"COMPARATIVE STUDY OF ETOMIDATE AND PROPOFOL FOR INDUCTION OF GENERAL ANAESTHESIA"

By

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Dissertation submitted to

BLDE UNIVERSITY, VIJAYAPUR, KARNATAKA.

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

ANAESTHESIOLOGY

Under the guidance of

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2017

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I hereby declare that this dissertation entitled **"COMPARATIVE STUDY OF ETOMIDATE AND PROPOFOL FOR INDUCTION OF GENERAL ANAESTHESIA"** is a bonafide and genuine research work carried out by me under the guidance of **DR. VIJAYKUMAR.T.K**., Professor Department of Anaesthesiology, Shri. B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

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ACKNOWLEDGEMENT

On completion of my post graduation journey and this scientific document, I would like to acknowledge the immense help received from my mentors in the department of anaesthesiology.

With privilege and respect I would like to express my gratitude and indebtedness to my Guide, **Dr. Vijaykumar.T.K** for his constant inspiration, extensive encouragement and loving support, which he rendered in pursuit of my post-graduate studies and in preparing this dissertation.

I would also like to express my gratitude and indebtedness to **Dr.D.G.TALIKOTI H.O.D** for his extensive encouragement in pursuit of my post-graduate studies and in preparing this dissertation.

I am forever grateful to Professor **Dr.Vidya Patil** for hes guidance and encouragement provided to me, to achieve new heights professionally over my course period.

I am grateful to Associate Profs. **Dr.R.R.Kusugal, Dr.Vijay Katti, Dr.Renuka Holyachi, Dr.Nirmala Devi, Dr.Sridevi Mulimani,** for their guidance, encouragement and inspiration.

I am thankful to **Dr. Shivanand K.L, Dr.Pratibha.S.D , Dr.Basavaraj.Patil , Dr. Sunil Kyadi, Dr.Sharath, Dr.Mala Nair, Dr. Santosh Karjagi, Dr.Ramesh Sajjanar, Dr.Lalita, Dr.Geetha** for their great help.

I am extremely thankful to **Dr. S. P. Guggarigoudar,** Principal of B.L.D.E.U'S Shri. B.M. Patil Medical College, Hospital and Research Centre, VIJAYAPUR, for permitting me to utilize resources in completion of my work.

I am thankful to the members of ethical committee of B.L.D.E.U'S Shri. B.M. Patil Medical College, Hospital and Research Centre, Vijayapur for permitting me to carry out this study.

I am thankful to **Mr. Shahnawaz** for his help in statistical analysis

My thanks to one and all staff of Library, Anaethseia Department and Hospital for their co-operation in my study.

I am thankful to my seniors **Dr.Vaibhav, Dr.Danish, Dr.Shishir** for their advice and support.

I am thankful to colleagues **Dr.Abhishek, Dr. Manjunath, Dr.Tom, Dr.Aman, Dr.Meghana, Dr.Yashaswani, Dr.Vaishnukumar, Dr.Vikas** for their advice, suggestions and co-operation in my journey.

I would also like to thank all my juniors for their help and cooperation during my study.

I am deeply indebted to my parents for their blessings, which helped me to complete this dissertation.

Last but not the least; I convey my heartfelt gratitude to all my patients, without whose co-operation, this study would be incomplete.

DR. SANTOSHKUMAR ALALAMATH

LIST OF ABBREVATIONS USED

ASA – American Society of Anaesthesiologists

Bpm – Beats per minute

DBP – Diastolic Blood Pressure

HR – Heart rate

Inj – Injection

iv – Intravenous

kg – Kilogram

MAP – Mean Arterial Pressure

mcg – Microgram.

mg – Milligram

min – Minute

ml – Millilitre

SBP – Systolic Blood Pressure

sec – Second

yrs – Years

ABSTRACT

An ideal induction agent for general anaesthesia should have hemodynamic stability, minimal respiratory side effects and rapid clearance. Sudden hypotension has a deleterious effects on maintaining the circulation to vital organs. Presently Etomidate and Propofol are popular rapid acting inducing agents.

MATERIALS AND METHODS:

Present randomized study was conducted on eighty patients after informed consent, comprising of forty patients each. Both received Fentanyl 2 microgm/kg and Glycopyrrolate 0.2 mg as premedication ten minutes before induction, followed by Etomidate 0.3 mg/kg given slowly over 45 seconds in the first group and Propofol 2.5 mg/kg for induction of anaesthesia in the second group.

RESULTS:

In this study the heart rate changes are significant between the groups. Maximum decrease in SBP, MAP and DBP is seen in group P compared to group E at 2-3 minutes of induction and 5minutes of post intubation. Group E is more hemodynamically stable compared to group P.

Pain on injection was more in group P, 11 patients had grade I, 6 patients grade II and 2 patients grade III pain on injection respectively, where as in group E 7 patients had grade I pain, 1 patient had grade II pain on injection.

Among forty patients in group E, 10 patients developed grade I myoclonus, grade II and grade III in 5 and 1 patients respectively. Among forty patients in group P, 3 patients developed grade I myoclonus.

The apnea occurred in 14 out of 40 patients in group E and 39 out of 40 patients in group P patients.

Among forty patients in group E, 17 patients had nausea post operatively as compared 8 patients in group P. Among forty patients in group E, 14 patients out of 40 had vomiting post operatively as compared 4 patients in group P.

CONCLUSION:

Patients induced with Propofol had significant decrease in systolic, diastolic blood pressure and mean arterial pressures at 10minutes after induction compared to Etomidate. This characteristic indicates that Etomidate caused hemodynamic stability. Heart rate changes were significant between the two groups. Incidence of apnea and pain on induction were more with Propofol group, however, Etomidate caused more of myoclonus than Propofol. Post operative Nausea and vomiting were more in Etomidate group compared to Propofol group.

So Etomidate is better inducing agent than Propofol with regard to cardiovascular stability.

Key Words : Etomidate, Propofol, Hemodynamic stability, Myoclonus

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INTRODUCTION

Inducing agents are drugs that are given intravenously in an appropriate dose causes rapid loss of consciousness. Induction agents are used to induce anaesthesia prior to other drugs being given to maintain anaesthesia, as the sole drug for short procedures, to mainatain anaesthesia for longer procedures by intravenous infusion, to provide conscious sedation during procedures undergoing in local anaesthesia and intensive care unit. An ideal induction agent for general anaesthesia should have hemodynamic stability, minimal respiratory side effects and rapid clearance and with minimal side effects drug interaction. Presently Etomidate and Propofol are rapid acting inducing agents.

Etomidate is a carboxylate imidazole containing compound characterized by hemodynamic stability, minimal respiratory depression and cerebral protective effects[1]. Its lack of effect on sympathetic nervous system, baroreceptor reflex regulatory system[1,2] and its effect on increased coronary perfusion even on patients with moderate cardiac dysfunction makes it an induction agent of choice. Propofol decreases blood pressure, cardiac output and systemic vascular resistance[3,4]due to inhibition of sympathetic vasoconstriction and impairment of baroreceptor reflex regulatory system[1,5]. This effect may be exaggerated in hypovolemic and elderly patients with compromised left ventricular function due to coronary artery disease. It produces dose dependent depression of ventilation. However the adverse effects such as pain on injection, thrombophlebitis and myoclonus for both the agents have been corrected by premedicating with the Fentanyl, an opioid[6].

This study is an attempt to compare the hemodynamic, respiratory and other effects of both the drugs so that we can choose a safe induction agent.

AIMS AND OBJECTIVES

To compare:

A) Primary Objectives

- Time of onset of induction between Etomidate and Propofol.

- To compare the hemodynamic effects of both drugs in relation to heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure.

B) Secondary Objectives

- To compare adverse effects such as pain on injection, myoclonus and post-operative nausea and vomiting.

ANATOMY AND PHYSIOLOGY[7,8]

HEART RATE

INTRODUCTION

Heart rate can vary during normal physiological condition such as exercise, emotion etc. however under physiological conditions, the altered heart rate is quickly brought to normal by various regulatory mechanisms.

REGULATION OF HEART RATE:

Heart rate is regulated by nervous mechanisms such as:

- Vasomotor center,
- Efferent nerve fibers to the heart,
- Afferent nerve fibers from the heart

VASOMOTOR CENTRE: It is situated in the reticular formation of medulla oblongata and the lower part of pons consisting of three areas, The Vasoconstrictor area, Vasodilator area and Sensory area

Vasoconstrictor area increases the heart rate by sending impulses through sympathetic nerves. This is under the control of hypothalamus and cerebral cortex.

Vasodilator area decreases the heart rate by sending impulses through vagus, which is under the control of hypothalamus. It is also controlled by baro receptors and chemo receptors.

