original work of **Dr.U.GEETHA SOUNDARYA** in partial fulfillment of the requirements for M.D Branch -X (Anaesthesiology) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in OCTOBER - 2016. The period of study was from JUNE 2014 - JULY 2015.

PROF.DR.S.ILANGOVAN M.D.,
THE DEAN,
THANJAVUR MEDICAL COLLEGE AND HOSPITAL
THANJAVUR 613004

PROF. DR. R.MUTHUKUMARAN M.D., DA.,
HEAD OF THE DEPARTMENT
DEPT. OF ANAESTHESIOLOGY
THANJAVUR MEDICAL COLLEGE
THANJAVUR 613004

DECLARATION

I, **Dr. U.GEETHA SOUNDARYA**, solemnly declare that dissertation titled entitled **"COMPARATIVE STUDY BETWEEN INTRAVENOUS 50% MAGNESIUM SULPHATE AND DEXMEDETOMIDINE FOR ATTENUATION OF CARDIOVASCULAR STRESS RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION"** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during June 2014 to July 2015 under the guidance and supervision of **PROF Dr.R.MUTHUKUMARAN M.D,DA.,** Department of Anaesthesiology, Thanjavur Medical College, and Thanjavur.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University towards partial fulfillment of requirement for the award of **M.D Degree (Branch -X) in Anaesthesiology.**

Place: Thanjavur

Date: (**DR. U.GEETHA SOUNDARYA)**

ACKNOWLEDGEMENT

I gratefully acknowledge and express my sincere thanks to **Prof.Dr.S.IlangovanMD.,** Dean, Thanjavur Medical College and hospital, Thanjavur for allowing me to do this dissertation and utilizing the Institutional facilities.

I am extremely grateful to **Prof Dr.R.Muthukumaran M.D.,** my guide and Professor and Head of the Department of Anaesthesiology, Thanjavur Medical College and hospital, Thanjavur, for his full-fledged support, valuable suggestions and guidance during my study and my post graduate period.

I am greatly indebted to **Prof Dr. ShanthiPaulraj M.D.,**for her valuable suggestions, constant encouragement and scholarly guidance in my study and post graduate period.

I express my gratitude to **Prof.Dr.S.Uthirapathy.M.D.,D.A.,** for his guidance, valuable suggestions and constant encouragementwhich enabled me to do this work effectively

I express my gratitude to my respected Co-Guide, Assistant Professor **Dr.B.MOHAMMED SAMEER M.D.,** for his scholarly guidance and valuable time he has rendered to do this work effectively.

I would also like to extend my warmest gratitude toall my Assistant Professors for their constant encouragement and support.

I express my gratitude tothe faculty of Department of Surgery and E.N.T for their co-operation and support in conducting this study.

I would like to thank all my colleagues and friends who have been a constant source of encouragement to me.

Special thanks to all the volunteers and who whole heartedly co-operated and participated in this study.

Last but not the least; I would like to express my most sincere gratitude to my beloved husband and family for their help and constant support for this thesis

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA - 613001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

INSTITUTIONAL ETHICAL COMMITTEE

CERTIFICATE

Approval No. : 137

This is to certify that The Research Proposal / Project titled

COMPARISON BETWEEN IV 50% MAGNESIUM SULPHATE AND DEXMEDITOMIDINE FOR ATTENUATION OF CARDIOVASCULAR STRESS RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION.

submitted by Dr. U. GEETHA SOUNDARYA **of**

Dept. of ANAESTHESIOLOGY **Thanjavur Medical College, Thanjavur**

was approved by the Ethical Committee.

Thanjavur

Dated : 10.04.2015

THANJAVUR MEDICAL COLLEGE M.E UNIT THANJAVUR-4

Secretary
Ethical Committee
TMC, Thanjavur.

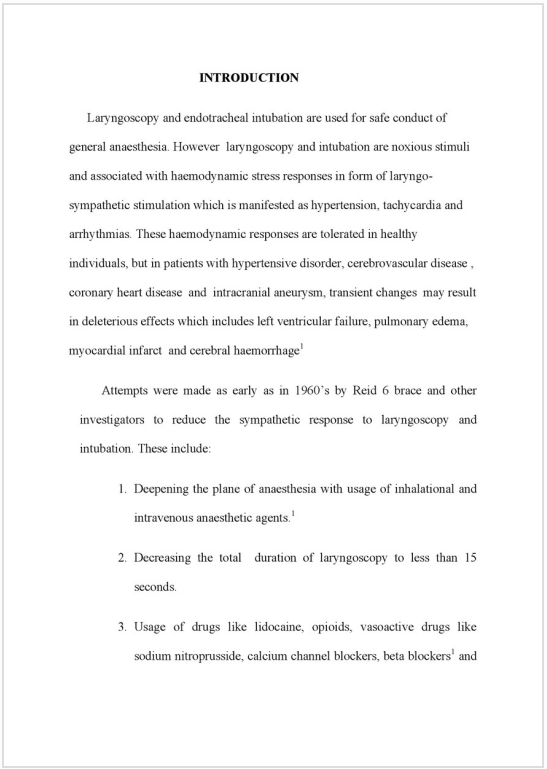
THE SECRETARY
INSTITUTIONAL ETHICAL COMMITTEE
THANJAVUR MEDICAL COLLEGE,
THANJAVUR.

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201320202.md Anaesthesiology Dr…
Assignment title:	2015-2015 plagiarism
Submission title:	comrarison between iv magnesium…
File name:	plagiarism_1.docx
File size:	317.63K
Page count:	77
Word count:	8,151
Character count:	46,644
Submission date:	04-Jul-2016 07:02PM
Submission ID:	687515599

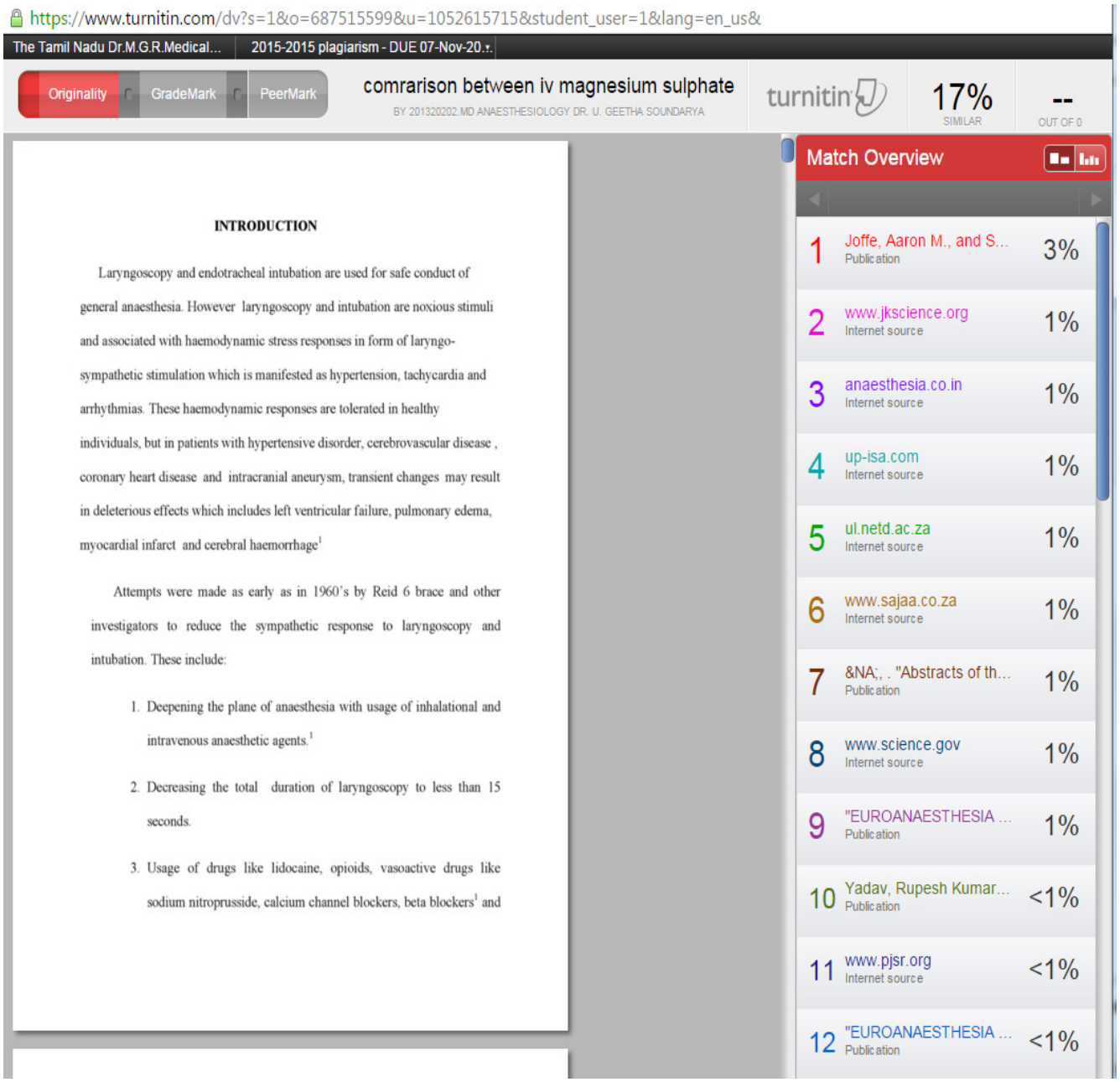


INTRODUCTION

Laryngoscopy and endotracheal intubation are used for safe conduct of general anaesthesia. However laryngoscopy and intubation are noxious stimuli and associated with haemodynamic stress responses in form of laryngo-sympathetic stimulation which is manifested as hypertension, tachycardia and arrhythmias. These haemodynamic responses are tolerated in healthy individuals, but in patients with hypertensive disorder, cerebrovascular disease , coronary heart disease and intracranial aneurysm, transient changes may result in deleterious effects which includes left ventricular failure, pulmonary edema, myocardial infarct and cerebral haemorrhage[1]

Attempts were made as early as in 1960's by Reid 6 brace and other investigators to reduce the sympathetic response to laryngoscopy and intubation. These include:

1. Deepening the plane of anaesthesia with usage of inhalational and intravenous anaesthetic agents.[1]
2. Decreasing the total duration of laryngoscopy to less than 15 seconds.
3. Usage of drugs like lidocaine, opioids, vasoactive drugs like sodium nitroprusside, calcium channel blockers, beta blockers[1] and


https://www.turnitin.com/dv?s=1&o=687515599&u=1052615715&student_user=1&lang=en_us&
The Tamil Nadu Dr.M.G.R.Medical...
2015-2015 plagiarism - DUE 07-Nov-20...
Originality
GradeMark
PeerMark
comrarison between iv magnesium sulphate
BY 201320202.MD ANAESTHESIOLOGY DR. U. GEETHA SOUNDARYA
turnitin
17%
SIMILAR
--
OUT OF 0
INTRODUCTION
Laryngoscopy and endotracheal intubation are used for safe conduct of general anaesthesia. However laryngoscopy and intubation are noxious stimuli and associated with haemodynamic stress responses in form of laryngo-sympathetic stimulation which is manifested as hypertension, tachycardia and arrhythmias. These haemodynamic responses are tolerated in healthy individuals, but in patients with hypertensive disorder, cerebrovascular disease , coronary heart disease and intracranial aneurysm, transient changes may result in deleterious effects which includes left ventricular failure, pulmonary edema, myocardial infarct and cerebral haemorrhage[1]
Attempts were made as early as in 1960's by Reid 6 brace and other investigators to reduce the sympathetic response to laryngoscopy and intubation. These include:
1. Deepening the plane of anaesthesia with usage of inhalational and intravenous anaesthetic agents.[1]
2. Decreasing the total duration of laryngoscopy to less than 15 seconds.
3. Usage of drugs like lidocaine, opioids, vasoactive drugs like sodium nitroprusside, calcium channel blockers, beta blockers[1] and
Match Overview
1 Joffe, Aaron M., and S... Publication 3%
2 www.jkscience.org Internet source 1%
3 anaesthesia.co.in Internet source 1%
4 up-isa.com Internet source 1%
5 ul.netd.ac.za Internet source 1%
6 www.sajaa.co.za Internet source 1%
7 &NA;, . "Abstracts of th... Publication 1%
8 www.science.gov Internet source 1%
9 "EUROANAESTHESIA ... Publication 1%
10 Yadav, Rupesh Kumar... Publication <1%
11 www.pjsr.org Internet source <1%
12 "EUROANAESTHESIA ... Publication <1%

CONTENT

INTRODUCTION

Laryngoscopy and endotracheal intubation are used for safe conduct of general anaesthesia. However laryngoscopy and intubation are noxious stimuli and associated with haemodynamic stress responses in form of laryngo-sympathetic stimulation which is manifested as hypertension, tachycardia and arrhythmias. These haemodynamic responses are tolerated in healthy individuals, but in patients with hypertensive disorder, cerebrovascular disease , coronary heart disease and intracranial aneurysm transient changes may result in deleterious effects which includes left ventricular failure, pulmonary edema, myocardial infarct and cerebral haemorrhage[1]

Attempts were made as early as in 1960's by Reid[6] brace and other investigators to reduce the sympathetic response to laryngoscopy and intubation. These include:

1. Deepening the plane of anaesthesia with usage of inhalational and intravenous anaesthetic agents.[1]

2. Decreasing the total duration of laryngoscopy to less than 15 seconds.

3. Usage of drugs like lidocaine, opioids, vasoactive drugs like sodium nitroprusside, calcium channel blockers, beta blockers[1] and other drugs mainly alpha 2 agonists like clonidine and Dexmedetomidine.[2]

Intravenous Dexmedetomidine, a central alpha 2 agonist is being used in anaesthesia as a premedicant. The advantages of Dexmedetomidine as premedicant in anaesthesia include sedation, analgesia, anxiolysis and improved hemodynamic stability. Because of these beneficial properties it has been found that minimum alveolar concentration (MAC) of volatile anaesthetic agent also decreases significantly up to 90% and hence decreases the requirement of anaesthetics.[3]It has been found that it decrease the haemodynamic response to laryngoscopy and intubation. [4,5]

Dexmedetomidine is being used in various other countries since many years as premedicant. Since it has been introduced in India in last decade (only in 2009) and not many studies have been done in our country, there is a need to study the role of Dexmedetomidine in obtunding the hemodynamic pressor response to laryngoscopy and intubation.

Magnesium sulphate blocks release of catecholamines from adrenergic nerve terminals. Increased Magnesium levels can also inhibit the relese of catecholamines.

Magnesium also causes vasodilation by acting directly on blood vessels and in high doses, it attenuates vasopressin-mediated vasoconstriction.