Sensory area lies in the nucleus tractus solitaries in medulla and pons. This area receives sensory impulses via glossopharyngeal and vagus nerves.

Efferent nerve fibers are Parasympathetic arising from dorsal nucleus of vagus. They innervate the heart by cardiac branch of vagus, terminating in sinoatrial node (SAN) and atrioventricular node(AVN) causing decrease in heart rate.

Factors affecting vasomotor center: Impulses from higher center:

Cerebral cortex : area13 is concerned with emotional reactions of the body. It Causes cardiac acceleration with emotions.

Hypothalamus : stimulation of posterior and lateral hypothalamic nuclei causes tachycardia, where as stimulation of preoptic and anterior nuclei causes bradycardia.

Impulses from respiratory center:

In forced breathing heart rate increases during inspiration and decreases during expiration. This variation is called respiratory sinus arrhythmia.

Impulses from baroreceptors (marey'sreflex)

Baroreceptors respond to change in blood pressure, Carotid baroreceptors are situated in the carotid sinus, which is present in the internal carotid artery near bifurcation of common carotid artery. The aortic baroreceptors are situated in the arch of aorta.

Afferent from carotid sinus passes through herings branch of glossopharyngeal nerve, Afferent from aortic sinus passes through vagus nerve. The nerve fibers reach the nucleus of tractus solitaries situated adjacent to vasomotor center.

Marey's law: the heart rate is inversely proportional to blood pressure. The Baroreceptor produces the marey's reflex only during resting conditions.

Impulse from chemoreceptors:

Chemoreceptors respond to change in chemical constituents of blood, particularly Oxygen, carbon dioxide and hydrogen ion concentration. These area adjacent to baroreceptors.

ARTERIAL BLOOD PRESSURE:

Arterial blood pressure is defined as the lateral pressure exerted by the column of blood on the wall of arteries.

Mean arterial pressure is the average pressure existing in the arteries. It is diastolic pressure plus one third of pulse pressure. Since the diastolic period(0.53s) is longer than the systolic period (0.27s).

Normal mean arterial pressure is 93 mm Hg.

Regulation of arterial blood pressure:

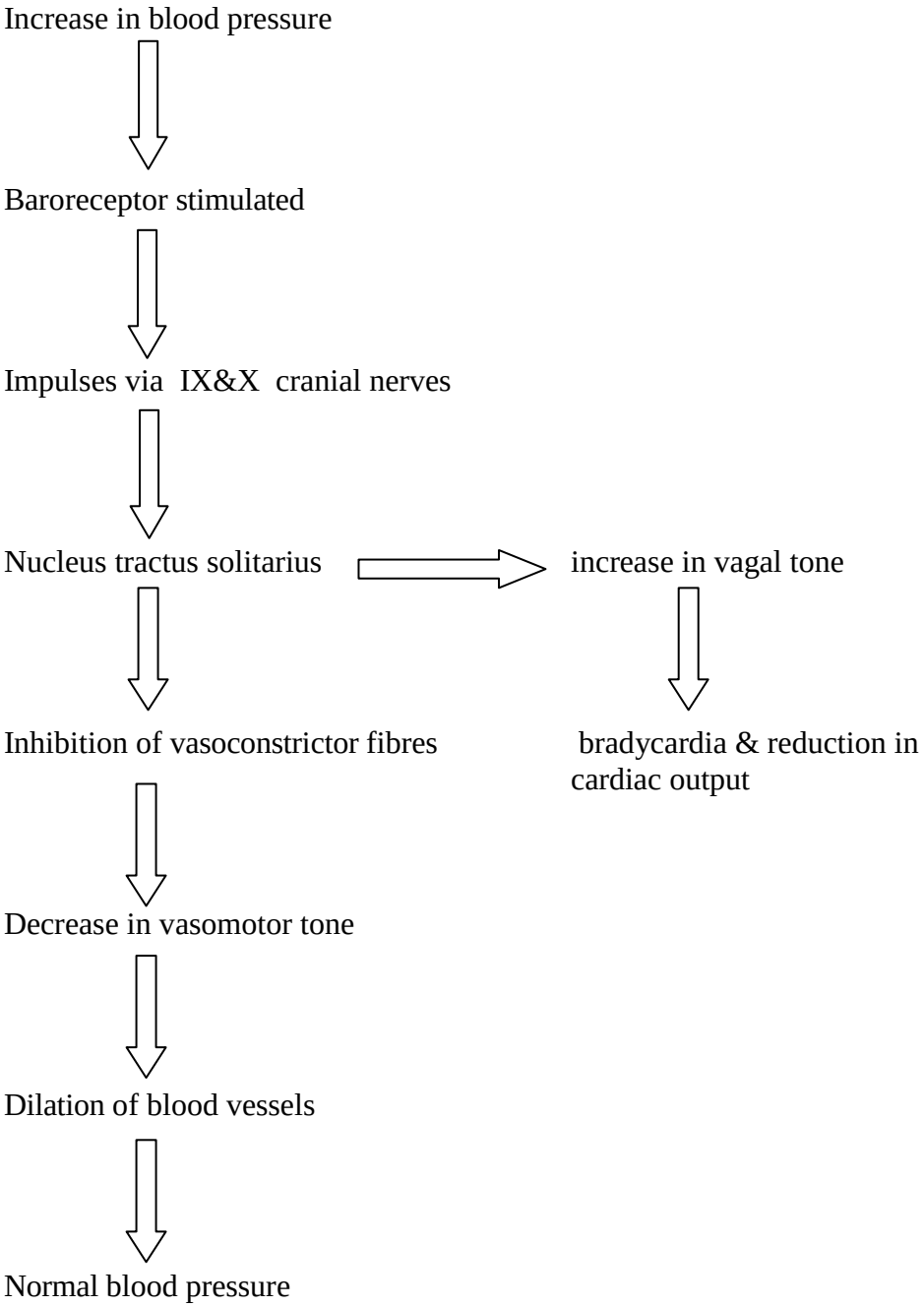
Regulatory mechanisms:

1. Nervous mechanism or short-term regulatory mechanism.
2. Renal mechanisms or long-term regulatory mechanism.
3. Hormonal mechanisms
4. Local mechanisms.

Nervous mechanism:

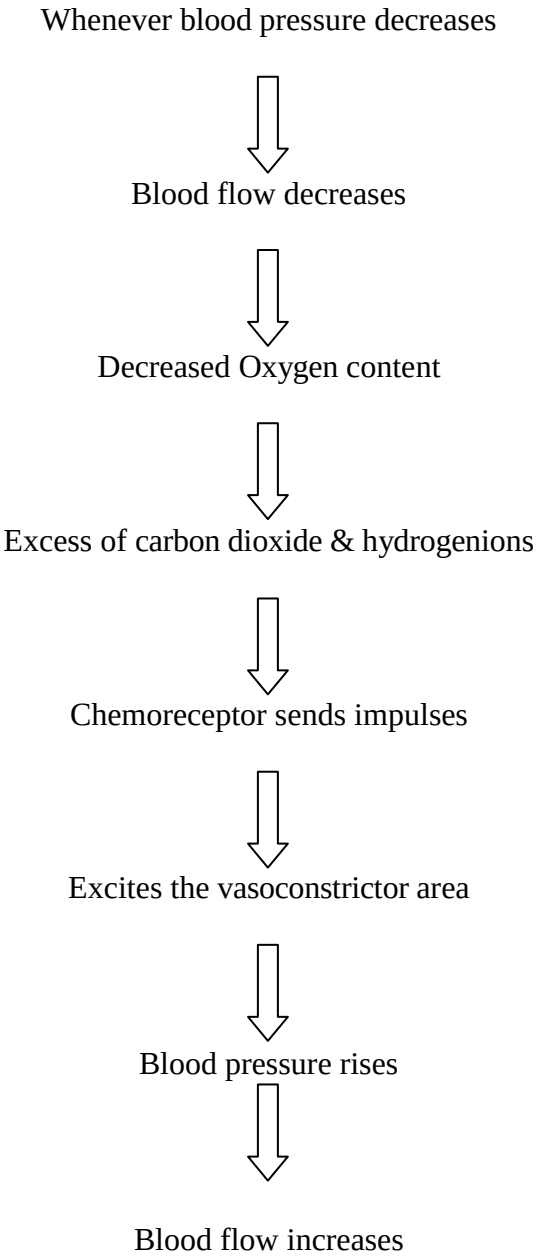
Baroreceptor mechanism:

Fig.1 Flowchart showing baroreceptor mechanism.



Chemoreceptor mechanism:

Fig.2 Flow chart showing chemoreceptor mechanism.



PHYSIOLOGY OF EMESIS[9,10]

Definitions

Nausea is defined as a subjectively unpleasant sensation associated with the awareness of the urge to vomit. It is usually felt in the back of the throat and epigastrium and is accompanied by the loss of gastric tone, duodenal contractions and reflux of intestinal contents into stomach.