The present study was undertaken to compare the effectiveness of intravenous Magnesium sulphate and Dexmedetomidine for attenuation of stress response to laryngoscopy and endotracheal intubation.

AIM OF THE STUDY

The aim of this study was to compare between intravenous 50% Magnesium Sulphate and Dexmedetomidine for attenuation of cardiovascular stress response during laryngoscopy and tracheal intubation with respect to

1. Heart rate
2. Systolic blood pressure
3. Diastolic blood pressure
4. Mean arterial pressure
5. Level of sedation on recovery
6. Adverse effects

PATHOPHYSIOLOGY OF LARYNGOSCOPY ANDINTUBATION

I.Cardiovascular Responses during Airway Manipulation:

A. Cardiovascular Reflexes:

The cardiovascular responses to noxious airway manipulation are initiated by proprioceptors responding to tissue irritation which is located in the supraglottic region and in the trachea.[7]Located in close proximity to the airway mucosa, proprioceptors consist of mechanoreceptors of small-diameter myelinated fibers, slowly-adapting stretch receptors with large-diameter myelinated fibers, and polynodal endings with nonmyelinated nerve fibers.[8](The superficial location of proprioceptors and their nerves explains why topical local anesthesia of the airway is an effective means of blunting cardiovascular responses to airway interventions.)

The glossopharyngeal and vagal afferent nerves transmit these impulses to brainstem, which in turn, causes widespread autonomic activation by means of sympathetic and parasympathetic nervous systems.

Bradycardia, often elicited in infants and small children during laryngoscopy or intubation is autonomic equivalent of the laryngospasm response. Although seen rarely in adults, this reflex are due to increase in vagal tone at the sinoatrial node

In adults and adolescents, more common response to airway manipulation are hypertension and tachycardia mediated by the cardioaccelerator nerve fibres and sympathetic chain ganglia. This response includes enormous release of noradrenaline from adrenergic nerve terminals and secretion of epinephrine from the adrenal medulla. [8]Some of hypertensive stress response to laryngoscopy and endotracheal intubation also results from activation of the renin-angiotensin system, which includes release of renin from the renal juxtaglomerular apparatus, which is innervated by β-adrenergic nerve terminals.

In addition to activation of autonomic nervous system, laryngoscopy and endotracheal intubation can result in stimulation of the central nervous system, as seen by increases in electroencephalographic activity, cerebral metabolic rate, and cerebral blood flow . In patients with compromised intracranial compliance, increase in Cerebral blood flow may result in increased intracranial pressure which, in turn, may cause herniation of brain contents and severe neurologic compromise.

The effects of endotracheal intubation on the pulmonary vasculature are often accompanied by changes in airway reactivity associated with intubation. Acute bronchospasm or a main stem bronchial intubation results in a marked maldistribution of perfusion to poorly ventilated lung,

ultimately causing desaturation of pulmonary venous blood and subsequent reduction in systemic arterial oxygen tension.

In addition, giving positive end-expiratory pressure after endotracheal intubation causes reduction in cardiac output which is due to impaired venous return to the left side of the heart from the pulmonary circulation. The impact of these changes can be profound in case of patients with severely compromised myocardial function or intravascular volume depletion.

B. Intubation in the Presence of Cardiovascular Disease:

Myocardial ischemia results when there is imbalance between myocardial oxygen supply and demand. The chief components of myocardial oxygendemand were beat frequency or heart rate and myocardial wall tension. Of the two, increases in heart rate are of more important , because cardiac ionotropism (contractility) subserves cardiac chronotropism (rate). Tachycardia increases myocardial oxygen consumption per minute at constant wall tension, but also the rise in heart rate effectively reduce the diastolic period.

Neuroendocrine responses to airway manipulation results in tachycardia and hypertension which may result in a variety of complications in patients with cardiac disease, myocardial ischemia

mainly. Episodes of ischemic electrocardiographic ST-segment depression and increased pulmonary artery diastolic blood pressure may be seen in patients with arteriosclerosis when intubation is done .

Patients with aneurysmal disease of the cerebral and aortic circulation were in risk of complications related to a sudden increase in Blood pressure during airway manipulation . Leaking aortic aneurysms are partially tamponaded by intra-abdominal pressure but it can suddenly expand into the retroperitoneal space during arterial hypertension

The results are significant blood loss , hypotension and technical problems for surgeon trying to resect the lesion and insert a vascular prosthesis.

C. Implications for Patients with Neurovascular Disease:

Intracranial aneurysms and arteriovenous malformations are likely to rebleed during elevated arterial blood pressure which may result in sudden and permanent neurologic injury, mostly when blood pressure and the heart rate were increased in response to endotracheal intubation.

D.Intubation in Patients with Neuropathologic Disorders:

In patients with impaired cerebral autoregulation (e.g., brain trauma, cerebrovascular accidents, neoplasms), the tendency for cerebral blood flow to remain constant over mean blood pressure range of 50 to

150 mm Hg is impaired. When endotracheal intubation causeing increase in blood pressure , there is a marked increase in cerebral blood flow and cerebral blood volume, which in turn cause dangerous increases in intracranial pressure . The effect is magnified by the fact that noxious stimuli, such as airway manipulation may result in increased cerebral blood flow , which summates with hypertensive response, causing profound increases in intracranial pressure .

E. Cardiopulmonary Consequences of Positive-Pressure Ventilation

Increase in mean intrathoracic pressure due to positive-pressure ventilation may be transmitted to the compressible superior and inferior venae cavae, effectively increasing the downstream pressure for venous return and reducing venous return to the right atrium thus result in decreased cardiac output and subsequently blood pressure may fall with positive-pressure ventilation . Patients with decreased intravascular volume have an exaggerated hypotensive response as a result of this phenomenon.

II. Prevention of Cardiovascular Responses:

A. Minimizing Stimulation of Airway Proprioceptors:

Laryngoscopy itself is a stimulating procedure, and use of a straight blade (Miller blade) with elevation of vagally innervated posterior

epiglottis results in significantly higher blood pressure than does use of a curved blade (Macintosh or Corazzelli–London–McCoy [CLM]).

Newer video and optical laryngoscopes, which does not require alignment of laryngeal axes have potential to minimize the pressor response to airway manipulation by reducing the amount of force needed to displace oropharyngeal tissues and also by limiting cervical spine motion compared to traditional laryngoscopy with a Macintosh laryngoscope blade.

Insertion of a conventional laryngeal mask airway after induction of general anesthesia with use of thiopental or propofol and fentanyl has shown to cause less cardiovascular and endocrine response than laryngoscopy or endotracheal intubation.[10] The laryngeal mask airway has advantage of avoiding vagally mediated infraglottic stimulation entailed by the use of laryngoscope, thus enabling lighter levels of general anesthesia.

In contrast, endotracheal intubation using the intubating laryngeal mask airway resulted in a hemodynamic and endocrine response similar to that of direct laryngoscopy and intubation

B. Topical and Regional Anesthesia:

Topical anesthesia applied to the upper airway is very much effective in blunting hemodynamic stress responses to endotracheal intubation,[17,18] but it has almost less effective than systemic administration of lidocaine.

If topical lidocaine is administered to the upper airway, there should be an gap of at least 2 minutes to allow initiation of anesthetic effect before airway instrumentation begins.

During general anesthesia, rigid laryngoscopy and instillation of lidocaine solution initiate the same adverse reflexes caused by placement of an endotracheal tube. Furthermore, a laryngotracheal spray of lidocaine solution may, in itself, produce profound cardiovascular stimulation in adults, and in children it may produce the same sort of bradycardic response associated with endotracheal intubation.

In contrast to topical anesthesia of airway, which does not provie desirable effect , regional nerve blocks involving the sensory pathways from airway prevent hemodynamic responses to intubation.

The superior laryngeal nerve innervates the superior surface of the larynx, and the glossopharyngeal nerve innervates the oropharynx. Depositing local anesthetic mixture on each cornu of the hyoid bone can

block the superior laryngeal nerve. Blockade of the glossopharyngeal nerve at the tonsillar pillars (sensory distribution above the level of the epiglottis) potentiates the effect by decreasing the stimulus of laryngoscopy.[11]

The inferior surfaces of the larynx and trachea also require topical anesthesia, but they are innervated by the recurrent laryngeal nerve and the vagus, which cannot be directly blocked.

Instillation of lidocaine via anendotracheal tubedone to prevent alterations in cerebrovascular hemodynamics in patients with severe head injury may result in some benefit. A dose of 1.7 mg/kg lidocaine given slowly (1 mL/sec) through a fine tube advanced to the end of the endotracheal tube was reported to be efficacious in half of the patients treated.[23]

C Inhalational Anaesthetics:

Agents that are capable of preventing intubation stress responses may also cause profound cardiovascular depression before and after endotracheal intubation .

Dose of volatile anesthetic required to block the cardiovascular response to endotracheal intubation must be high, resulting in profound cardiovascular depression before endotracheal intubation.

High doses of volatile anesthetic agent may cause cerebral vasodilation and marked increases in intracranial pressure in patients with compromised intracranial compliance. Arterial hypotension and reduced cerebral perfusion pressure before intubation were unacceptable for patients with cerebrovascular disease and brain injury.

D. Intravenous Agents:

Propofol, barbiturates, and benzodiazepines are all associated with profound hypotension at doses which is used to suppress the hemodynamic and intracranial pressure responses to intubation.[24,25,26]

In the case of etomidate, the effective dose for blocking the cardiovascular response to intubation can identified by burst-suppression pattern on cortical surface electroencephalography, indicating fairly deep cerebral depression.[27] Because etomidate maintains blood pressure at deep levels of anesthesia, it is probably the only agent thatcan achieve suppression of cardiovascular responses without producing arterial hypotension and compromised coronary and cerebral perfusion.

Because it is clinically impractical to achieve anesthetic depth for preventing a response to intubation with single intravenous or inhalational agent (etomidate excepted), a wide variety of anesthetic drug combinations, adjuvants, or both has been used to potentiate anesthetic effects while minimizing hemodynamic depression.

Opioids are adjuvants most commonly used in addition to other Intravenous or inhaled agents to facilitate the induction of anesthesia and subsequent airway manipulation. Opioids with shorter onset and offset times have advantages over fentanyl for modulating circulatory responses to intubation.

Intravenous lidocaine also blunt hemodynamic and cerebrovascular responses to intubation. When given in a bolus dose of 1.5 mg/kg it adds approximately 0.3 MAC of anesthetic potency.[12] Significant reductions in hemodynamic stress response to endotracheal intubation noted when lidocaine (3 mg/kg) was used as an adjunct to high-dose fentanyl anesthesia.[12]

The general anesthetic properties of lidocaine are to reduce cerebral metabolic rate for oxygen and cerebral blood flow, thus decreasing intracranial pressure in patients with compromised intracranial compliance.

E Nonanesthetic Adjuvant Agents:

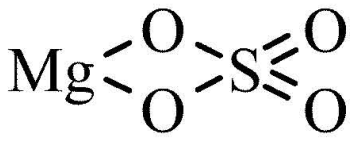
Prophylactic administration of vasoactive substances which directly affects cardiovascular system modifies the cardiovascular responses to endotracheal intubation .[13]

In 1960, DeVault and associates found that pretreatment with phentolamine 5 mg intravenously , prevents hypertensive-tachycardic response to endotracheal intubation during a thiopentone-succinylcholine induction[14]

Various vasodilators and adrenergic blocking agents were used as pretreatment before endotracheal intubation including diltiazem, verapamil, and nicardipine, hydralazine, nitroglycerin, nitroprusside, labetalol, esmolol, clonidine, Magnesium sulphate and Dexmedetomidine.

PHARMACOLOGY OF DRUGS

Pharmacology of Magnesium sulphate



Magnesium plays a major role in enzymatic reactions as cofactor and also in neurochemical transmission , muscular excitability.[16]

Large stores of Magnesium was seen intracellularly and also in bones of adults. Parenteral Magnesium therapy were used to repair plasma deficit and deficiency signs and symptoms cease to exist. Hypomagnesemia develop within 3 to 4 days with magnesium level <1.5meq/L.

They present as twitching , muscular irritability, and tremors. Hypocalcemia and hypokalemia often follows.

Mechanism of Action

1. A competitive antagonistic action seen on presynaptic calcium channels which regulates neurotransmitter release in the Central nervous system.[17]
2. Decrease in catecholamine release and calcium antagonistic action on smooth muscle cells responsible for anaesthetic effect.
3. **Neuromuscular blockade:** At neuromuscular junction it inhibits acetylcholine release. [18]
4. **Anticonvulsive effect**: Blockade of neuromuscular transmission and decreased acetylcholine secretion
5. **Antinociceptive effects**
6. **Central sensitization:** Inhibition of dorsal horn NMDA receptors causes attenuation of central sensitization.
7. **Central nervous system**: Depressive effect

Pharmacokinetics:

Normal range for Magnesium are 1.5 to 3.0 mEq/L.As plasma levels of Magnesium increase above 4mEq/L, Deep tendon reflexes are first decreased, later they disappear when magnesium increase to 10 mEq/L. Respiratory paralysis and heart block even occur.[19]

On intravenous route the anticonvulsive effect is immediate, by intramuscular route, the action starts by 1 hour and persists for a duration of 3 to 4 hours. Anticonvulsive effect occurs when magnesium levels are of 2.5 to 7.5 mEq/L. Magnesium excreted mainly by kidney.

Pharmacodynamics

Indications and Clinical Uses:

1. **Deficiency**: As replacement therapy
2. **Total parenteral nutrition :** to prevent hypomagnesemia.
3. **Anticonvulsant:** to prevent eclampsia
4. **Antihypertensive:** Magnesium sulfate effective in controlling hypertension and seizure especially children with acute nephritis.
5. **Laparoscopic surgeries:** Attenuates the stress response caused by increased intra abdominal pressure by blocking the catecholamine release[19,20,21,22]

Adverse Reactions:

1. **Magnesium intoxication**
2. **Hypocalcemia**
3. **Central nervous system:** Drowsiness and loss of muscle tone

Precautions:

Patient with renal impairment

Urine output should be monitored

Monitoring serum magnesium levels

Knee jerk and respiratory rate to be monitored

Symptoms and Treatment of Overdose:

Magnesium intoxication causes hypotension and respiratory paralysis.

Absent knee jerk sign of overdosage

On over dosage, artificial ventilation should be given until calcium gluconate started .

In extreme cases, peritoneal dialysis or hemodialysis should be performed.

Dosage and Administration:

Preeclampsia or eclampsia : 4-6g intravenous loading dose followed by 1-2 g continuous infusion or 4-5 g intramuscularly every 4 hr.

Torsades de pointes : 1-2 g

Asthma: 2g over 30-60 min

Severe deficiency :1-2 g IV followed by 0.5g/hrIV as needed

Clinical Uses:

Attenuation of Laparoscopic stress response

Antihypertensive

Eclampsia:

In case of severe pre-eclampsia or eclampsia dosage of magnesium is 10 to 14g

Nephritic Seizures:

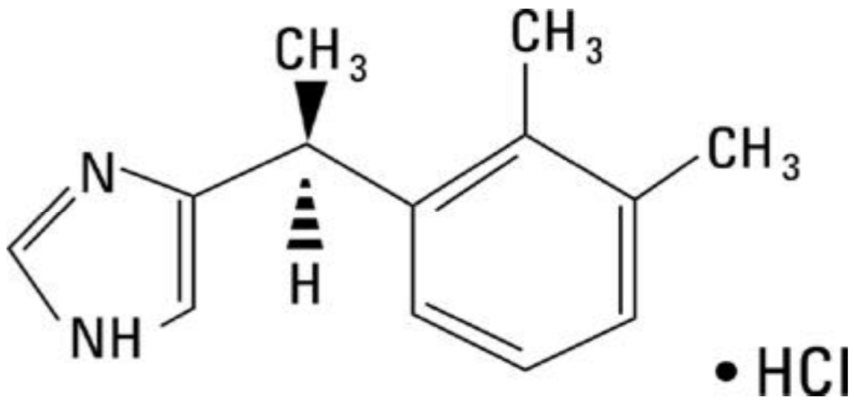
20-40mg/kg body weight given intramuscularly in children seizures due to nephritis.

PHARMACOLOGY OF DEXMEDETOMIDINE

Dexmedetomidine is dextro enatiomer of medetomidine,[22] methylsated derivative of etomidine. It is being used in various settings outside Intensive care unit, including anesthesia in adults and children in case of minimally invasive procedures, sedation for diagnostic procedures and other applications.

Chemical structure

4-S-[1-(2, 3 dimethylphenyl)ethyl] -1 H-imidazole monohydrochloride



Chemical structure of Dexmedetomidine

Physical properties

Molecular weight-236.7, pH:6-7, pKa:7.1

Mechanism of action

Stimulation of α_2 receptors in brain and spinal cord inhibit neurons which results in fall in blood pressure , heart rate , sedation and analgesia.

Sedative effect is due to hyper polarization of nonadrenergeric neurons in the locus ceruleus of brain stem. α_2 receptors inhibit adenylyl cyclase, which catalyses cAMP.[23]Dexmedetomidine by decreasing cAMP favors anabolic pathways . In spinal cord it decreases the transmission of pain by reducing the substance P and glutamate release by activating potassium channels.[24]

PHARMACOKINETICS

When injected intravenously, effect starts in 15 minutes. Metabolites are eliminated in urine mostly and in feces. Volume of distribution is 118 Litres, with distribution t1/2 is 6 min and elimination t1/2 is 2-2.5 hours. Clearance is estimated to be 39 Litres/hr.

Pharmacodynamics

Cardiovascular System

Dexmedetomedine causes dose dependent cardiovascular response. Due to stimulation of α_2b receptors first blood pressure rises and heart rate falls, after bolus dose of 1mcg/kg. Initial response lasts for 5-10minutes followed by fall in blood pressure due to inhibition of central sympathetic outflow.[26] Decrease in blood pressure and heart rate are due to decrease in noradrenaline release due to stimulation of α_2 receptors at presynaptic terminals. Decrease in heart rate is due to decrease in sympathetic tonepartly by baroreceptor reflex and also by enhanced vagal activity.

Central Nervous System

It produces sedation, hypnosis, anxiolysis, amnesia and analgesia as well as sedation and hypnosis.

Analgesia

Analgesic effect is due to its action at dorsal horn of spinal cord and at supraspinal site.

Respiratory System

Dexmedetomidinereduces minute ventilation though the response to increase in carbondioxide concentration is preserved. Apnea threshold is decreased.

Clinical applications

Dexmedetomidine is used as sedation in Intensive care unit patients, also as sole anesthetic agent in case of monitored anaesthesia care. It is also used as premedicant and also as an adjunct with local anaesthetic agent during peripheral nerve blocks, intravenous regional anesthesia and spinal anesthesia.[27]

Anaesthesia

When used as premedication it reduces the requirements of induction agents by 30%, Opioid and volatile anaesthetics by 25%. It is used as an adjuvant to general anesthesia. It suppresses the hemodynamic response when administered as premedicant at desired dose.[25] In neurosurgeries, where neurocognitive testing is necessary during surgical procedure, Dexmedetomidine seems to better option than general anesthesia. It is effective in reducing pulmonary hypertension in patients undergoing mitral valve replacement. In morbid obese patients who were

undergoing bariatric surgeries, Dexmedetomidine appears to attenuate postoperative pain relief and reduces further opioid requirement without causing respiratory depression. It has been used in treatment of symptoms of withdrawal of narcotics, benzodiazepines and alcohol.

Sedation in Intensive Care Units

Dexmedetomidine has been used as sedating agent in Intensive care unit settings. It allows sedated patients to be aroused without any discontinuation of drug prior to weaning. It reduces opioid consumption by 50% and significantly has high Pao2/Fio2 ratio. Also used in pediatric patients in procedural sedation.[24]

DOSAGE and ADMINISTRATION

For adults, it is administered intravenously at the loading dose of 0.5-1 mcg/kg slow infusion followed by 0.2-0.7 mcg/kg/hr as maintenance.

ADVERSE EFFECTS

Transisent hypertension, hypotension, nausea, bradycardia,AV block, atrial fibrillation, delirium .

REVIEW OF LITERATURE

K. Montazeri et al (2005) conducted a Study of Magnesium in suppressing stress responses to laryngoscopy and intubation . It is done in 100 ASA-1 patients of 15-50 years old in 5 groups (20 patients in each). The basal heart rate and blood pressure was noticed. According to groups, patients took Magnesium sulphate (10, 20, 30, 40, 50mg/kg). After intubation heart rate and Blood pressure recorded at 1, 3, 5 and 10 minutes. In conclusion, administration of different doses of Magnesium at the time of the induction of anaesthesia improves hemodynamic pressor responses to intubation, that the dose of 30mg/Kg of Magnesium was the most effective with the less unexpected effects

Xuexin Feng et al *(2006)*done a study to investigate that single dose of Dexmedetomidine on attenuating of stress response to intubation.60 ASA I and II patients of age 20-51 years scheduled for gynecologic surgery with general anaesthesia was divided into two groups. 28 patients of group D received Dexmedetomidine at dose of 0.6mcg/kg diluted with normal saline before induction. 25 patients of group C received the same amount of normal saline . Hemodynamics were recorded before and after induction. He concluded that single dose of Dexmedetomidine given before induction decreases the stress response .

Yildiz et al(2006) evaluated the effect of Dexmedetomidine 1 mcg/kg on stress responses during intubation. Here 50 patients were randomized into Dexmedetomidine and control group ramsay sedation score scale , vital parameter,adverse reaction noticed. Blood pressure and heart rate were decreased in Dexmedetomidine group than in control group.

Abbady A. Ahmed etal (2009) conducted to study the efficacy of preoperative administration of Magnesium in reducing intubation stress response.50 patients were randomly assigned to Magnesium and the control group. 50mg/kg magnesium was given to group M. The same volume of isotonic normal saline was administered to the control group, hemodynamic parameter was recorded. They conclude that the use of Magnesium sulphate attenuates the stress response to laryngoscopy and intubation.

R.saraf et al (2013) investigated that, Dexmedetomidine can be used as the ideal drug for attenuating the pressor response or not. 100 patients aged 14-55 years old were assigned. Group C got 10 ml normal saline,group D got Dexmedetomidine 0.6µg/kg diluted to 10 ml Normal saline. vital parameter were noted at various time interval after intubation. Dexmedetomidine at dose of 0.6µg/kg given intravenously 10 min before induction was effective in blunting intubation stress response.

Arpita Laha et al (2013) evaluated attenuation of sympathoadrenal response with Dexmedetomidine. Group 1recieved Dexmedetomidine at dose of 1 μg /kg and control group got normal saline. Vital parameter recorded after intubation. He concluded that preoperative administration of Dexmedetomidineat dose of 1mcg/kg results in sedation, and blunting stress response to intubation.

Gautham pillai et al (2013)studied with 80 patients undergoing spine surgery . Group M received Magnesium sulphate 30 mg/kg a bolus and 10 mg/k/hr1 intravenously by continuous infusion. Group C got normal saline. Hemodynamic parameters were recorded. They concluded that Perioperative iv Magnesium sulphate effective in attenuating intubation stress response when compared to lignocaine.

Dr. Rajdip Hazra et al (2014) conducted a randomized controlled trial regarding attenuation of hemodynamic pressor response to intubation by Dexmedetomidine in cardiac surgery. 90 patients of ASAII and III, of age18 to 45 years of both the sex scheduled for elective cardiac surgery (ASD and VSD closure) are included . Control group (group C) received 50 ml of normal saline. Dexmedetomidine group (group D) received Dexmedetomidine 0.5 μg/kg in normal saline. After induction of anaesthesia,hemodynamic parameter was noticed. In his study,

administration of Dexmedetomidine before commencement of anesthetic induction effectively bluntedstress response to intubation.

Krishna chaitanya et al (2014) done comparative Study between intravenous 50% Magnesium Sulphate and Dexmedetomidine. Group-M received 30mg/kg of Magnesium sulphate . Group-D patients received 1 mcg/kg Dexmedetomidine. Both the groups were observed for changes in hemodynamic parameters. Both Magnesium sulphate and Dexmedetomidine attenuated the rise in systolic diastolic, mean blood pressure, but Magnesium failed to attenuate increase in the heart rate.The study proves that Magnesium sulphate is as effective as Dexmedetomidine in attenuating the hemodynamic pressor response to laryngoscopy and endotracheal intubation.

Venugopal S et al (2015) studied the efficacy of Dexmedetomidine in attenuating hemodynamic response to intubation . From the study it is concluded that premedication with Dexmedetomidineat dose of 1mcg/kg attenuates the pressor response associated with laryngoscopy and tracheal intubation. The attenuation occurs within 5 minutes following laryngoscopy and endotracheal intubation and becomes maximum by 10 minutes. The haemodynamic parameters reach near normal values after 5 to 10 minutes of intubation.

Azin honarmand et al (2015) conducted study withdifferent doses of intravenous Magnesium sulphate .120 patient divided into 4 groups (Group I: 30 mg/kg, Group II: 40 mg/kg, Group III: 50 mg/kg) and control.

They concluded that use of Magnesium sulphateless than 50 mg/kg effective in reducing cardiovascular instability related to intubation.

Sridhar kalkeri et al(2015) investigated whether Dexmeditomidine decrease hemodynamic pressor response to intubation in patients undergoing CABG . It is a placebo controlled, double blinded, randomized study, The patients will be randomlyseparated into two Placebo and Dexmedetomidine group . Dexmedetomidine group received total dose of 1 mcg/kg diluted in 100ml normal saline given over 15 min. Placebo group will receive 100 ml normal saline over 15 min. Hemodynamic measurements repeated after administration of Dexmedetomidine and placebo at 1,3 and 5 minutes after intubation. Use of Dexmedetomidine preoperatively obtunded the haemodynamic stress during intubation significantly and this drug can be used regularly in Intensive care unit setting as alternative to other drugs because of its lower side effects.

Rashi kadam et al (2016) did a prospective, single blinded randomized controlled clinical investigation to assess the effectiveness of Magnesium

sulphate on haemodynamic change on intubation. Group A received equivalent amount of normal saline (over 5 min, 5 min prior to induction) Group B received 30 mg/kg Magnesium sulphate intravenously (over 5min, 5 min prior to induction). Hemodynamic parameter were recorded. They concluded that, Magnesium sulphate in single bolus dose can be used regularly to attenuate haemodynamic pressor response to intubation because of its properties like more efficacy and minimum side effects.

Arum kumar et al (2016) was carried out a study the effect of Dexmedetomidine on intubation. All the patients were premedicated with Fentanyl and Midazolam. The study group received Dexmedetomidine 1mcg/Kg in 100ml saline over 15 minutes and the group C received normal saline. Dexmedetomidine given at dose of 1 mcg/kg during intubation significantly blunted the heart rate, systolic blood pressure, mean arterial pressure and diastolic blood pressure in comparison to control group.

Nabin Pokhrel et al (2016)studied the effect of Dexmedetomidine on intubation. Dexmedetomidine when given at the dose of 1 mcg/kg blunts pressor response to intubation.

MATERIALS AND METHODS

This is a double blinded , prospective, randomized control study conducted in Thanjavur medical college hospital, Thanjavur during the period 2014-15. After obtaining institutional ethical committee approval and informed consent, 60 ASA I and II subjects in the age group of 20-60 years planned for elective non cardiac surgeries were enrolled in this study. They were randomly allocated to one of the two study groups. Group D (Dexmedetomidine group) and Group M (Magnesium sulphate group).

Statistical test of significance

Comparison of parameters was done using One-Way ANOVA and categorical data was compared by using Chi-square test. p Value <0.05 was considered as statistically significant.

INCLUSION CRITERIA includes

1.ASA grade I or II patient

2.Elective non cardiac surgery

3.Patients of either sex

4.Aged 20-60 years

EXCLUSION CRITERIA includes

1. Patient refusal
2. Patients <20years and >60 years of age
3. Heart rate<70/min
4. Systolic blood pressure <100 mmhg
5. Mallampatti Grading iii and iv
6. Anticipated difficult intubation
7. Total duration of laryngoscopy more than 30 seconds
8. ASA grade III or IV patients
9. Patients with systemic disorders like left ventricular failure, any degree of heart block, ischemic heart disease, aortic stenosis and bronchial asthma.

Preanaesthetic evaluation

All patients were kept on 6 hours starvation. Patients were premedicated with intravenous ranitidine 0.25 mg/kg, metoclopramide 0.15 mg/kg and glycopyrrolate 0.04 mg/kg intra muscularly in preoperative room 60 minutes before surgery.

In the Operating room

On arrival in the operation theater, monitors was connected (heart rate, NIBP, oxygen saturation, ECG) and baseline vital parameters like heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were recorded.

Group M patients were administered 30mg/kg of 50% Magnesium sulphate in 100 ml of normal saline over a period of 15 min, given 10 min before intubation

Group D patients received intravenous Dexmedetomidine 1mcg /Kg in 100 ml of normal saline over a period of 15 min, given 10 min before intubation.