Retching is defined as labored, spasmodic, contraction of respiratory muscles including the diaphragm, chest wall and abdominal wall muscles without expulsion of gastric contents, against a closed glottis.

Vomiting or emesis is the forceful expulsion of gastric contents from the mouth and is brought about by the powerful sustained contraction of the abdominal muscles, descent of the diaphragm and opening of gastric cardia.

Physiology

Nausea and vomiting are important defense mechanisms against ingestion of toxins. The act of emesis involves a sequence of events that can be divided into pre-ejection, ejection and post- ejection phase.

The pre-ejection phase comprises of prodromal symptoms of nauseas, along with autonomic signs such as salivation, swallowing, pallor, and tachycardia. The ejection phase comprises of retching and vomiting.

During retching the hiatal portion of the diaphragm doesnot relax, and intra-abdominal pressure increases are associated with decreases in intrathoracic pressure. In contrast relaxation of hiatal portion of the diaphragm permits a transfer of intra-abdominal pressure to the thorax during act of vomiting. Contraction of the rectus abdominals and external oblique muscles of anterior abdominal wall, relaxation of esophageal sphincter, an increase in intrathoracic and intra-gastric pressure, reverse

peristalsis, and an open glottis and mouth results in the expulsion of gastric contents.

The post ejection phase consists of autonomic and visceral responses that return the body to a quiescent phase or without residual nausea.

The complex act of vomiting involves coordination of respiratory, gastrointestinal and abdominal musculature and is controlled by the emetic center. The evidence of such a center is based on electrical stimulation and on brain stem lesion studies.

Anatomical studies how that the parvicellular reticular formation has access to the motor pathways responsible for the visceral and somatic output involved in vomiting. This area is situated in the lateral reticular formation close to the tractus solitarius in the brainstem and is thought to be the emetic center.

Electrical stimulation of the tractus solitaries will cause immediate vomiting. Ablation of the emetic center abolishes the vomiting response to direct chemical stimulation. However, at present there are no known anesthetic drugs or chemicals that act directly on the emetic center.

Stimuli from several areas within CNS can affect the emetic center this include afferents from the pharynx, gastrointestinal tract and mediastinum, as well as afferents from the higher cortical centers (including the visual center and the vestibular portions of the 7^{th}cranial nerve) and the Chemoreceptor trigger zone(CTZ) in the area postrema.

The area postrema is highly vascularized and the vessels terminate in fenestrated capillaries surrounded by large perivascular spaces. No effective blood brain barrier exists in these areas and thus the CTZ can be activated by chemical

stimuli received through the blood as well as the cerebrospinal fluid, direct electrical stimulation of the CTZ does not result in emesis.

In addition to the CTZ, chemosensory in puts to the emetic centers can come from the forebrain (example, pilocarpinestimulation) and the nodosa ganglion of the vagus nerve.

The area postrema of the brain stem is within dopamine, Opioid and serotonin are 5HT3 receptors. The nucleus tractus solitatius is rich in encephalins and histaminic and muscarinic cholinergic receptors. These receptors may play an important role in the transmission of impulses to the emetic center. It has been speculated that block of these receptors is an important mechanism of action of the currently used antiemetic drugs.

A specific anatomical site for a possible anti-emetic center has not been described. Although opioid receptors have been identified in the brain stem, their role in the emesis is uncertain.

The concept of chemosensory activation of the CTZ by a parallel array of independent receptor sites has recently been questioned and a sequential activation model which linkages between effector nuclei has been suggested. In this model, the control of emesis does not depend on the existences of a discrete group of neurons in an emetics center but is the expression of a local circuit involving sequential stimulation of separate effector nuclei. This model attempts to explain why some of the phenomena usually associated with emesis can occur without the actual expulsion of gastric contains. However the discovery of the parvi cellular reticular formation and it effect on emesis provides support for the parallel array model.

PHARMACOLOGY

Propofol

Propofol chemically 2,6–diisoPropofol, one of the group of alkyl phenols. These are oils at room temperature, are insoluble in water and highly lipid soluble.

Formulation:

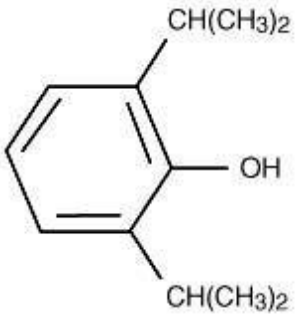
1. Older formulation with cremophor associated with anaphylactic reactions.
2. 1% Propofol,

   10% soyabean oil 2.25% glycerol

   1.2% purified egg phosphatide

   0.005% disodium edentate as retardant of bacterial growth.
3. Propofol with medium chain fatty acids.[12]

Structure:



Metabolism:

Propofol is rapidly metabolized in liver by conjugation with glucuronide and sulphate to produce water soluble compounds, which are excreted in the kidney. The metabolites of Propofol are inactive. Extra hepatic metabolism for Propofol is in kidneys and lungs.[13]

Pharmacokinetics:

- Initial distribution half-life of Propofol is 2-8 minutes[14]
- Elimination half-life is 4-23hours.
- Volume of distribution in central compartment is 20-40seconds.
- Clearance of Propofol is 1.5– 2.2 litre/min.[15]
- Time of peak effect is 90-100seconds.
- Pharmacokinetics of Propofol is altered by various factors like gender, weight, preexisting diseases, age and concomitant medication.[14,16,17]

Effects on respiratory system:

Propofol causes apnea after induction, dose and speed of injection decides the incidence and duration of apnea. Incidence is 25 to 30%. It leads to initial decrease in tidal volume and increase in respiratory rate then apnea.[18]

Maintenance infusion causes decrease in tidal volume and increase in respiratory rate. Ventilatory response to hypercarbia or hypoxia is decreased during infusion. Arterial carbon dioxide concentration increases Propofol induces bronchodilation in patients with chronic obstructive airway disease.[19]

Effects on cardiovascular system:

It produces 25-40% reduction in systolic blood pressure, 0-40% reduction in mean arterial blood pressure and diastolic pressure is also reduced. Cardiac output and cardiac index are reduced by 15%, stroke volume index ± 20%.[20,21].

Vasodilation by Propofol is due to the following factors:[11]

- Reduction of sympathetic activity
- Direct effect on intracellular calcium mobilization
- Inhibition of prostaglandin synthesis in endothelial cells.

- Reduction in angiotensin II mediated calcium entry.
- Stimulation of nitric oxide
- Activation of potassium ATP channels.

Heart rate does not change after an induction of Propofol, it reset or inhibit the baroreceptor reflex mechanism reducing tachycardia response to hypotension.

Propofol causes decrease in myocardial blood flow and myocardial Oxygen consumption. So that the global myocardial Oxygen supply to demand ratio is preserved.[20,22]

Propofol also possesses significant antiemetic activity at low (sub-hypnotic)doses[23,24] This effect can be achieved by a 10 to20 mg loading dose followed by infusion at 10μg/kg/min. Propofol used as a maintenance anesthetic during breast surgery was more effective than 4mg of Ondansetron given as prophylaxis in preventing postoperative nausea and vomiting.

Uses and doses of Propofol[11]

- Induction of general anaesthesia : 1–2.5 mg/kg IV.
- Maintenance of general anaesthesia : 50–150μg/kg/min IV combined with N2O or an opiate.
- Sedation : 25–75μg/kg/min IV.
- Antiemetic : 10–20mgIV; can be repeated every 5-10 min or start infusion of 10μg/kg/min..

Side Effects and Contraindications

Pain on injection is less than or equal to that with Etomidate.[25,26,27,28,29] Pain on injection is reduced by:

- Using a large vein
- Avoiding veins in the dorsum of the hand
- Adding Lidocaine to the Propofol solution.
- Adding medium chain fatty acids.
- Pretreatment with an Opioid, NSAIDS, Ketamine also reduces the pain on injection.

Myoclonus occur but less frequently than after Etomidate[25]

Apnea after induction with Propofol is common. The incidence of apnea is greater when compared to Etomidate.[30]

Hypotension the most significant side effect on induction is the decrease in systemic blood pressure.

Propofol infusion syndrome is a rare, but lethal syndrome associated with infusion of Propofol at 5mg/kg/hr. or greater for 48hours or longer.

ETOMIDATE

History

Etomidate (Amidate, Hypnomidate) was synthesized in 1964 and was introduced into clinical practice.