Patients were induced with intravenous injection of thiopental 5mg/kg followed by intravenous succinylcholine 1.5mg/kg and inj. fentanyl 2mcg /kg to facilitate intubation. Total duration of laryngoscopy was noted. Patients whose total duration of laryngoscopy more than 30 seconds were excluded from the study. Heart rate, Systolic blood pressure , Diastolic blood pressure and Mean arterial pressure were noted at 0, 1, 3, 5 and 10 minutes after intubation. Anesthesia was maintained with O2, N2O, isoflurane and inj. vecuronium. At the end of the surgery patients were reversed with neostigmine 0.05mg/kg and glycopyrollate 0.08 mg/kg.

Assessment of parameters

The parameters like Heart rate , Systolic blood pressure, Diastolic blood pressure and Mean arterial Blood pressure were recorded at the following point of time .

1. Prior to induction (baseline value)
2. At the end of intubation (t0)
3. 1 min after intubation (t1)
4. 3 min after intubation (t3)
5. 5 min after intubation (t5)
6. 10 min after intubation (t10)

Patients were monitored for any adverse effects like bradycardia, and hypotension during intraoperative period

Adverse effects

Immediately after extubation, the patients were monitored for nausea, vomiting, shivering and level of sedation assessed by Modified Ramsay Sedation Score.

Hypotension was said to have occurred if Systolic blood pressure fell below 100 mm of Hg or if Diastolic blood pressure fell below50 mm of Hg or if the Mean arterial blood pressure fell below60 mm of Hg. Patient was treated with 100% O2, increasing the infusion rate of intravenous fluids and Inj. Ephedrine in incremental doses given at interval of 2 minutes.

Bradycardia was defined if heart rate was less than 60/min and was treated with intravenous atropine 0.6mg .

Modified Ramsay Sedation Score

score	Level of activity
0	Paralyzed , unable to evaluate
1	Awake
2	Lightly sedated
3	Moderately sedated, follows simple commands
4	Deeply sedated , responds to non-painful stimuli
5	Deeply sedated, responds only to painful stimuli
6	Deeply sedated, unresponsive to painful stimuli

OBSERVATIONS AND RESULTS

This is a prospective, double blinded, randomized, controlled study done in 60 ASA I and II patients of either sex aged between 20-60 years, posted for elective non cardiac surgeries under general anaesthesia. The study was undertaken to compare the efficacy of intravenously administered Dexmedetomidine and Magnesium sulfate to attenuate the cardiovascular stress response during laryngoscopy and endotracheal intubation.

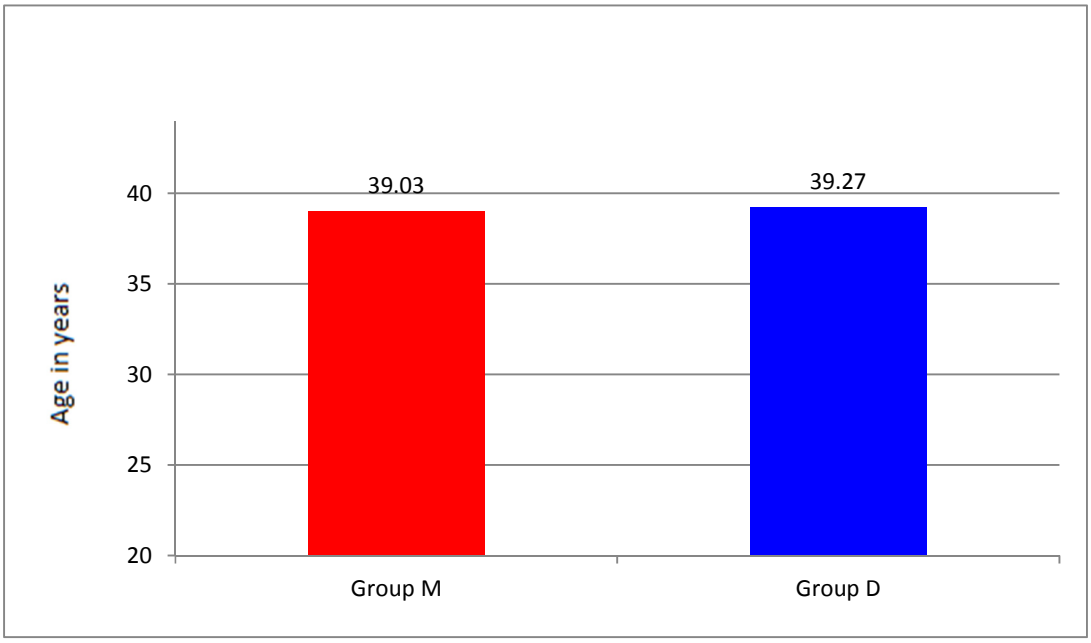
DEMOGRAPHIC CHARACTERISTICS OF STUDY POPULATION

AGE

Comparison of groups on basis of age

Study group	MEAN ± SD (Age in years)	p value
M	39.03 ± 3.86	p = 0.806 Not Significant
D	39.27 ± 3.45	

AGE



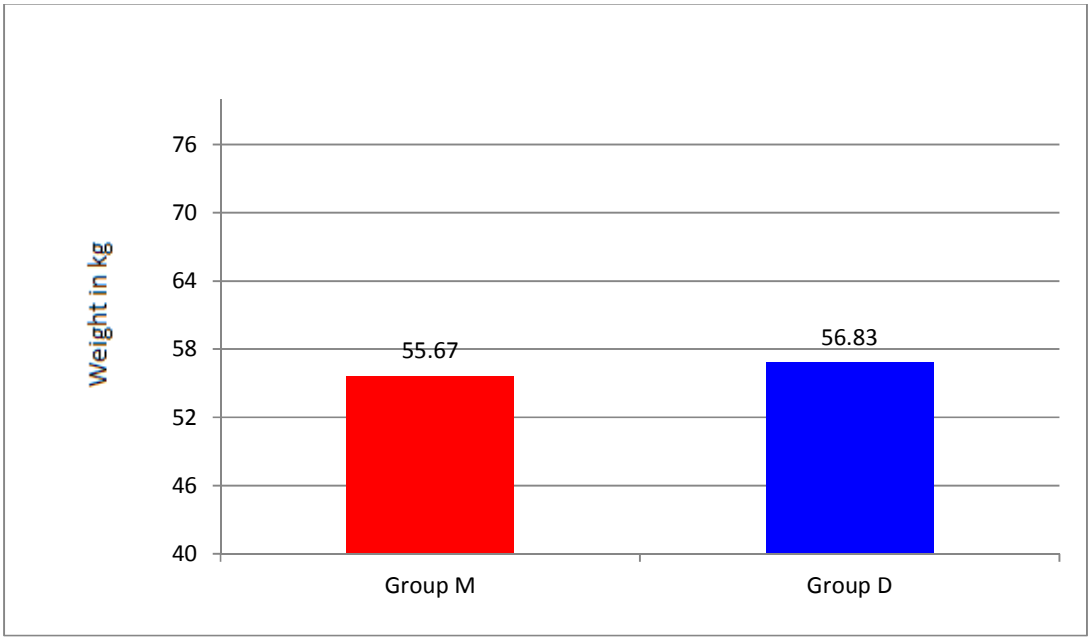
Mean age in both groups were around 39. The p value for mean age was not stastitically significant (p value = 0.806)

WEIGHT DISTRIBUTION

Comparison of groups on basis of weight

Study group	MEAN ± SD (weight in kg)	p value
M	55.67 ± 7.468	p = 0.602 Not significant
D	56.83 ± 8.272	

WEIGHT DISTRIBUTION



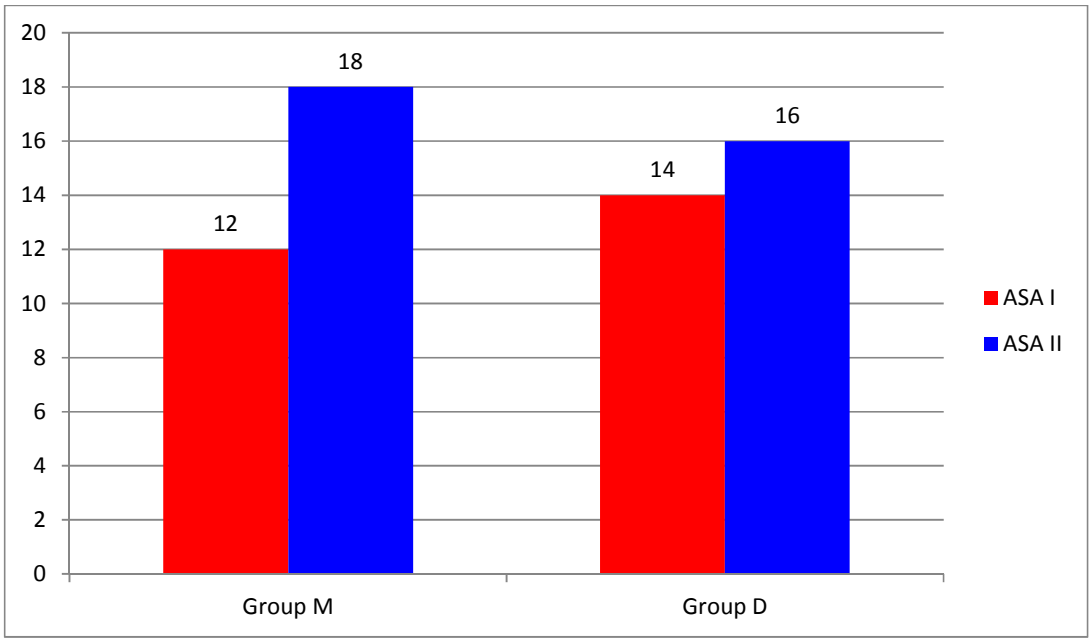
Mean weight in both groups were around 55. The P value for mean age was not statistically significant (p = 0.602)

ASA STATUS

Comparison of groups on basis of ASA Status

Study group	ASA I	ASA II	Total	p value
M	12	18	30	p = 0.710 Not significant
D	14	16	30	
Total	26	34	60	

ASA STATUS



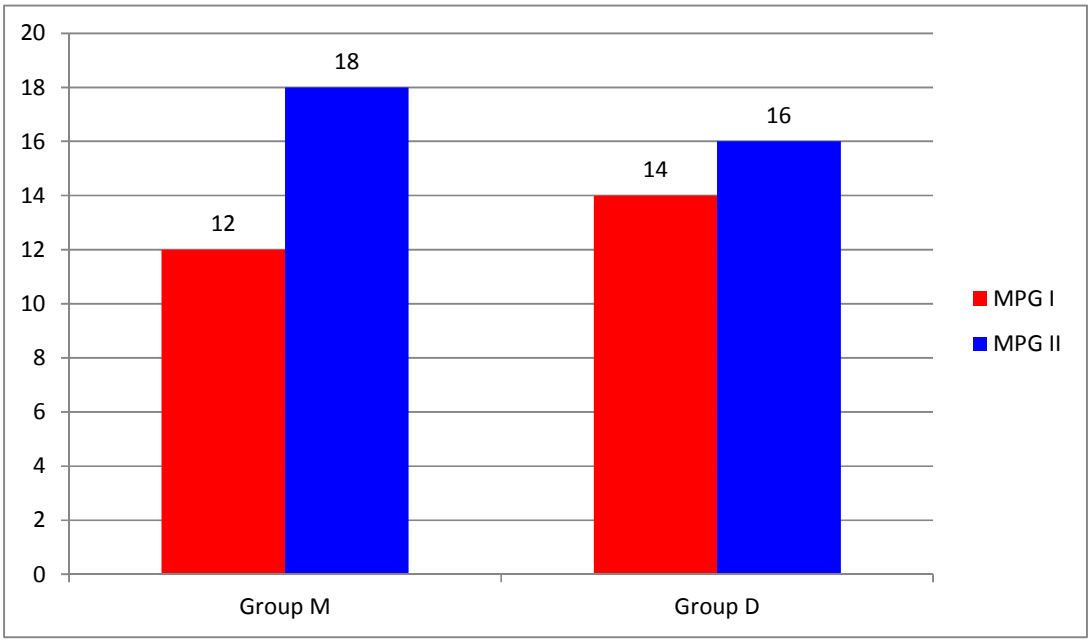
The p value for ASA status was not statistically significant (p=0.710). ASA status were comparable in both groups.

MALLAMPATTI GRADING

Comparison of group on basis of Mallampatti Grading

Study group	MPG I	MPG II	Total	p value
M	12(40%)	18(60%)	30	P = 0.722 Not significant
D	14((42%)	16(58%)	30	
Total	26(44%)	34(56%)	60	

MALLAMPATTI GRADING



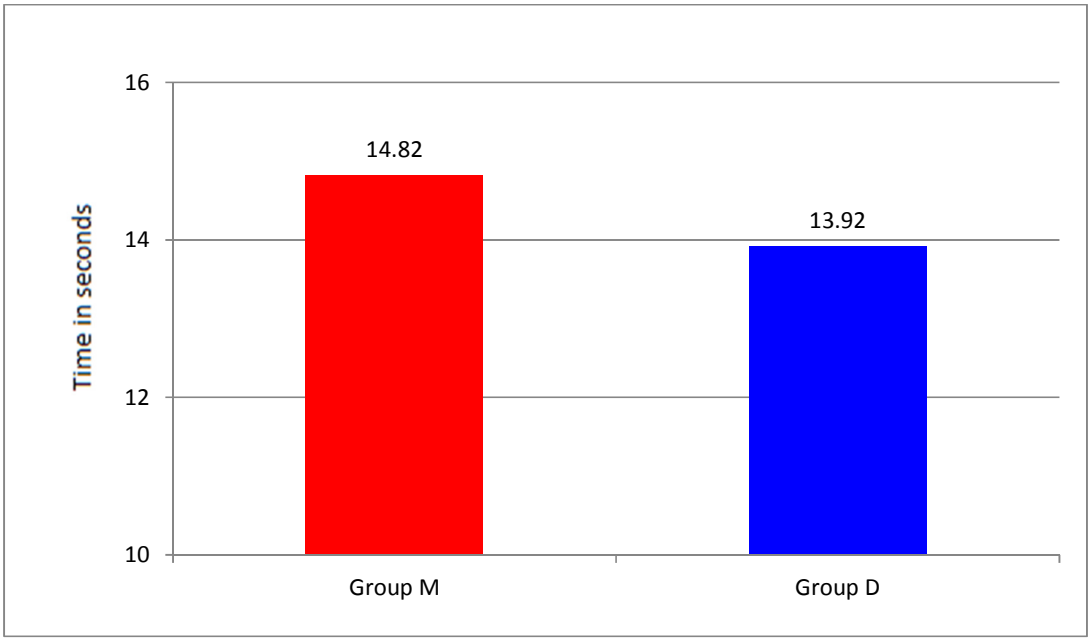
The p value for Mallampatti Grading was not statistically significant (p=0.722). Mallampatti Grading were comparable in both groups.

TOTAL DURATION OF LARYNGOSCOPY

Comparison of groups on basis of total duration of laryngoscopy

Study group	MEAN ± SD (Duration in seconds)	p value
M	14.82±5.88	P = 0.892 Not significant
D	13.92±6.02	
Total	14.37±5.23	

TOTAL DURATION OF LARYNGOSCOPY



The mean duration of the laryngoscopy in the group M was 14.8 seconds and group D was 13.9 seconds . The p value was 0.892 and statistically insignificant.

HEMODYNAMIC PROFILE

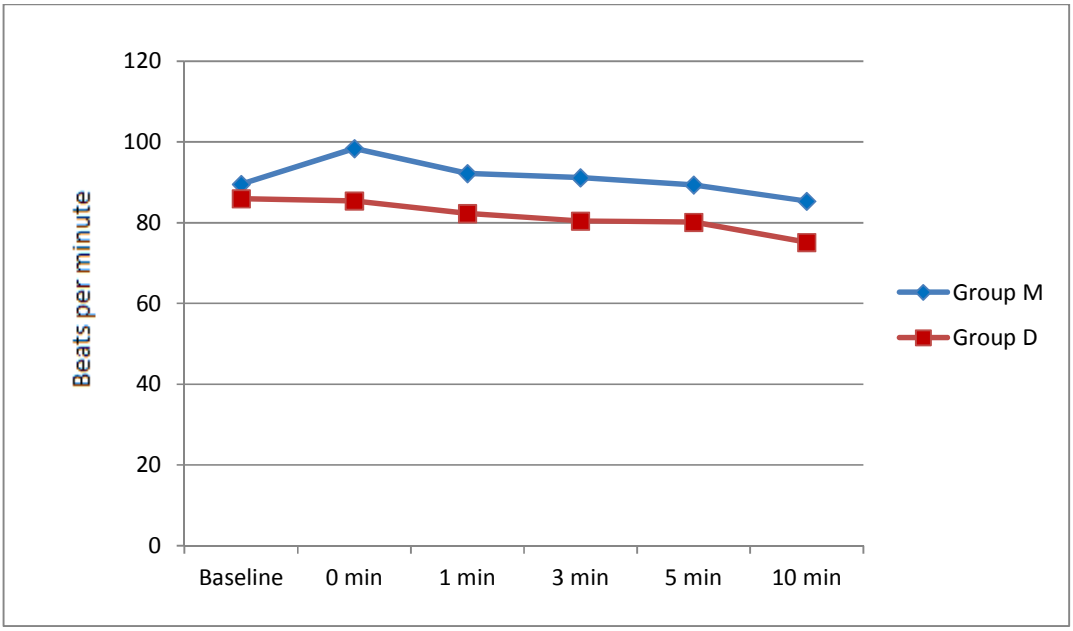
HEART RATE

Comparison of groups on basis of heart rate

Time	HEART RATE (per minute)		p value
	GroupM N=30 (mean ± SD	Group D N=30 (mean ± SD)	
Baseline	89.51± 15.2	85.92± 12.23	0.720
0 min	98.37± 13.92	85.43± 13.32	0.001
1 min	92.23± 10.62	82.27± 13.53	0.002
3 min	91.13± 11.89	80.47± 12.85	0.001
5 min	89.37± 13.55	80.12± 13.86	0.010
10 min	85.37± 12.47	75.17± 12.90	0.003

HEMODYNAMIC PROFILE

HEART RATE



Decrease in heart rate was more in Dexmedetomidine when compared to Magnesium sulphate. Heart rate values was statistically significant in Dexmedetomidine group when compared to Magnesium sulphate group ($p<0.005$) . At preinduction, the mean value for heart ratenoticed in group M was 89.51 and in group D was 85.92. At 0 minute that is immediately after intubation, mean heart rate noticed in group M was 98.37 and in group D was 85.43. At 1 minute, in group M was 92.23 and in group D was 82.27. At 3 minutes, in group M was 91.13 and in group D was 80.47. At 5 minutes, in group M was 89.37 and in group D was 80.12. At 10 minutes, in group M was 85.37 and in group D was 75.17.

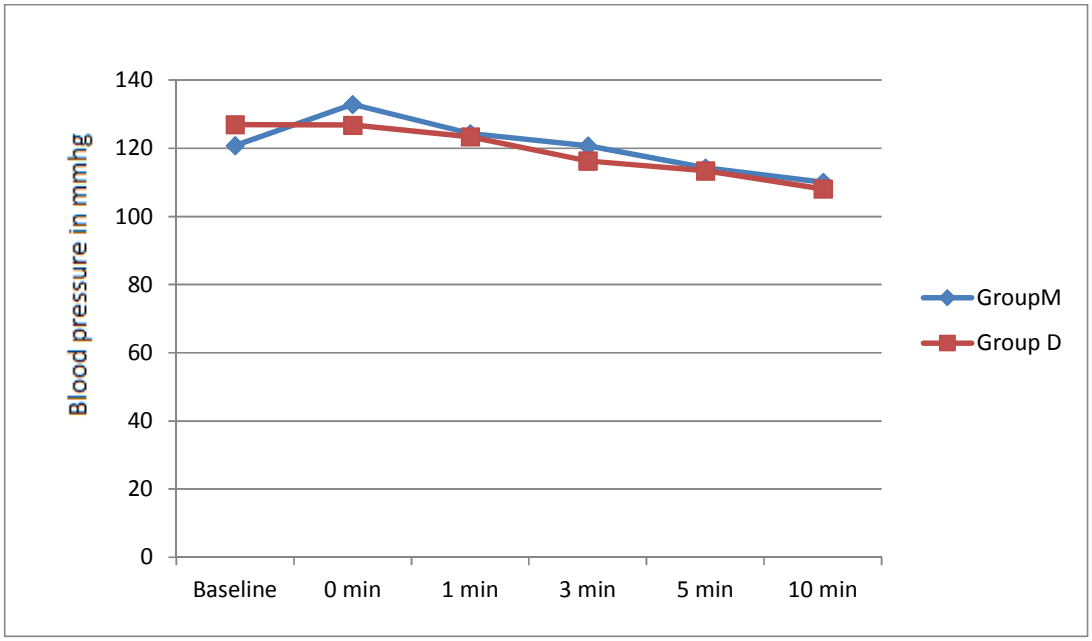
Though the p value for Heart rate was statistically significant, the mean values for heart rate in Magnesium sulphate group does not change more than 10 beats/minute. Heart rate returned to normal within 5 minutes interval. This shows that Magnesium sulphate was also effective in attenuating the cardiovascular stress response.

SYSTOLIC BLOOD PRESSURE

Comparison of groups on basis of systolic blood pressure

Time in min	SYSTOLIC BLOOD PRESSURE (mmhg)		p value	significance
	Group M (MEAN ±SD)	Group D (MEAN ± SD)		
Baseline	170.74± 12.2	126.93± 15.27	0.057	Not significant
0 min	132.89±25.91	126.74± 18.86	0.344	Not significant
1 min	124.32± 18.55	123.38± 14.55	0.920	Not significant
3 min	120.70± 18.41	116.32± 12.82	0.342	Not significant
5 min	114.23± 17.01	113.33± 14.29	0.761	Not significant
10 min	110.05± 14.41	108.08± 13.87	0.639	Not significant

SYSTOLIC BLOOD PRESSURE



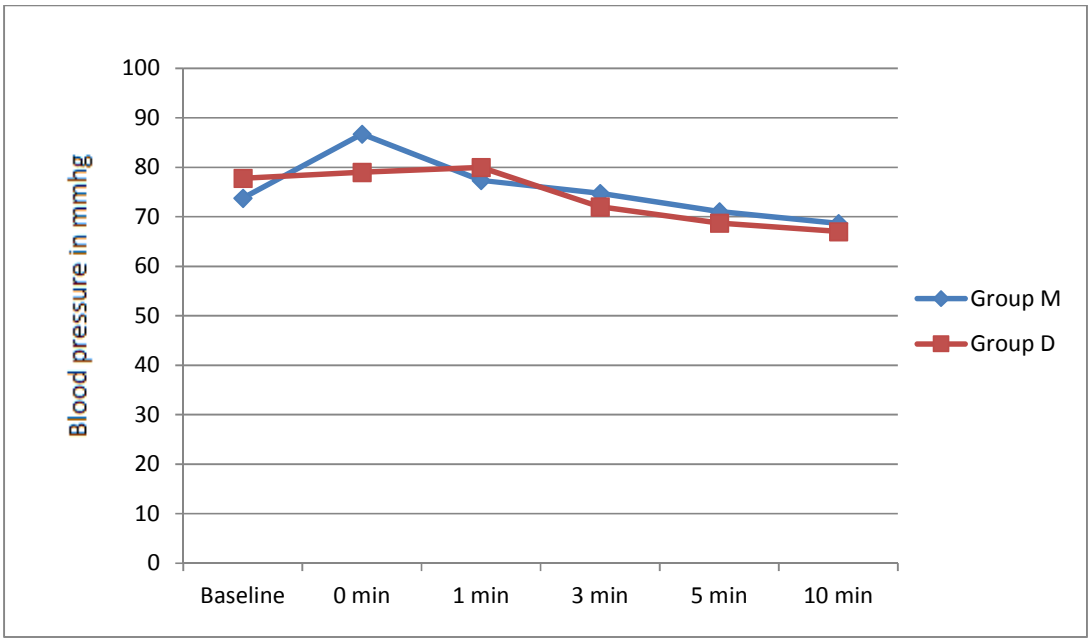
No statistically significant difference noted in systolic blood pressure between the groups

DIASTOLIC BLOOD PRESSURE

Comparison of two groups on basis of Mean Diastolic blood pressure

Time in min	DIASTOLIC BLOOD PRESSURE (mmhg)		p value	Significance
	Group M (MEAN ±SD)	Group D (MEAN ± SD)		
Baseline	73.79± 9.34	77.78± 8.59	0.148	Not significant
0 min	86.73± 17.34	79.03± 14.23	0.092	Not significant
1 min	77.37± 12.76	76.96±9.69	0.795	Not significant
3 min	74.74± 12.76	72.01± 12.76	0.393	Not significant
5 min	71.06± 10.55	68.73± 10.53	0.231	Not significant
10 min	68.70± 9.23	67.05± 10.74	0.825	Not significant

DIASTOLIC BLOOD PRESSURE



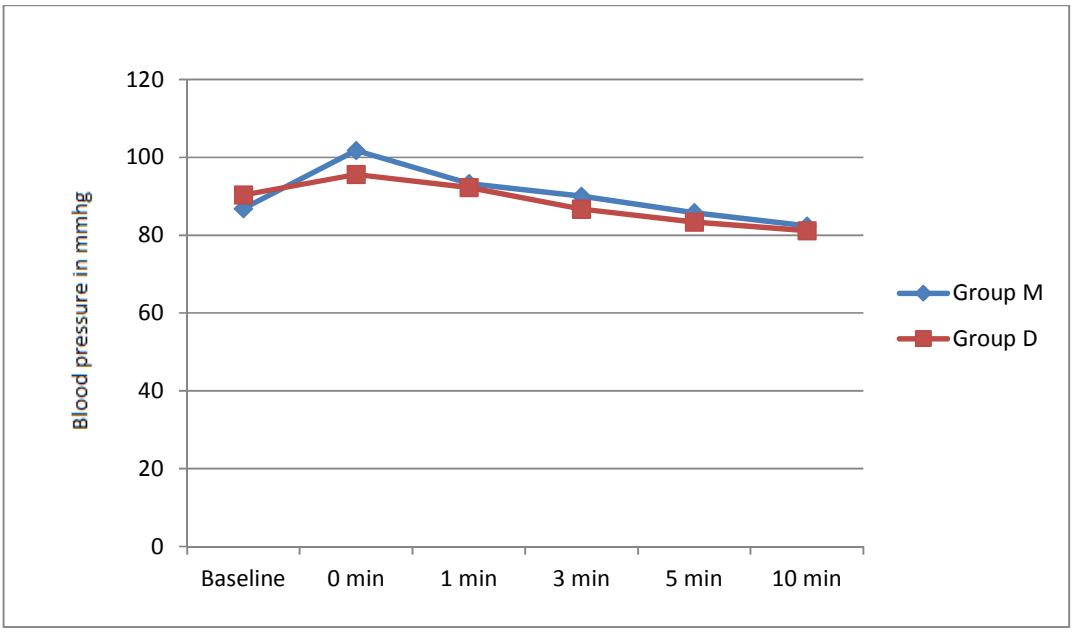
No statistically significant difference in diastolic blood pressure seen between these two groups

MEAN ARTERIAL BLOOD PRESSURE

Comparison of group on basis of men arterial pressure

Time in min	MEAN ARTERIAL BLOOD PRESSURE (mmhg)		p value	Significance
	Group M (MEAN ±SD)	Group D (MEAN ±SD)		
Baseline	86..88± 9.34	90.33± 8.59	0.076	Not significant
0 min	101.73± 17.34	95.55± 14.23	0.062	Not significant
1 min	93.23± 12.76	92.27±9.69	0.795	Not significant
3 min	90.02± 12.76	86.67± 12.76	0.361	Not significant
5 min	85.76± 10.55	83.33± 10.53	0.231	Not significant
10 min	82.35± 9.23	81.11± 10.74	0.825	Not significant

MEAN ARTERIAL BLOOD PRESSURE

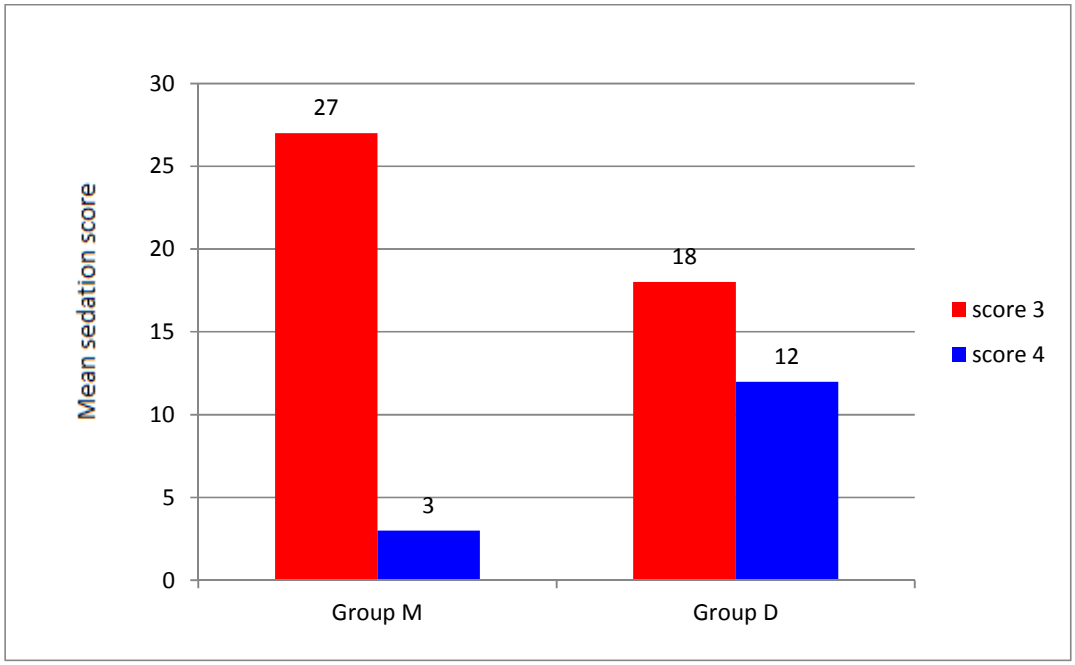


No statistically significant difference was noted in Mean arterial blood pressure between these two groups.