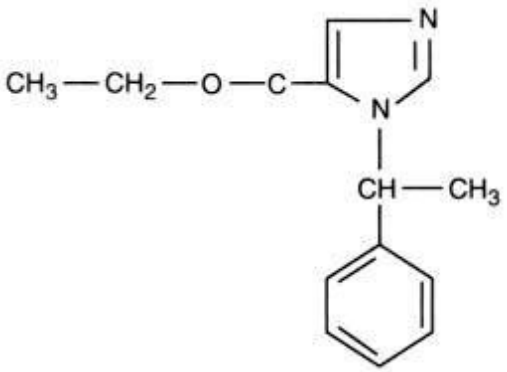
Advantages:

- Hemodynamic stability,
- Minimal respiratory depression,
- Cerebral protection, and
- Pharmacokinetics enabling rapid recovery after either a single dose or a continuous infusion.

Disadvantages:

- Reports of temporary inhibition of steroid synthesis after both single doses and infusions.
- Pain on injection
- Myoclonus
- Nausea and vomiting

Structure:



Physicochemical Characteristics

Etomidate is an imidazole derivative (R-(+)-pentylethyl-1H-imidazole-5carboxylatesulfate). Its molecular weight is 342.36kd. Etomidate is water insoluble and is unstable in a neutral solution[31].

Solvents: 2mg/mL propylene glycol (35%byvolume) solution with a pH of 6.9 lipid emulsion to reduce some of the side effects of Etomidate[25].

Metabolism:

Etomidate is metabolized in the liver by

- Ester hydrolysis primarily
- N-dealkylation.

The main metabolite is inactive. Only 2% of the drug is excreted unchanged, the rest being excreted as metabolites by the kidney (85%) and in bile (13%).[32,33]

Induction and Maintenance of Anaesthesia

- Induction of general anaesthesia-0.2–0.6 mg/kg IV.[34,35]
- Maintenance of general anaesthesia-10μg/kg/min IV with N2 O and an opiate
- Sedation-5–10μg/kg/min IV.

Prolonged periods of sedation are contraindicated because of inhibition of corticosteroid synthesis.

The duration of anaesthesia after a single induction dose is linearly related to the dose each 0.1mg/kg administered provides about 100seconds of loss of consciousness. Repeat doses of Etomidate, either by bolus or infusion, prolong the duration of hypnosis. Recovery after multiple doses or an infusion of Etomidate is still usually rapid.

Pharmacokinetics

The kinetics of Etomidate is best described by an open three-compartment model. The drug has an initial distribution half-life of 2.7minutes, are distribution half-life of 29minutes, and an elimination half-life that varies from 2.9 to 5.3hours. Clearance of Etomidate by the liver is high (18to25 mL/kg/min).[32,33]

Etomidate is 75% protein bound. In patients with cirrhosis, the volume of distribution is doubled, but clearance is normal; the result is an elimination half-life that is twice normal.

Pharmacology

Effects on the Central Nervous System

The primary action of Etomidate on the CNS is hypnosis, which is achieved in one arm-brain circulation after a normal induction dose (0.3mg/kg). Etomidate has no analgesic activity. Etomidate binds to β2 and β3 subunits of $GABA_A$ and causes GABA potentiation.

Etomidate reduces CBF (by34%) and CMRO2 (by45%) without altering mean arterial pressure. Thus, cerebral perfusion pressure is maintained or increased, and there is a beneficial net increase in the cerebral Oxygen supply-demand ratio. When given in doses sufficient to produce EEG burst suppression, Etomidate acutely lowers ICP by upto 50% in patients with already increased ICP.[36,37]

Etomidate produces initial increase in alpha-wave amplitude with sharp beta bursts followed by mixed delta-theta waves. Features of Increased EEG activity in epileptogenic foci and associated grandmal seizures have been proved useful for intraoperative mapping of seizure foci before surgical ablation. Etomidate is also

associated with a high incidence of myoclonic movement. The myoclonic movement is believed to result from activity either in the brain stem or in deep cerebral structures.

Effects on the Respiratory System

It has minimal effect on ventilation and does not induce histamine release in either healthy patients or those with reactive airway disease. The ventilator response to carbon dioxide is depressed. Induction with Etomidate produces a brief period of hyperventilation, sometimes followed by a similarly brief period of apnea. Incidence of apnea is altered by premedication. Hiccups or coughing may accompany Etomidate induction.

Effects on the Cardiovascular System

An induction dose of 0.3mg/kg of Etomidate given to cardiac patients for non-cardiac surgery results in almost no change in heart rate, mean arterial pressure, mean pulmonary artery pressure, pulmonary capillary wedge pressure, central venous pressure, stroke volume, cardiac index, and pulmonary and systemic vascular resistance[38,39,40,41] A large dose of Etomidate, 0.45mg/kg also produces minimal changes in cardiovascular parameters. Etomidate produces a 50% decrease in myocardial blood flow and Oxygen consumption and a 20% to 30% increase in coronary sinus blood Oxygen saturation. The myocardial Oxygen supply demand ratio is thus well maintained. It lacks analgesic effect, may not totally ablate the sympathetic response to laryngoscopy and intubation.

Endocrine Effects[42]

Etomidate causes dose dependent reversible inhibition of the enzyme 11β-hydroxylase, which converts 11-deoxy cortisol to cortisol, These effects result in an increase in the cortisol precursors 11-deoxycortisol and 17-hydroxy progesterone, which inhibits cytochrome P450. This results in inhibition of ascorbic acid resynthesis, which is required for steroid production in humans. This minor adrenocortical suppression effects were shown to follow even single bolus doses.

Side Effects:[43,44,45,46]

- Nausea and vomiting(30-40%).[47]
- Pain on injection and superficial thrombophlebitis[48,49,50].
- Myoclonus and hiccups(0-70%): myoclonus can be reduced by premedicating with a narcotics or 0.015mg/kg of Midazolam 90 seconds before induction[51,52,53]
- Propylene glycol can be associated with some degree of hemolysis.[54]

REVIEW OF LITERATURE

R.Carlos *et al* (1979) conducted a study on 74 patients and incidence of myoclonus was compared between patients received Etomidate 0.3 mg/kg alone, Etomidate premedicated with Fentanyl 10mcg/kg and Diazepam 150mcg/kg with Atropine 10mcg/kg before Etomidate. They observed myoclonus in 14 of 26 patients received Etomidate alone, 6 of 25 patients receiving Etomidate with Fentanyl and 5 of 23 pateints receiving Etomidate with Diazepam, They concluded that incidence of myoclonus reduces on premedication.[6]

A.W.Doenicke *et al* (1999) compared the pain on injection between Etomidate in propylene glycol and Etomidate-lipuro with medium chain fatty acids. Nine out often patients reported moderate to severe pain on injection where as one of ten patients had pain. Hence they came out with conclusion that Etomidate-lipuro causes less pain on injection.[49]

Y.Nyman *et al* (2006) studied 110 pediatric patients aged 2-16 years for incidence of injection pain on induction using four point scale. A significantly lower incidence of injection pain was found in the Etomidate-lipuro group as compared to Propofol-Lidocaine group.[50]

John M *et al* (1979) in their study cardiovascular and pulmonary responses following Etomidate induction of anaesthesia in 22 patients with cardiac disease under ASA III &IV .[38]

Thomas Brussel *et al* (1989*)* conducted a study to compare the hemodynamic effects on induction with Propofol 2.5mg/kg and Etomidate 0.3mg/kg in eight dogs using left

ventricular catheter. Propofol was associated with significant decrease in systolic (19.5%)and diastolic (25.3%) arterial pressures and 17.3% decrease in cardiac output, 11.6% reduction in systemic vascular resistance , without change in PCWP.[55]

J.S.C.Mccollem and J.W.Dundee (1986) studied the induction characteristics of four induction agents Thiopentone, Etomidte, Propofol and Methohexitone . Propofol caused significantly more hypotension and pain on injection compared to others.[56]

A.Crido *et al* (1980) studied hemodynamic characteristics of 36 patients on induction with Etomidate there was a reduction in cardiac output, stroke volume and arterial pressure and compensatory increase in heart rate.[40]

A.Gauss, H.Heinrichand, O.H.G Wilder-Smith (1991) performed echocardiographic assessment of hemodynamic effects of Propofol with Etomidate and Thiopentone in 30 ASA I patients, systolic blood pressure and end systolic quotient decreased in Propofol and Thiopentone group. Diastolic blood pressure and end diastolic diameter did not change in any of the groups and the Etomidate group showed no changes in the hemodynamic variables.[41]

In a double blind randomized study, M.St pierre (2000) studied the incidence and severity of post-operative nausea and vomiting was investigated with Etomidate and Propofol. They concluded that Etomidate does not increase nausea during early post-operative period.[47]

Lars Huter *et al* (2007) conducted study on 80 patients using low dose Midazolam for reducing the incidence of myoclonus by Etomidate. In 40 patients who received Midazolam along with Etomidate 2 patients developed myoclonus whereas 10 patients developed myoclonus in Etomidate group. So he concluded that Midazolam 0.015mg/kg as premedication is effective in reducing myoclonus.[51]

Thomas J Ebert (1992) compared sympathetic response to induction of anaesthesia with Propofol and Etomidate on 25 patients. Systolic blood pressure and diastolic blood pressures were well maintained in Etomidate but were decreased after Propofol administration and found there was significant decrease in forearm vascular resistance by Propofol than by Etomidate.[2]

John M. Gooding *et al* (1977) conducted a study on 11 patients of ASA I-III to know the effects of Etomidate on cardiovascular system. They observed only 10 % increase in heart rate, no statistically significant change in other parameters like systolic blood pressure, diastolic blood pressure, pulmonary arterial pressure, pulmonary capillary wedge pressure, central venous pressure, stroke volume, cardiac index, systemic vascular resistance, pulmonary vascular resistance.[39]

Giese JL, Stockham RJ, Stanley TH, *et al* (1985) noted the hemodynamic changes and side effects of anaesthesia induction with Etomidate or Thiopental in 83 ASA class I or 11 patients. Patients were randomly assigned to one of 12 groups according to pretreatment drug (Fentanyl 100 mcg, or normal saline intravenously), induction agent (Etomidate 0.4 mg/kg, or Thiopental 4 mg/kg), and maintenance anesthetic technique (Isoflurane-Oxygen, Isoflurane-Nitrous oxide-Oxygen, or Fentanyl-Nitrousoxide Oxygen). There were significant increases in heart rate in all groups, especially after tracheal intubation. These increases were attenuated but not eliminated by Fentanyl pretreatment. Systolic arterial blood pressure increased significantly after intubation and was not affected either by Fentanyl pretreatment or by the induction agent. Patients in whom anaesthesia was induced with Etomidate had a greater incidence of pain on injection and myoclonus and a lesser incidence of apnea than patients in whom anaesthesia was induced with Thiopental. Fentanyl

pretreatment significantly decreased the incidence of pain on injection and myoclonus, but it increased the incidence of apnea when anaesthesia was induced with Etomidate.[44]

Schaeuble *et al* (2005) did a double study to compare Etomidate(0.2mg/kg) and Propofol(2mg/kg) for fibre optic naso tracheal intubation as a part of an airway management algorithm and they concluded that Etomidate is better agent because spontaneous breathing recovers faster than with Propofol.[57]

Molly Sarkar *et al* (2005) studied hemodynamic responses to Etomidate(0.3mg/kg) on induction of anaesthesia in 12 pediatric patients undergoing cardiac catheterization and they concluded that Etomidate is safe in children as it does not produces clinically significant hemodynamic changes.[58]

Tae Kwan Kim, Ik Seong Park (2011) did a comparative study of brain protection effects between Thiopental and Etomidate using bispectral index(BIS) during temporary arterial occlusion in 41 patients, general anaesthesia was induced and maintained with 1.5-2.5% sevofluranne and 50%N2O. The pharmacological burst suppression was induced by bolus injection of Thiopental 5mg/kg or Etomidate 0.3mg/kg, hemodynamic variables, onset time of burst suppression, numerical values of BIS were recorded at every minute and they concluded that Thiopental and Etomidate have same duration and similar magnitude of burst suppression with conventional doses and Etomidate is safer substitute for Thiopental in aneurysm surgery.[59]

J.Morel *et al* (2011) studied hemodynamic consequences and vasopressor requirements of Etomidate(0.3mg) and Propofol(0.5mg) administration in two groups

of 50 patients each undergoing elective cardiac surgery and corticotrophin test was performed 12, 24 and 48 hours after anaesthesia induction and they found that the incidence of relative adrenal insufficiency (RAI) was higher in Etomidate group at 12hours and 24hours and they concluded that single bolus of Etomidate blunts the hypothalamic-pituitary-adrenal axis response for more than 24hr in patients undergoing elective cardiac surgery, but this was not associated with an increase in vasopressor requirements.[60]

Marina kalogridaki *et al* (2011) did a comparative study with Propofol and Etomidate combined with Fentanyl for external electric cardioversion in 46 patients with persistent atrial fibrillation, one group received Fentanyl 50mcg and Propofol 0.5mg/kg nad another group received 50mcg Fentanyl and 0.1mg/kg Etomidate while breathing spontaneously 100% Oxygen and cardioversion was achieved with extracardiac biphasic electrical shock ranging from 200-300J, performed three times atmost. They concluded that both anaesthetic regimens provided excellent condition for cardioversion in addition Etomidate with Fentanyl had shorter induction time and ensured hemodynamic stability.[61]

Saricaoglu *et al* (2011) were conducted study to compare Etomidate-lipuro and Propofol and 50%, (1:1) admixture of these agents at induction with special reference to injection pain, hemodynamic changes, and myoclonus. The hemodynamic (systolic, diastolic and mean blood pressures, heart rate) changes were minimal in group PE than other two groups (*P = 0.017). The intensity* of myoclonus was more in the group E (76.3%). Myoclonus was not observed in group PE and group P. There were no injection pain in group PE as the incidence were (83.8%) in group P and in (63.2%) group E. They concluded that 1:1 admixture of Etomidate-lipuro and Propofol is a valuable agent for induction.[62]

Anil K. Pandey *et al* (2012) were conducted a study to compare the effects of Etomidate and Propofol induction on hemodynamic and endocrine response in patients undergoing coronary artery bypass graft surgery on cardiopulmonary bypass(CPB) on hundred American Society of Anesthesiologists (ASA) grade II or III patients. They concluded that the surge in serum cortisol levels on the initiation of CPB seen after the use of Propofol is prevented by the use of Etomidate. Serum cortisol levels in both groups are well above the baseline at twenty-four hours without any untoward effects. Etomidate provides more stable hemodynamic parameters when used for induction of anaesthesia as compared to Propofol.[63]

Mehrdad Masoudifar and Elham Beheshtian (2013) were conducted a comparative study to know the cardiovascular response to laryngoscopy and tracheal intubation after induction of anaesthesia in 50 patients ASA I and II undergoing elective orthopedic surgeries and their cardiovascular response including systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate and O2 saturation were measured before laryngoscopy, during induction with Etomidate(0.3mg/kg) and Propofol (2.5mg/kg) and at 1, 3, 5, 10min after the induction. They found that changes in systolic, diastolic and mean arterial blood pressure were more in Propofol group and there were no significant difference among both group in terms of heart rate and O2 saturation. They concluded that Etomidate have more stable hemodynamic condition and preferred over Propofol for general anaesthesia.[64]

Kahlon A.Singh, Gupta Ruchi, Aujla K.Singh, Bindra T.Kaur (2014) did a study to know the efficacy of Lignocaine versus Midazolam in controlling Etomidate induced myoclonus on 75 ASA grade I and II patients undergoing elective surgeries under general anaesthesia, observed for myoclonus which was assessed by a four point

scale. They found that incidence of myoclonus was 76% in control group, 28% in midazolam group and 44% in lignocaine group and concluded that both midazolam and lignocaine were effective in reducing the incidence and severity of myoclonus.[65]

Mohammadreza safavi *et al* (2014) did a study to comapare the Magnesium Sulphate and Lignocaine pretreatment for prevention of pain on Etomidate induction on 135 patients undergoing elective surgeries under general anaesthesia and pain on injection was assessed by a four point scale. They found that 60% patients in control group had pain on injection and 22.2% and 40% in Lignocaine and Magnesium Sulphate group respectively and concluded that both the drugs are comparably effective in reducing Etomidate induced pain.[66]

Shagun Bhatia Shah *et al* (2015) did a study to comapare the hemodynamic effects of intravenous Etomidate and Propofol during induction and intubation using entropy guided hypnosis levels on 60 ASA I and II patients undergoing modified radical mastectomy. They measures heart rate, systolic, diastolic and mean arterial blood pressure and response entropy, state entropy at baseline induction and upto three minutes post intubation. They found that Etomidate provided hemodynamic stability without the requirement of any rescue drug in 96.6% patients whereas rescue drug ephedrine was required in 36.6% in Propofol group.[67]

Ram Prasad Kaushal, Ajay Vatal, Radhika Pathak (2015) did a study to comapare the effect of Etomidate(0.2mg/kg) and Propofol (2mg/kg) induction on hemodynamic and endocrine response in 60 ASA II and III patients undergoing elective coronary aretery bypass grafting (CABG)/mitral valve and aortic valve replacement(MVR/AVR) surgery on cardiopulmonary bypass(CPB). Hemodynamic variable like heart rate, systolic, diastolic and mean arterial blood pressure, cardiac output, central venous

pressure were measured at baseline, induction, immediately after intubation and after 5min of intubation. Cortisol and blood sugars were measured at baseline, during cardiopulmonary bypass, after bypass and at 24hour and concluded that Etomidate provides more stable hemodynamic pararmeters as compared to Propofol and can therefore be safely used for induction in patients with good LV function for CABG/MVR/AVR on CPB without cortisol suppression.[68]