MODIFIED RAMSAY SEDATION SCORE

Study group	Score 3	Score 4	(MEAN±SD)	p value
M	27	3	3.11± 0.29	p = 0.003 significant
D	18	12	3.42±0.49	

MODIFIED RAMSAY SEDATION SCORE

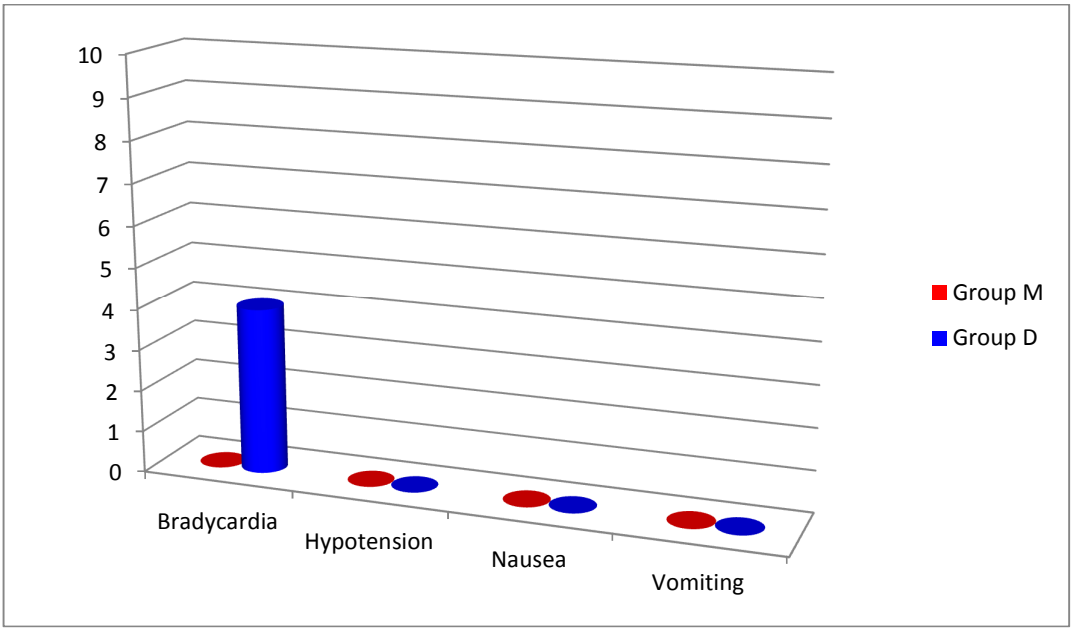


Statistically significant difference was observed in the Modified Ramsay sedation Score between the two groups. Magnesium sulphate was found to have significantly lesser sedation than Dexmedetomidine at the time of extubation.

ADVERSE EFFECTS

Adverse effects	Group M		Group D	
	No	%	No	%
Bradycardia	0	0	4	13
Hypotension	0	0	0	0
Nausea	0	0	0	0
Vomiting	0	0	0	0
Total	30	100	30	100

ADVERSE EFFECTS



In our study , we observed bradycardia as side effect which was noted in 13% of Dexmedetomidine group.

DISCUSSION

The hemodynamic response to laryngoscopy has been a topic of discussion since 1940. These responses can be worse in case of aged and patients with haemodynamic compromised state due to increased blood pressure, heart rate and oxygen consumption. Hypertension and tachycardia during laryngoscopy can occur even in normotensive patient and is rather surprising that complications are not that much common probably because of its transient nature. Therefore controlling this perioperative stress response is an important goal of anaesthesia.

Various drugs were evaluated during premedication or during induction to blunt pressor responses but the drugs which were used was either partially effective or they produced undesirable effects.

Most of the studies have compared the effect of intravenous Dexmedetomidine or Magnesium sulphate with that of the control. Very few studies were available that compare intravenous Dexmedetomidine and Magnesium sulphate for blunting pressor response. So, we planned for a prospective, randomized, double blinded study in Thanjavur Medical College Hospital, Thanjavur to compare the effect of Dexmedetomidine and Magnesium sulphate for attenuating stress response to intubation.

60 ASA physical status I and II subjects of v age 20-60 years planned for elective non cardiac surgeries were enrolled in this study. They were randomly allocated to one of the two study groups, Group D (Dexmedetomidine group) and Group M (Magnesium sulphate group).

Patient Characteristics across the group

The demographic characteristics like age, weight, ASA status of the study population were similar in both the two groups, with no statistically significant difference. The mean age in Magnesium sulphate group was 39.03 ± 3.86 years and in Dexmedetomidine group was 39.27 ± 3.45 years.

The mean weight in Magnesium sulphate group was 55.65 ± 7.468 kg and in Dexmedetomidine group was 56.83 ± 8.272 kg. The patients belonged to either ASA I or ASA II, and were comparable in both groups. Mallampatti Grading of patients were comparable in both group. Total duration of laryngoscopy in Magnesium sulphate group was 14.82 ± 5.88 seconds and in Dexmedetomidine group was 13.92 ± 6.02 seconds.

Dosage of the drug

In Group M , we used Magnesium sulphate 30mg/kg in 100ml normal saline and in group D Dexmedetomidine 1 μg/kg diluted in 100ml normal saline were used. The drug was given over a period of 15 minutes and were given 10 minutes before intubation.

In the Study done by Krishna chaitanya , they used Magnesium sulphate 30mg/kg and Dexmedetomidine 1 μg/kg . Both the drugs were effective in controlling the blood pressure but Dexmedetomidine was more effective in controlling the heart rate. Patients in both groups were hemodynamically stable. Both the drugs reduced the requirements of opioids, muscle relaxants and volatile anesthetics agents. Recovery in both the groups was uneventful. Similarly study conducted by Rashi kadam et al, used Magnesium sulphate 30 mg/kg and compared with norml saline. Systolic , Diastolic blood pressure values and heart rate in Magnesium group fell significantly during and after induction. Extubation and recovery were slower in study group.

Azin honarmand studied using different doses of Magnesium sulphate. Group 1: 30 mg/kg, Group 2: 40 mg/kg, Group 3: 50 mg/kg Hypotension is more seen in group 3 who received 50mg/kg Magnesium sulphate. No significant difference in extubation time between four groups.

K.Montazeri et aldivided patients into 5 groups , they took Magnesium sulphate (10, 20, 30, 40, 50mg/kg) 5 min before induction . In group C (30 mg/kg of Magnesium sulphate) change in heart rate and mean arterial pressure was less when compared to other groups ($p > 0.05$) .The incidence of complications of Magnesium sulphate like hypotension, arrhythmia, nausea, sweating, flushing had no significant differences .

Gautham pillai et al used Magnesium sulphate 30 mg/kg bolus before induction and 10 mg/kg/hr continous infusion. Hemodynamic parameter was significantly less in M group.

Kalkeri et al used Dexmedetomidine1µg /kg body weight diluted in 100 ml of normal saline given over 15 minutes before induction , significantly obtunded the haemodynamic stress responses intubation without significant side effects like hypotension and bradycardia .

Similarly venugopal et al used iv Dexmedetomidine 1 µg /kg in attenuation of stress response during intubation. Attenuation occurs within 5 minutes following laryngoscopy and intubation and becomes maximum by 10 minutes. The haemodynamic parameters reach near base line values after 5 to10 minutes of intubation.

Similarly Nabin porkhel et al used iv Dexmedetomidine 1 µg /kg in attenuating the intubation stress response.

Rajdip Hazra et al used Dexmedetomidine at the dose of 0.5 μg /kg in attenuating cardiovascular stress response

Saraf et al studied the efficacy of 0.6 μg/kg Dexmedetomidine to obtund the pressor response of tracheal intubation,

In our study, we avoided infusion of Magnesium sulfate and Dexmedetomidine. We used Magnesium sulphate at the dose of 30mg /kg which showed less incidence of hypotension and sedation when compared to 50mg/kg used by montazeri et al. We used Dexmedetomidine at the dose of 1μg /kg which showed desirable effect with less incidence of adverse effects.

Hemodynamic stability

Various studies have demonstrated the cardiovascular stress response with the use of intravenous Magnesium sulphate and Dexmedetomidine for laryngoscopy and intubation.

The variation in hemodynamic parameters like heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure was observed in the following intervals; baseline, during intubation (t0), 1 minute after intubation(t1), 3 minutes after intubation(t3), 5 minutes after intubation(t5), 10 minutes after intubation(t10).

HEART RATE

Krishna chaitanya et al observed decreasein heart rate was more with Dexmedetomidine compared to Magnesium sulphate. Heart rate values were less which were significant in Dexmedetomidine when compared to Magnesium sulphate ($p<0.005$). Though the p value for heart rate is statistically significant, the mean values for heart rate in Magnesium sulphate group did not change more than 10 beats/minute and heart rate returned to normal with in 5 minutes. The results were comparable with our study.

Rashi kadam et al also infused Magnesium sulphate 30mg/kg 5 minutes prior to intubation and observed significant fall in heart rate in Magnesium group at 1,3,5 and 10 minutes after intubation ($P< 0.001$) than the placebo group. This is in contrast with our study. But their baseline heart rate itself statistically significant and they used placebo as control. So that ,they observed significant decrease in heart rate in Magnesium sulphate group.

Gautham pillai et al used Magnesium sulphate 30 mg/kg bolus before induction and 10 mg/kg/hr continuous infusion , and compared with normal saline . Mean arterial pressure and heart rate just after intubation were significantly lower in Group M ($p < 0.05$) . This is in contrast to our

study. They observed bradycardia during the study, as they used continuous infusion. So we avoided continuous infusion during our study.

Kalkeri et al used Dexmedetomidine 1μg/kg. significant reduction in heart rate was noted in D group. The results was comparable with our study.

Venugopal et al used 1 μg/kg iv Dexmedetomidine . Group D showed better suppression of mean heart rate when compared to the control group(p=0.000) which was statistically significant. The results was comparable with our study.

Saraf et al used Dexmedetomidine 0.6μg/kg, showed decrease in heart rate with group D . The results was comparable with our study.

Gautham pillai et al used Magnesium sulphate 30 mg/kg bolus and 10 mg/kg/hr continuous infusion , and compared with normal saline . Heart rate were significantly lower in Group M ($p < 0.05$)

Our study showed statistically significant decrease in heart rate in Dexmedetomidine group when compared to Magnesium sulphate which was ($P<0.001$).

Systolic blood pressure

Krishna chaitanya et al used Magnesium sulphate 30 mg/kg and Dexmedetomidine 1 μg/kg and they observed no significant difference

among the drugs at 0, 1, 3, 5, and 10 minutes for systolic blood pressures ($p > 0.005$). The results were comparable with our study.

Rajan sunil et al did a randomised control study using iv Magnesium sulphate 50mg/kg and those in group B received 1.5mg/kg lignocaine 90sec before intubation . There was no significant difference between the groups. The results were comparable with our study.

Rashi kadam et al used Magnesium sulphate 30mg/kg 5 min prior to induction . The average systolic blood pressure in study group in first 5 min after intubation was significantly low. As he used placebo as control the difference in systolic blood pressure were statistically significant. This was in contrast with our study.

Arun kumar patra et al used Dexmedetomidine 1 µg/kg The average systolic blood pressure in study group in first 5 min after intubation was significantly low which was statistically significant. This was in contrast with our study.

Kalkeri et al used Dexmedetomidine 1µg/kg, 5 min prior to induction and normal saline as control . He observed statistically significant decrease in blood pressure when compared to control group. This was in contrast with our study, the reason behind was, they used normal saline as control.

Rajdip hazra et al used Dexmedetomidine 0.5µg/kg , 5 min prior to induction and normal saline as control. He observed statistically significant

decrease in blood pressure when compared to control group. This was in contrast with our study.

In our study, there was no statistically significant variations in systolic blood pressure observed between the two groups at various intervals.

Diastolic blood pressure

Krishna chaitanya et al used Magnesium sulphate 30 mg/kg and Dexmedetomidine 1 μg/kg and there was no statistically significant difference between both the drugs at 0, 1, 3, 5, and 10 minutes for diastolic blood pressures ($p > 0.005$). The results were comparable with our study.

Rajan sunil et al did a randomised control study, he used iv Magnesium sulphate 50mg/kg body weight given 10 min before induction and those in group B received 1.5mg/kg lignocaine 90sec before intubation. There was a decrease in diastolic blood pressure from induction, there was no significant difference between the groups. The results were comparable with our study

Rashi kadam et al used Magnesium sulphate 30mg/kg 5 min prior to induction.The average diastolic blood pressure in study group in first 5 min

after intubation was significantly less(p<.005)when compared to control group.

Saraf et al used Dexmedetomidine 0.6µg/kg and he observed significant decrease in diastolic blood pressure than control group.

Arun kumar patra et al used Dexmedetomidine 1 µg/kg .The average Diastolic blood pressure in study group in first 5 min afterintubation is significantly less as compared to control group which was statistically significant.. This was in contrast with our study.

The reason behind may be that normal saline had been used as a control which has no role in controlling cardiovascular stress resonse to laryngoscopy and endotracheal intubation. Thus during laryngoscopy they showed significant increase in systolic, diastolic and mean arterial pressure.

In our study, No statistically significant variations in Diastolic blood pressure observed among the two groups at various time intervals.

Mean arterial blood pressure

Krishna chaitanya et al used Magnesium sulphate 30 mg/kg and Dexmedetomidine 1 µg/kg and there is no statistically significant(p> 0.005) difference between mean arterial pressures for both the drugs at 0,

1, 3, 5, and 10 minutes interval.. The results were comparable with our study.