Pushkar M. Desai, Deepa Kane, Manjula S. Sarkar (2015) did a single blinded study to compare Etomidate and Propofol as sedative during cardioversion on sixty ASA I/II/III patients undergoing elective cardioversion. They concluded that Etomidate/Fentanyl is superior over Propofol/Fentanyl during cardioversion for quick recovery and hemodynamic stability.[69]

Supriya Aggarwal *et al* (2016) conducted study to compare Propofol and Etomidate for their effect on hemodynamics and various adverse effects like myoclonus, pain on injection and apnea on patients in general anaesthesia in 100 ASA I and II of aged between 18-60 years. Patients in Etomidate(0.3mg/kg) group showed little change in mean arterial pressure (MAP) and heart rate (HR) compared to Propofol(2mg/kg) ($p > 0.05$) from baseline value pain on injection was more in Propofol group while myoclonus activity was higher in Etomidate group and the episodes of apnea were transient and not associated with ant fall Oxygen saturation. They concluded that Etomidate is a better agent for induction than Propofol in view of hemodynamic stability and less pain on injection[70].

Kavita Meena, et al., (2016) conducted a randomized control trial to compare the effect of Propofol, Etomidate and Propofol plus Etomidate induction on hemodynamic response to endotracheal intubation. The primary objective of this

study was to compare the efficacy of three different anaesthesia induction approach (Inj. Propofol, Inj. Etomidate and Inj. Propofol plus Inj Etomidate) in maintaining hemodynamic stability during induction and following endotracheal intubation on 90 patients aged 15 to 60 years of either sex and ASA physical status I or II scheduled for elective surgery under general anaesthesia. Group I induced with Inj.Propofol (2.5 mg/kg) intravenous, Group II with Inj. Etomidate (0.3 mg/kg) intravenous and Group III with Inj.Propofol (1 mg/kg) plus Inj. Etomidate (0.2 mg/kg) intravenous. They concluded that the combination of Etomidate plus Propofol has better hemodynamic stability than Etomidate alone at 1 min after intubation, though Etomidate was equally stable at other points of time. The combination proved to be significantly better than either Propofol or Etomidate alone.[71]

MATERIALS AND METHODS

This randomized study was done from December 2014 to June 2016 on patients who were admitted to BLDE UNIVERSITY Shri. B. M. Patil Medical College Hospital and Research Centre, Vijayapur and posted for elective surgeries requiring general anaesthesia. The study has been conducted after obtaining clearance from ethical committee of the institution. Informed consent was taken from all the patients who participated within the study.

80 patients were selected based on inclusion criteria and were randomly divided into two groups by computer generated randomized numbers.

Group E: Induction with Etomidate 0.3mg/kg (n=40)

Group P: Induction with Propofol 2.5mg/kg (n=40)

INCLUSION CRITERIA:

- Patients between the age group of 18 and 60 years.
- American society of anaesthesiologist grade I and II.
- Undergoing elective surgery under general anaesthesia.

EXCLUSION CRITERIA:

- Emergency surgeries.
- Patients allergic to any drugs.
- History of seizure disorder.
- Presence of known primary or secondary adrenal insufficiency or on steroid medication.
- Presence of hypotension

PROCEDURE :

Preanaesthetic evaluation and counseling for surgery was done on the previous day of surgery and reviewed on the day of surgery. A detailed medical history had been taken and systemic examination was carried out and relevant investigations were advised. Patients were informed about known effects and side effects of study drugs. On arrival to operation theatre

- IV line secured
- Monitors for electrocardiogram, Non invasive blood pressure, Pulse oximeter and ETCO2 were connected
- Oxygen delivered via face mask 6 litre/min
- The patients recording like heart rate, systolic, diastolic and mean arterial blood pressure were taken one minute before premedication (baseline) and every minute for first three minutes after induction and post intubation 3,5 and 10minutes

Parameters monitored:

- Blood pressure: systolic, diastolic and mean arterial pressure.
- Heart rate
- Respiratory rate
- Oxygen saturation
- Myoclonus. If present graded as mild, moderate or severe.
- Pain on injection by four point scale
- Post operative Nausea and vomiting.
- Drug intervention done.

Patients were premedicated with inj.Ondensetron 0.1mg/kg, inj.Glycopyrrolate 0.2 mg and inj.Fentanyl 2mg/kg[6,57] IV ten minutes before induction and the patients were randomized into two groups group E and group P for patients receiving Etomidate(0.3mg/kg) and Propofol(2.5mg/kg) respectively. Induction of anaesthesia was either with Propofol 2.5 mg/kg or Etomidate 0.3 mg /kg[6], loss of eye lash reflexes was considered to be the end point.

This was followed by inj. Vecuronium 0.1mg/kg, ventilation was assisted manually using bain circuit with 66% N2O inO2 and Isoflurane.

Observation was made for presence of **myoclonus** and graded as

Mild – Short movement of body segment (a finger or shoulder).

Moderate -Slight movement of two different muscles or muscle groups of the body.

Severe-Intense clonic movements in two or more muscle groups of the body (fast abduction of a limb).

Pain on injection graded as:

Grade 0 - No pain

Grade 1 - Verbal complain of pain

Grade 2 - Withdrawal of arm

Grade 3 – Both Verbal complain of pain and Withdrawal of arm

Three minutes after the administration of muscle relaxant intubation was attempted. After intubation was confirmed the patient was connected to bain circuit and intermittent positive airway pressure ventilation was continued until the completion of surgery with 66% N2O in O2 supplemented with Isoflurane and intravenous Vecuronium 0.08-0.1mg/kg iv.

At the end of the surgery neuromuscular blockade was reversed by using intravenous neostigmine 0.05 mg/kg and glycopyrrolate 10 mcg/kg. The extubation was performed after the patient was fully awake. The patient was monitored for 24 hours in the postoperative period for nausea and vomiting.

Figure 3: Photograph showing instruments used.

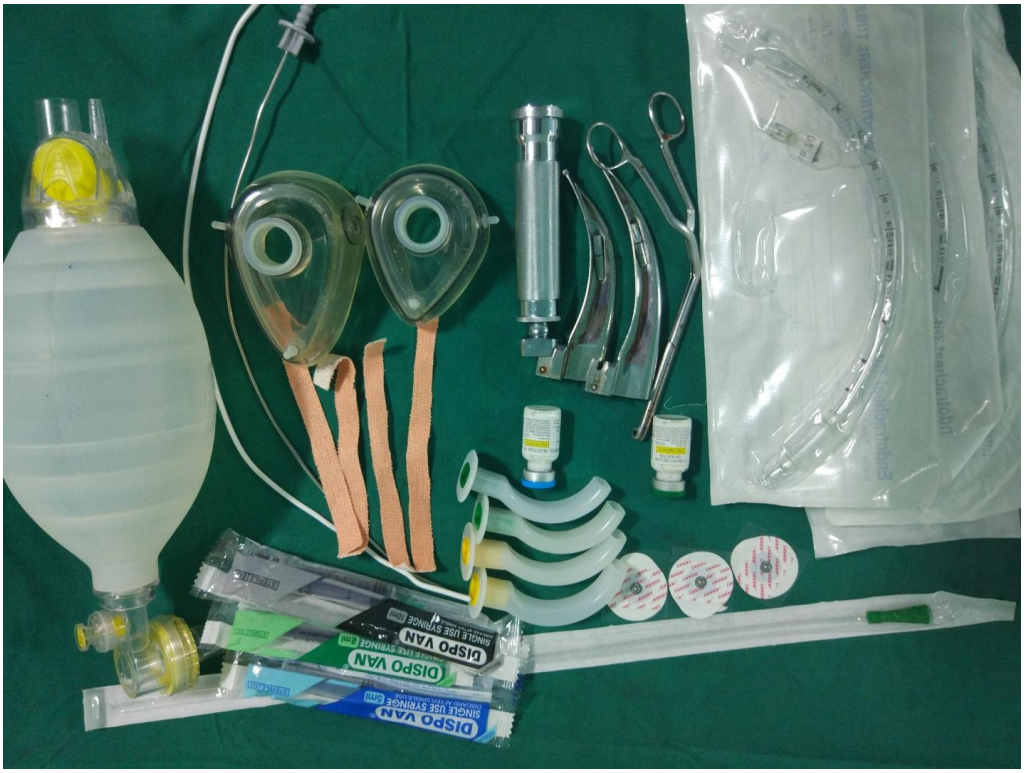
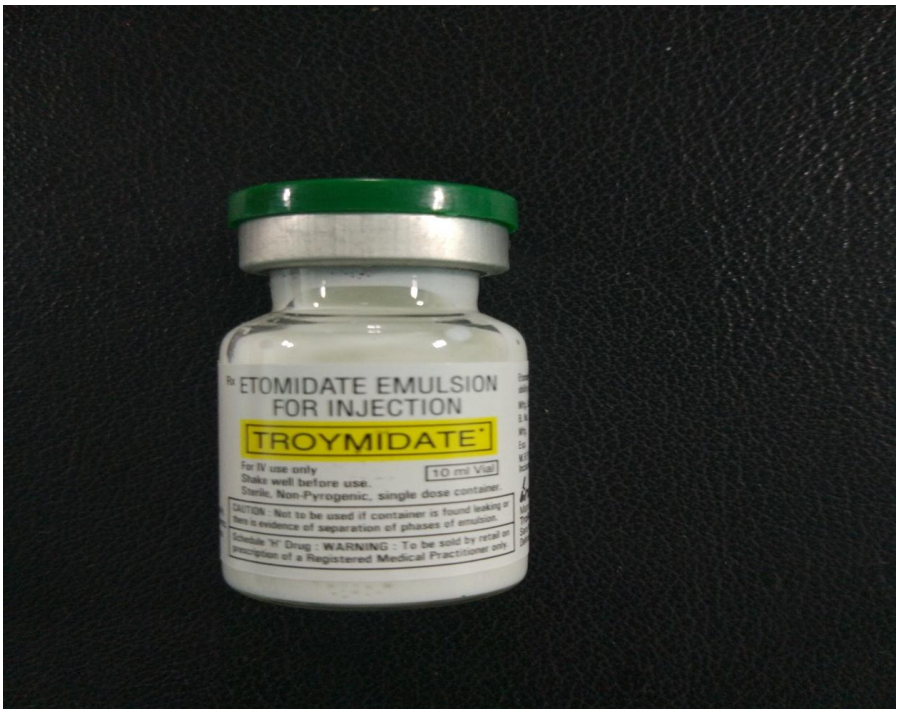
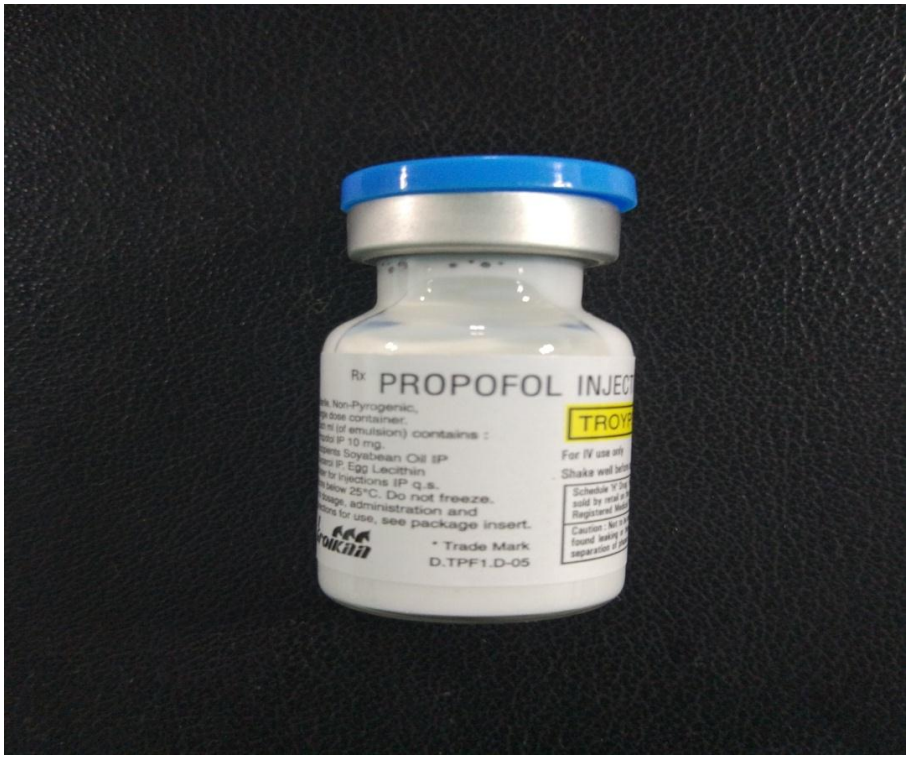


Figure 4: Photographs showing drugs used.





OBSERVATION AND RESULTS

Statistical analysis

All characteristics were summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries. Chi-square (χ^2)/Fisher exact test was employed to determine the significance of differences between groups for categorical data. The difference of the means of analysis variables was tested with the unpaired t-test. If the p-value was < 0.05, then the results were considered to be significant. Data were analyzed using SPSS software v.23.0.

Table 1: Age distribution between the study groups

Age groups (Yrs)	Group E		Group P		p value
	N	Percent	N	Percent	
18-30	20	50	11	27.5	
31-40	12	30	15	37.5	
41-50	5	12.5	11	27.5	0.158
>50	3	7.5	3	7.5	
Total	40	100	40	100	

Figure 5: Age distribution between the study groups

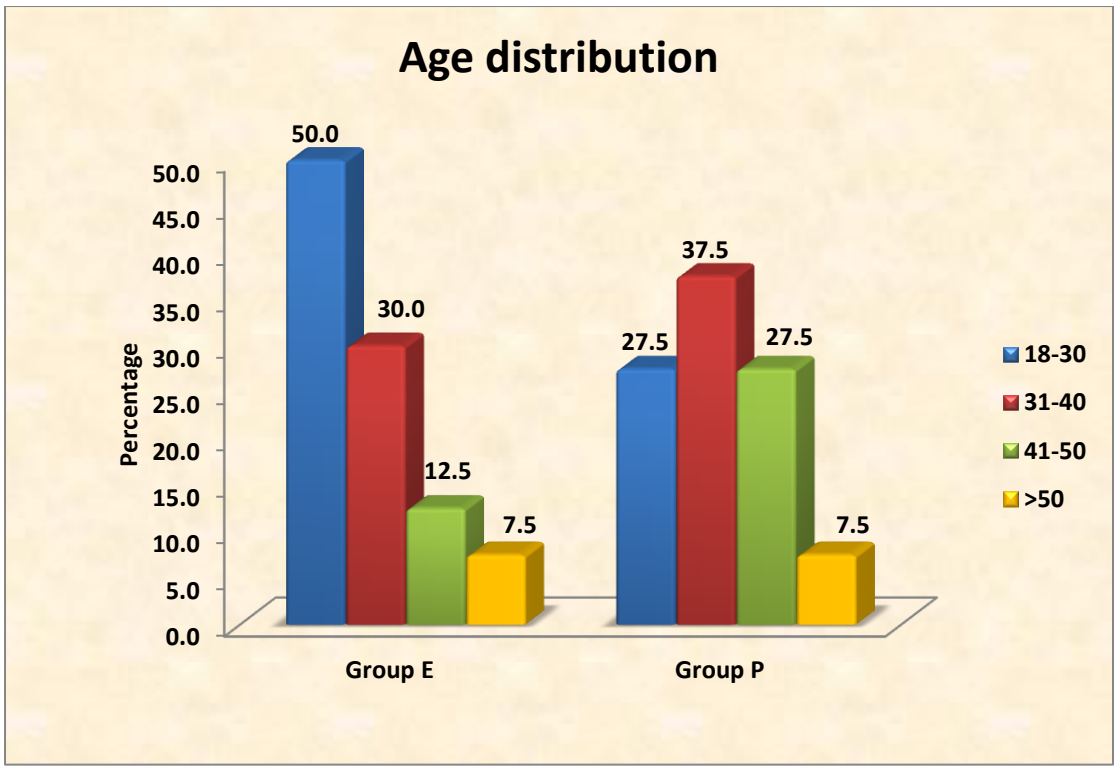


Table 1 shows the age distribution among the study population

In group E majority of the patients were in the age group 18-30

In group P majority of the patients were in the age group 31-40

There was no significant difference in age distribution between the groups

Table 2: Mean Age between the study groups

AGE (yrs)	Group E				Group P				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
	18	56	33.7	10.9	20	55	37.0	10.3	0.161