Rajan sunil et al did a randomised control study . He used iv Magnesium sulphate 50mg/kg body weight 10 min before induction and those allocated in group B received 1.5mg/kg lignocaine 90sec before intubation. There was a decrease in mean arterial pressure from inductionat 0, 1, 3, 5, and 10 minutes interval but no significant difference between the groups. The results were comparable with our study.

Abbady A. Ahmed etal done the study with Magnesium sulphate 3omg/kg and noticed significant difference in mean arterial blood pressure between Magnesium and control group. The result was contrast to our study.

K.montazeri et al did a randomised control study using iv Magnesium sulphate 30mg/kg body weight 10 min before inducting the patient and there was significant difference in mean arterial blood pressure between two group. The result was contrast to our study.

Venugopal et al used Dexmedetomidine 1µg/kg. He observed significant decrease in mean blood pressure than control group.

Saraf et al used Dexmedetomidine 0.6μg/kg, he observed significant decrease in mean arterial pressure than control group. This was in contrast to our study.

In our study, there was no statistically significant variations in mean arterial pressure observed between the two groups at various time intervals.

Level of sedation

Both Magnesium sulphate and Dexmedetomidine produce sedation. In our study, we compared the sedative effect of Magnesium sulphate and Dexmedetomidine by using the modified Ramsay Sedation Scale at the time of extubation. We have observed that 27 patients out of 30 in the Magnesium sulphate group had a score of 3 (90%) and 3 patients had a score of 4 (10%). But in the Dexmedetomidine group, out of 30 patients, 18 patients had a score of 3 (60%) and rest of the 12 patients had the score of 4 (40%). This difference in sedative effect could be explained by the prolonged sedative effect of Dexmedetomidine than Magnesium sulphate. The p value is <0.003 and found to be statistically significant.

Saraf et al observed that patient receiving Dexmedetomidine showed no significant difference in sedation score when compared to control group. This is in contrast to our study.

Arun kumar patra et al observed sedation in Dexmedetomidine group compared to control group.

Rajan sunil et al observed that using Magnesium sulphate 30mg/kg before induction, showed delayed recovery during extubation due to sedation when compared to control group.

Abass Sedighinejad et al conducted a study to compare Magnesium Sulfate and sufentanil for Patient-Controlled Analgesia in Orthopedic Surgery. They concluded that the Magnesium group showed no significant difference in sedation score with sufentanil.

Yildiz et al evaluated the single dose of Dexmedetomidine 1 ug/kg . He observed sedation in Dexmedetomidine group was significant when compared to control.

Adverse effects

In our study we observed for adverse effects like bradycardia, hypotension , nausea , vomiting and shivering,. In the Magnesium group no patients had any adverse effects. In dexmeditomidine group we observed episodes of bradycardia in 4 out of 30 patients (13%).

Kalkeri et al used Dexmedetomidine 1µg/kg 5 min before induction. In their study 2 patients developed bradycardia and 6 patients developed hypotension, 5min after intubation. One patient required inj. atropine 0.6mg for bradycardia and no patient required vasopressors for correction

of blood pressure. Hypotension was managed by decreasing volatile anaesthetic concentration and infusing I.V fluids.

Saraf et al used Dexmedetomidine 1μg/kg. 2 patient had bradycardia, 3 had hypotension and one patient had both bradycardia and hypotension in Dexmedetomidine group which did not need treatment.

Rajdip hazra et al used Dexmedetomidine 1μg/kg. In this study, out of 45 patients in Dexmedetomidine group, 2 patients developed bradycardia and another 3 patients developed hypotension

Nabin porkhel et al used Dexmedetomidine 1μg/kg and there was no adverse effects observed in his study. Slow infusion of Dexmedetomidine over 10 minutes might have prevented bradycardia and hypotension

Azin honarmad used different doses of Magnesium sulphate (Group i: 30 mg/kg, Group ii: 40 mg/kg, Group iii: 50 mg/kg) and control . There was no difference in the incidence of bradycardia or hypotension in his study.These results were comparable with our study.

Our study shows that fall in heart rate was seen in both Magnesium sulphate and Dexmedetomidine group, but the fall is more significant in Dexmedetomidine group. In our study we have observed bradycardia in 13% of patients.

SUMMARY

We conducted a prospective double blinded randomized control study in 60 patients undergoing elective non cardiac surgeries in Thanjavur medical college Hospital, Thanjavur. Patients of both sexes ranging between 20 to 60 years of age were included. Our aim was to evaluate and compare the effect of intravenously administered Magnesium sulfate 30mg/kg and Dexmedetomidine 1µg/kg for attenuation of the cardiovascular stress responses to laryngoscopy and endotracheal intubtion

Patients were divided randomly using closed cover technique into two groups of 30 each. Group M received Magnesium sulfate 30mg/kg in 100 ml of normal saline. Group D received Dexmedetomidine 1µg/kgin 100 ml of normal saline.

The test drug solution was given over 15 min and 10 min before induction. The heat rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, adverse effects and level of sedation assessed by Modified Ramsay Sedation Score were noted in both groups.

The categorical data collected were analyzed by Chi Square test and the nominal data were analyzed by One-Way ANOVA. The results were obtained in the form of range, mean and standard deviation. The probability value 'p' of less than 0.05 was considered statistically significant. In our study we noticed decrease in heart rate was more with Dexmedetomidine compared to Magnesium sulphate. Heart rate values are statistically significantly less in Dexmedetomidine group compared to Magnesium sulphate group ($p<0.005$) . But the increase in heart rate in Magnesium sulphate was not more than 10 beats/min and also the heart rate returned to baseline within 5minutes.

There was no statistically significant difference observed in systolic blood pressure, diastolic blood pressure and mean arterial pressure in our study between those groups. However, the level of sedation in Magnesium sulphate group is less. The patients were observed for adverse effects like bradycardia, hypotension, postoperative nausea, vomiting, and shivering. In Dexmedetomidine group, we observed bradycardia in 13% of patients.

In our study we compared Magnesium sulphate with Dexmedetomidine to attenuate the cardiovascular stress response to intubation.

Both the drugs were comparable in controlling the blood pressure but Dexmedetomidine was far more effective in controlling the heart rate Patients in both the groups were hemodynamically stable throughout the perioperative period. Both the drugs reduced the requirements of opioids, muscle relaxants and volatile anaesthetics. Recovery in both the groups was uneventful.

CONCLUSION

Intravenously administered Magnesium sulfate 30mg/kg given before intubation is equally effective as Dexmedetomidine 1μg/kgfor attenuation of the cardiovascular stress responses to laryngoscopy and endotracheal intubation

BIBLIOGRAPHY

1. King BD, Harris LC, "Reflex circulatory responses to Direct laryngoscopy and Tracheal intubation performed during General anaesthesia", *Anesthesiology, 1951;12: 556-566*
2. Stoelting RK, Hiller SC, " Pharmacology and physiology in anesthetic practice", Philadelphia, Lippincott Williams and Wilkins*,2006,340*
3. Bloor BC, Ward DS, Belleville JP, Maze M. Effects of intravenous Dexmedetomidine in humans. II Haemodynamic changes. Anaesthesiology 1992;77:1134-1142.
4. Scheinin B, Lindgren L, Randell T, Scheinin H, Scheinin M. Dexmedetomidine attenuates sympathoadrenal responses to tracheal intubation and reduces the need for thiopentone and peroperative fentanyl. British journal of anaesthesiology 1992;68:126-131
5. Jakola ML, Ali-Melkkila T, Kanto J, Kallio A, Scheinin H, Scheinin M. Dexmedetomidine reduces intraocular pressure, intubation response and anaesthetic requirements in patients undergoing ophthalmic surgery. British journal of anaesthesiology 1992;68:570-575
6. Reid, Brace: Irritation of respiratory tract and itsreflex effect on heart-Surgery GynaecologyObstetrics. 1940; 70:157

6.Deem SA, Bishop MJ, Bedford RF. Physiological and pathological response to intubation. In: Hagberg CA editor. Benumof's Airway Management. 2nd ed. Philadelphia: Mosby Elsevier, 2007;193-212.

7.Berry A.Brimacombe J,Keller C pulmonary airway resistance with endotracheal tube

8. Kovac AL. Controlling the hemodynamic response to laryngoscopy and endotracheal intubation. J Clin Anesth, 1996;8:63-79.

9. Kayhan Z, Aldemir D, Metler H, Ogus E. Which is responsible for the haemodynamic response due to the laryngoscopy and endotracheal intubation? Catecholamines, vasopressin or angiotensin? Eur J Anaesthesiol, 2005;22:780-785.

10. Edwards ND, Alford AM, Dobson PMS, Peacock JE, Reilly CS. Myocardial ischaemia during tracheal intubation and extubation. Br J Anaesth, 1994;73:537-539.

11.Stoelting RK, Hiller SC. Pharmacology and Physiology in Anesthetic Practice. 4th ed. Lippincott Williams and Wilkins, 2006;340-345.

12.Kayhan, Z., Aldemir, D., Metler, H., Ogus, E. 2005. Which is responsible for the haemodynamic response due to the laryngoscopy and endotrachealintubation? Catecholamines, vasopressin or angiotensin? *Eur. J.Anaesthesiol.,* 22: 780 5.

13.King, B.D., Harris, L., Greifenstein, F.,Elder, J., Dripps, R.D. 1951. Reflexcirculatory responses to directlaryngoscopy and intubation undergeneral anaesthesia. *Anaesthesiology,*12: 556 665. Virtanen R, Savola J, Nyman

14. Smith P., Smith F. J., Becker P. J. Haemodynamic response to laryngoscopy withand without intubation. SAJAA 2008; 14(3): 23-26.

15. Abbady A. Ahmed. Treatment of stress reponse to laryngoscopy and intubation20.Naghibi K. H., Akhtari M.Attenuation of the pressor responses to tracheal intubation by Magnesium sulphate. Res Med Sci J 2000; 1:42-4with Magnesium sulphate. El- Minia Med. Bull. 2009; 20(2): 191-196.

16.Telci L, Esen F, Akcora D, Erden T, Canbolat AT, Akpir K. Evaluation of effects of Magnesium sulphate in reducingintraoperative anaesthetic requirements. Br J Anaesth 2002 Oct;89(4):594-8.

17. Fawcett WJ, Haxby EJ, Male DA. Magnesium: physiology and pharmacology. Br J Anaesth1999 Aug;83(2):302-20.

18. Vigorito C, Giordano A, Ferraro P, Acanfora D, De Caprio L, Naddeo C, et al. Hemodynamic effects of Magnesiumsulfate on the normal human heart. Am J Cardiol1991 Jun 15;67(16):1435-7.

19. James MF, Beer RE, Esser JD. Intravenous Magnesium sulfate inhibits catecholamine release associated with trachealintubation. Anesth Analg 1989 Jun;68(6):772-6.

20. Puri GD, Marudhachalam KS, Chari P, Suri RK. The effect of Magnesium sulphate on hemodynamics and its efficacy inattenuating the response to endotracheal intubation in patients with coronary artery disease. Anesth Analg1998Oct;87(4):808-11.

21. Ghoneim MM, Long JP: The interaction between Magnesium and other neuromuscular blocking agents. Anesthesiology 1970; 32:23–7

22 Del CJ, Engbaek L: The nature of the neuromuscular block produced by Magnesium. Journal of Physiology 1954; 124:370 – 84 26 Iseri LT, French JH: Magnesium: Nature's physiologic calcium blocker. Am Heart J 1984; 108:188 –9327 Tramer MR, Schneider J, Marti RA, Rifat K: Role of Magnesium sulfate in postoperative analgesia. Anesthesiology 1996; 84:340

23. Characterization of the selectivity, specifityand potency of medetomidine as an α-2 adrenoceptor agonist. Eur JPharmacol 1988:9-14.

24. Scheinin H, Virtanen R, MacDonald E, Lammintausta R. Medetomidine-anovel α-2-adrenoceptor agonist: A review of its pharmacological effects.Prog Neuro-Psychopharmacol Biol Psychiatry 1989;13:635-51.

25. Keith A, Sergio D, Paula M, Marc A, Wisemandle W, Alex Y. Monitoredanesthesia care with Dexmedetomidine: A prospective, randomized,double-blind, multicenter trial. Anesth Analg 2010;110:47-56.

26. Bloor BC, Ward DS, Belleville JP, Maze M. Effects of intravenous Dexmedetomidine in humans. II. Hemodynamic changes. Anesthesiology1992;77:1134-42.

27. Khan ZP, Ferguson CN, Jones RM. Alpha-2 and imidazoline receptor agonists.Their pharmacology and therapeutic role. Anaesthesia 1999;54:146-65

PROFOMA

COMPARITIVE STUDY BETWEEN INTRAVENOUS 50% MAGNESIUM SULPHATE AND DEXMEDETOMIDINE FOR ATTENUATION OF CARDIOVASCULAR STRESS RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

Name: IP No:

Age:

Sex:

Weight:

ASA: I/II/III/IV

MPG:I/II/III/IV

Diagnosis:

Procedure:

Group: M/D

Pre-operative examination:

Pulse rate: /min

BP: mmhg

CVS:

RS:

Duration of laryngoscopy:

Intra-op findings:

Parameter	baseline	0 min	1 min	3min	5 min	10 min
HR						
SBP						
DBP						
MAP						

Level of sedation at the time of extubation:

Postop period:

Nausea

Vomiting

Shivering

Modified Ramsay Sedation Score

score	Level of activity
0	Paralyzed , unable to evaluate
1	Awake
2	Lightly sedated
3	Moderately sedated, follows simple commands
4	Deeply sedated , responds to non-painful stimuli
5	Deeply sedated, responds only to painful stimuli
6	Deeply sedated, unresponsive to painful stimuli

MASTER CHART

S.no	Name	Age	Sex	Wt	MPG	ASA	Surgery	group	duration of laryngoscopy	baseline			
										HR	SBP	DBP	MAP
1	Seetha	32	F	48	I	2	Fibroadenoma	M	16	73	114	74	88
2	Hari	33	M	62	I	2	Lipoma	M	15	75	120	76	86
3	Pavitha	46	F	52	II	1	Fibroadenoma	M	14	68	124	74	85
4	Gokul	34	M	50	I	2	Lipoma	M	20	84	140	78	90
5	Jeevitha	26	F	45	I	2	FESS	M	18	76	130	78	86
6	Saravanan	39	M	68	I	2	Mastoidectomy	M	12	85	100	88	84
7	Saradha	45	F	59	II	1	MLE	M	11	74	114	86	90
8	Mani	35	M	68	I	2	Rhinosporidiosis	M	14	76	120	84	84
9	Shiva	48	M	54	II	1	FESS	M	15	72	124	76	90
10	Guru	33	M	63	I	2	Mastoidectomy	M	14	66	126	78	85
11	Kannan	24	M	50	I	2	Tonsillectomy	M	16	73	130	65	80
12	Savitha	35	F	42	II	1	Lipoma	M	22	78	126	66	84
13	Moorthi	39	M	65	I	2	ORIF	M	15	71	104	62	76
14	Smitha	44	F	54	II	1	Lipoma	M	18	76	125	84	87
15	Vadivu	52	F	49	I	2	FESS	M	16	64	104	66	98
16	Murugesan	56	M	52	II	1	Mastoidectomy	M	14	66	124	70	86
17	Karthyikeyan	32	M	62	I	2	Mastoidectomy	M	15	87	134	72	84
18	Senthil	28	M	51	I	2	Tonsillectomy	M	19	70	114	64	97
19	Paunambal	36	F	58	I	2	FESS	M	11	68	126	68	74
20	Jayalakshmi	47	F	62	II	1	Mastoidectomy	M	16	61	134	74	86
21	Kumari	34	F	56	I	2	Tonsillectomy	M	17	68	124	76	77
22	Nirmala	44	F	48	I	2	FESS	M	14	78	128	68	86

S.no	Name	Age	Sex	Wt	MPG	ASA	Surgery	group	duration of laryngoscopy	baseline			
										HR	SBP	DBP	MAP
23	Paniyan	56	M	64	II	1	ORIF	M	13	87	126	74	96
24	Muthu	45	M	68	I	2	FESS	M	18	65	128	76	86
25	Venktachalam	47	M	61	II	1	Mastoidectomy	M	12	76	104	84	84
26	Chinna	45	M	45	II	1	Tonsillectomy	M	10	75	120	74	98
27	Subash	38	M	54	I	2	ORIF	M	14	62	116	68	84
28	Subramaniam	36	M	60	II	1	Mastoidectomy	M	13	77	124	66	76
29	Saraswathy	30	F	49	I	2	Mastoidectomy	M	12	76	104	74	94
30	Kabilan	32	M	45	II	1	Lipoma	M	10	82	110	68	97
31	Swathi	28	F	60	II	1	Tonsillectomy	D	11	77	128	84	88
32	Haribaskar	38	M	62	II	1	ORIF	D	15	78	120	76	86
33	Prabavathy	39	F	45	I	2	FESS	D	16	66	134	88	106
34	Lalitha	46	F	54	I	2	Lipoma	D	18	95	140	74	101
35	Palani	35	M	59	II	1	Mastoidectomy	D	14	88	130	76	78
36	Sakthivel	37	M	51	II	1	Mastoidectomy	D	12	68	118	86	92
37	Sumathi	29	F	40	I	2	MLE	D	10	92	114	78	96
38	Dharani	53	F	58	II	1	Fibroadenoma	D	16	65	136	78	98
39	Kumaran	29	M	55	I	2	Mastoidectomy	D	14	66	124	88	98
40	Kamalakannan	38	M	68	II	1	FESS	D	14	63	126	86	88
41	Thirupathi	44	M	60	I	2	Rhinosporidiosis	D	15	78	130	84	94
42	Selvam	57	M	59	I	2	ORIF	D	12	74	138	76	86
43	Thirumurugan	37	M	55	II	1	Mastoidectomy	D	16	85	104	88	84
44	Selvi	46	F	65	II	1	ORIF	D	12	90	125	65	82
45	Veluchamy	38	M	57	I	2	ORIF	D	13	64	138	76	104

S.no	Name	Age	Sex	Wt	MPG	ASA	Surgery	group	duration of laryngoscopy	baseline			
										HR	SBP	DBP	MAP
46	Chinnaiyan	25	M	51	I	2	FESS	D	18	68	140	62	99
47	Murugan	39	M	68	II	1	Mastoidectomy	D	11	75	134	84	86
48	Tamil	42	M	60	I	2	FESS	D	14	91	114	66	84
49	Thiruselvi	44	F	49	I	2	FESS	D	16	84	126	88	82
50	Kangam	54	F	54	II	1	Mastoidectomy	D	12	68	134	78	102
51	Thirumal	38	M	56	I	2	SMR	D	14	92	124	74	88
52	Vishvam	46	M	62	II	1	SMR	D	11	77	134	68	98
53	Kamalam	37	F	53	I	2	Mastoidectomy	D	12	76	126	74	79
54	Parvathi	36	F	68	I	2	FESS	D	14	64	128	76	88
55	Srimathi	46	F	51	II	1	MLE	D	16	77	114	88	84
56	Karkuzhali	36	F	55	II	1	MLE	D	14	92	120	74	87
57	Thirunavukarasu	37	M	56	I	2	Lipoma	D	10	81	116	76	78
58	Punniyamoorthi	46	M	60	I	2	FESS	D	16	65	128	84	96
59	Kavya	29	F	55	II	1	Fibroadenoma	D	14	82	136	74	97
60	Vanaroja	38	F	62	I	2	Fibroadenoma	D	18	88	110	68	84

S.no	Name	0 MIN				1 MIN				3 MIN			
		HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
1	Seetha	98	134	88	99	76	124	82	97	75	114	72	80
2	Hari	88	130	90	96	75	128	74	86	78	120	74	92
3	Pavitha	87	138	96	98	84	132	86	100	76	124	62	86
4	Gokul	88	130	84	102	78	134	84	94	75	140	66	105
5	Jeevitha	85	136	76	101	88	118	76	96	74	130	74	90
6	Saravanan	87	146	100	98	74	140	88	106	72	100	88	86
7	Saradha	88	136	94	102	85	126	74	92	75	114	76	90
8	Mani	98	130	94	111	90	114	76	90	84	120	74	84
9	Shiva	82	138	84	106	77	118	86	98	76	124	70	99
10	Guru	76	126	76	96	75	126	78	86	74	126	84	88
11	Kannan	99	134	88	114	75	120	78	92	78	130	76	92
12	Savitha	85	160	84	120	72	122	88	94	66	126	74	94
13	Moorthi	81	134	92	100	62	126	86	96	60	104	78	80
14	Smitha	86	140	96	116	75	114	84	90	70	125	67	90
15	Vadivu	88	140	84	118	76	124	76	94	66	104	72	84
16	Murugesan	76	126	82	90	86	116	88	96	82	124	81	98
17	Karthyikeyan	90	124	86	98	78	104	65	92	74	134	68	84
18	Senthil	86	123	78	94	76	128	76	94	76	114	76	94
19	Paunambal	78	134	78	100	70	140	62	102	65	126	77	91
20	Jayalakshmi	97	114	86	96	80	118	74	82	76	134	76	93
21	Kumari	98	128	88	98	64	114	66	90	60	124	80	94
22	Nirmala	88	126	79	96	75	137	78	98	70	128	72	96

S.no	Name	0 MIN				1 MIN				3 MIN			
		HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
23	Paniyan	97	120	86	97	87	128	78	84	84	126	64	91
24	Muthu	75	140	94	105	84	116	74	87	76	128	86	86
25	Venktachalam	96	128	78	100	80	116	68	96	84	104	72	98
26	Chinna	80	126	85	98	85	138	74	90	85	120	74	88
27	Subash	70	136	88	96	70	134	76	91	74	116	82	91
28	Subramaniam	81	140	84	110	74	146	88	110	75	124	76	99
29	Saraswathy	85	136	90	98	80	114	74	87	80	104	84	86
30	Kabilan	80	128	86	100	62	108	76	86	60	110	63	84
31	Swathi	89	128	82	88	76	114	84	92	75	114	84	88
32	Haribaskar	70	120	75	86	75	118	74	94	68	113	70	86
33	Prabavathy	65	140	76	98	54	122	68	88	66	125	68	85
34	Lalitha	88	134	74	92	78	134	76	97	75	124	72	90
35	Palani	78	130	74	94	88	118	74	86	74	102	60	86
36	Sakthivel	75	140	86	102	74	136	70	90	68	124	76	84
37	Sumathi	98	114	72	80	85	126	84	94	75	118	62	90
38	Dharani	76	136	86	99	90	114	76	96	84	124	74	84
39	Kumaran	80	126	84	96	77	123	74	106	76	126	72	90
40	Kamalakannan	84	124	88	98	75	136	78	84	74	102	74	85
41	Thirupathi	80	130	78	102	75	120	84	90	78	124	62	80
42	Selvam	76	138	71	101	58	122	72	98	66	120	66	84
43	Thirumurugan	80	126	84	98	74	126	81	86	60	118	74	76
44	Selvi	88	114	86	102	75	114	82	92	65	106	68	87
45	Veluchamy	70	138	80	90	76	124	76	94	66	104	76	98

S.no	Name	0 MIN				1 MIN				3 MIN			
		HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP
46	Chinnaiyan	70	134	78	106	76	116	77	92	82	124	74	86
47	Murugan	80	140	76	96	78	116	76	90	74	112	70	84
48	Tamil	86	114	85	80	76	128	80	94	76	104	84	97
49	Thiruselvi	88	126	74	88	70	140	72	96	65	118	76	74
50	Kangam	70	124	88	80	80	118	64	92	74	124	74	86
51	Thirumal	90	124	76	96	64	128	86	94	60	116	78	77
52	Vishvam	78	134	71	118	75	124	72	102	70	102	67	86
53	Kamalam	80	114	80	90	87	136	74	82	74	118	72	96
54	Parvathi	67	128	87	98	54	124	62	90	76	126	81	86
55	Srimathi	70	126	82	94	80	125	76	98	84	124	68	84
56	Karkuzhali	90	120	74	100	85	130	84	84	85	122	76	98
57	Thirunavukarasu	89	116	76	96	70	134	86	87	74	104	77	84
58	Punniyamoorthi	70	128	77	98	74	120	84	96	75	120	76	76
59	Kavya	80	110	72	96	80	116	70	90	76	123	62	94
60	Vanaroja	87	136	84	97	58	108	68	91	60	114	72	97

S.no	Name	5 MIN				10 MIN				Sedation	adverse effect
		HR	SBP	DBP	MAP	HR	SBP	DBP	MAP		
1	Seetha	70	110	74	86	66	114	66	82	3	
2	Hari	75	126	74	88	65	120	64	74	3	
3	Pavitha	74	124	68	84	72	110	70	88	3	
4	Gokul	65	114	62	98	71	120	68	85	3	
5	Jeevitha	66	114	75	89	75	104	76	87	3	
6	Saravanan	80	113	66	82	68	120	77	82	3	
7	Saradha	75	125	70	84	66	123	76	90	3	
8	Mani	87	124	68	86	64	114	62	70	3	
9	Shiva	71	102	72	90	74	106	72	76	4	
10	Guru	76	124	60	89	65	112	66	85	3	
11	Kannan	65	118	76	84	75	124	64	71	3	
12	Savitha	60	102	62	76	61	106	68	76	3	
13	Moorthi	74	126	74	90	66	112	63	84	3	
14	Smitha	68	102	72	76	74	114	65	88	4	
15	Vadivu	73	105	74	88	64	113	66	86	3	
16	Murugesan	64	120	62	89	85	120	64	80	3	
17	Karthyikeyan	74	118	66	84	66	127	65	84	3	
18	Senthil	75	106	74	86	64	118	69	97	3	
19	Paunambal	86	104	68	81	74	125	78	85	3	
20	Jayalakshmi	71	124	76	85	75	114	74	80	3	
21	Kumari	68	112	74	97	62	108	62	84	3	
22	Nirmala	65	104	70	71	66	116	76	94	3	

S.no	Name	5 MIN				10 MIN				Sedation	adverse effect
		HR	SBP	DBP	MAP	HR	SBP	DBP	MAP		
23	Paniyan	72	118	84	90	62	102	66	86	3	
24	Muthu	68	124	76	88	64	108	64	68	4	
25	Venktachalam	64	116	74	88	62	128	64	88	3	
26	Chinna	82	102	78	84	62	120	69	84	3	
27	Subash	75	118	67	90	66	102	70	70	3	
28	Subramaniam	77	104	72	76	71	110	71	76	4	
29	Saraswathy	62	124	81	84	78	114	80	82	3	
30	Kabilan	61	103	68	88	78	106	66	88	3	
31	Swathi	66	104	76	85	66	102	62	90	3	
32	Haribaskar	65	120	77	87	68	114	60	94	3	
33	Prabavathy	72	123	76	82	64	110	76	95	4	Bradycardia
34	Lalitha	71	114	62	90	60	120	74	80	3	
35	Palani	75	106	72	70	74	104	66	70	4	
36	Sakthivel	68	112	66	76	64	120	60	75	3	
37	Sumathi	66	104	64	85	66	123	66	76	3	
38	Dharani	64	106	68	71	61	114	58	78	4	
39	Kumaran	74	112	63	76	70	106	56	82	3	
40	Kamalakannan	65	114	65	84	64	112	64	74	3	
41	Thirupathi	75	113	74	88	65	124	70	78	4	
42	Selvam	61	125	64	76	62	106	68	85	4	Bradycardia
43	Thirumurugan	66	127	65	80	64	112	76	81	3	
44	Selvi	74	123	69	84	70	114	64	82	3	
45	Veluchamy	64	125	68	97	32	113	76	76	4	

S.no	Name	5 MIN				10 MIN				Sedation	adverse effect
		HR	SBP	DBP	MAP	HR	SBP	DBP	MAP		
46	Chinnaiyan	85	104	74	85	78	120	62	70	3	
47	Murugan	66	108	62	86	64	127	72	76	3	
48	Tamil	64	116	76	84	65	118	66	85	3	
49	Thiruselvi	74	102	66	94	74	125	64	71	3	
50	Kangam	75	108	64	86	72	114	68	76	4	
51	Thirumal	62	116	64	68	68	108	63	84	3	
52	Vishvam	66	120	69	88	71	116	65	88	3	
53	Kamalam	62	102	70	84	61	102	66	86	3	
54	Parvathi	64	110	71	70	68	108	64	80	4	Bradycardia
55	Srimathi	62	108	80	76	72	116	65	84	3	
56	Karkuzhali	62	124	66	82	74	120	69	97	3	
57	Thirunavukarasu	66	105	62	88	75	102	78	85	3	
58	Punniyamoorthi	71	110	60	90	74	110	74	80	3	
59	Kavya	78	116	76	94	78	108	62	84	3	
60	Vanaroja	78	124	74	95	66	106	76	74	4	Bradycardia

CONSENT FORM

I _____ hereby give consent to participate in the study being conducted by **DR.U.GEETHA SOUNDARYA,** post graduate in department of Anaesthesiology, Thanjavur medical college & hospital, Thanjavur and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations

Place :
Date : Signature of participant

KEY TO ABBREVATIONS

ASA	American society of Anaesthesiologist
F(sex)	Female
M(sex)	Male
M(study group)	Magnesium sulphate
D(study group)	Dexmedetomidine
HR	Heart rate
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
MAP	Mean arterial blood pressure