Figure 6: Mean Age between the study groups

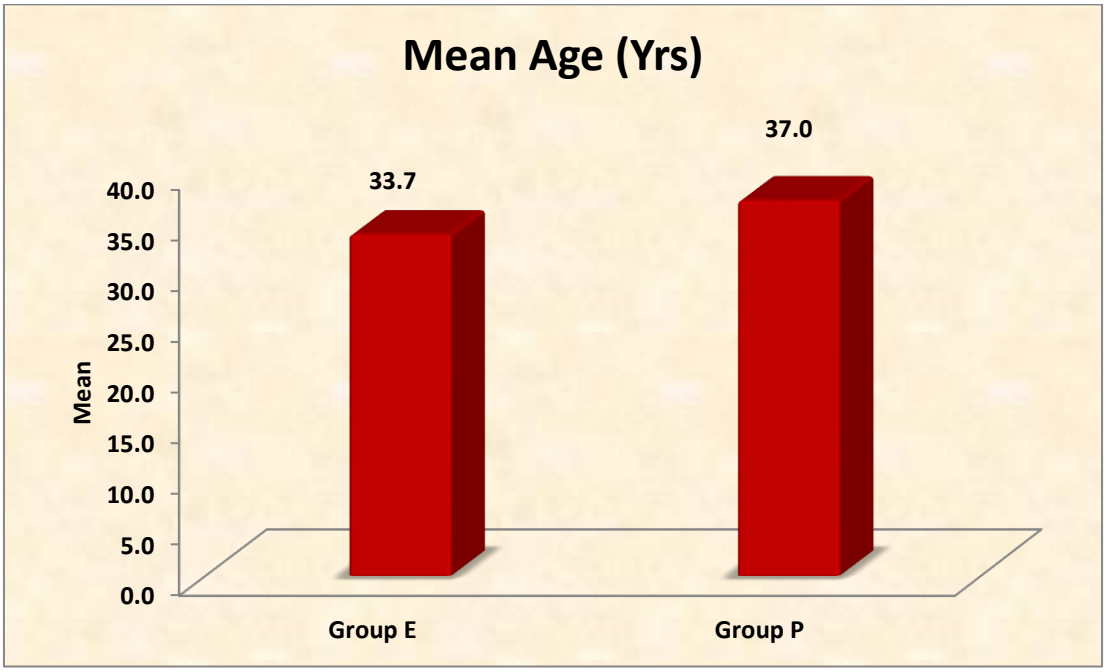


Table 2 shows mean age between the study groups in which

Mean age for group E is 33.7

Mean age for group P is 37

Table 3: Sex distribution between the study groups

Sex	Group E		Group P		p value
	N	Percent	N	Percent	
Male	12	30	20	50	0.068
Female	28	70	20	50	
Total	40	100	40	100	

Figure 7: Sex distribution between the study groups

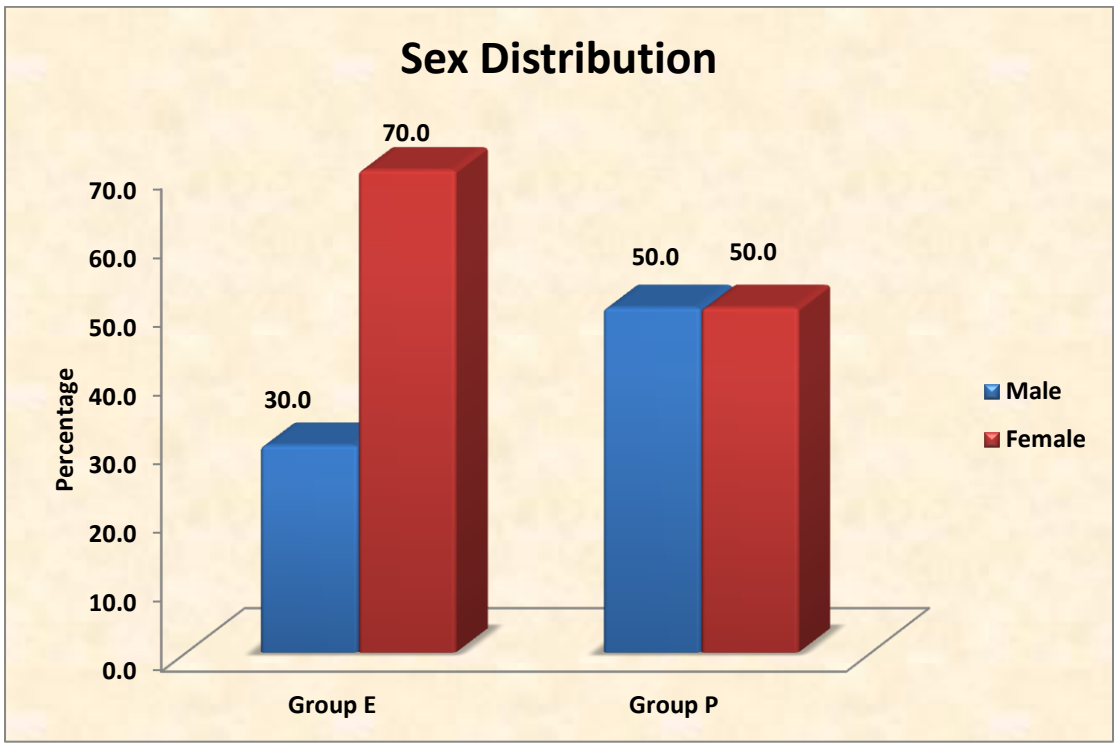


Table 3 shows sex distribution among the study population, where among group E females are more than males and males and female are equal in group P.

There was no significant difference in sex distribution between the groups

Table 4: Weight distribution between the study groups

Weight	Group E		Group P		p value
	N	Percent	N	Percent	
35-44	3	7.5	7	17.5	0.534
45-54	19	47.5	15	37.5	
55-64	11	27.5	12	30.0	
>64	7	17.5	6	15.0	
Total	40	100.0	40	100.0	

Figure 8: Weight distribution between the study groups

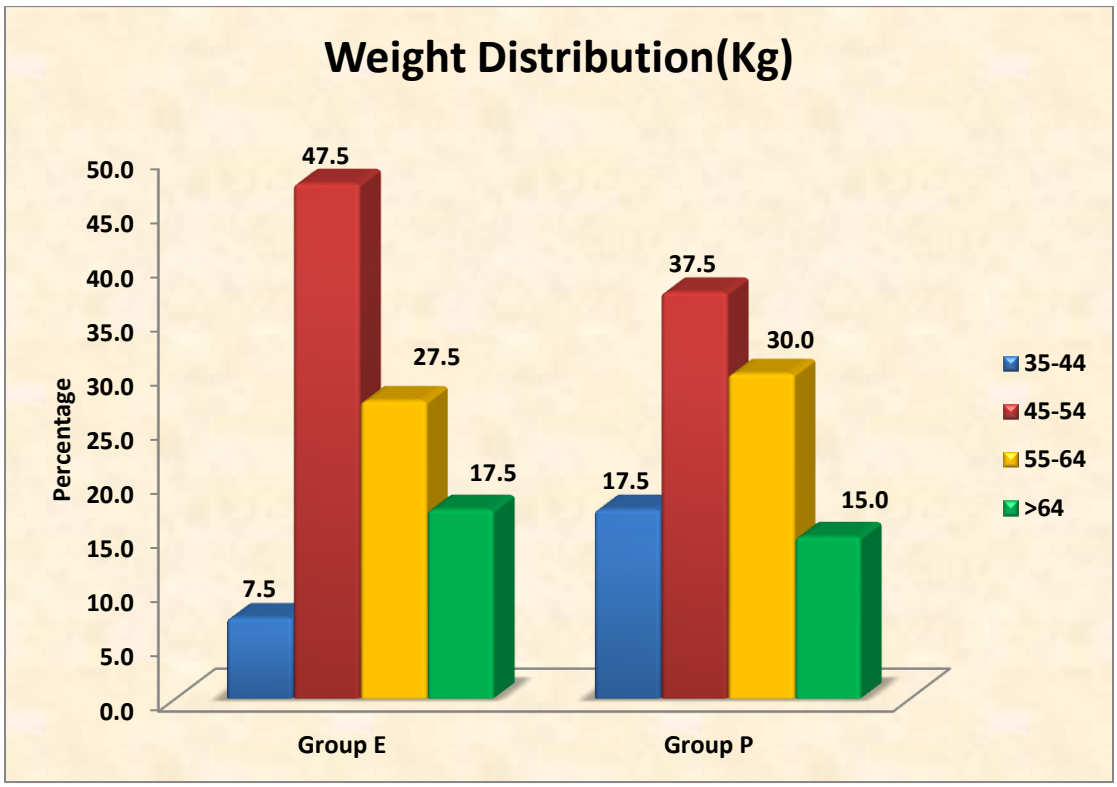


Table 4 shows the weight distribution among the study population, where in both Groups [Group E majority (47.5%) and Group P(37.5%)] of patients had their weight in range og 45-54kg.

There was no significant difference in weight distribution between the groups

Table 5: Mean Weight between the study groups

Weight	Group E				Group P				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
	35	80	54.8	10.6	38	71	53.6	8.8	0.567

Figure 9: Mean Weight between the study groups

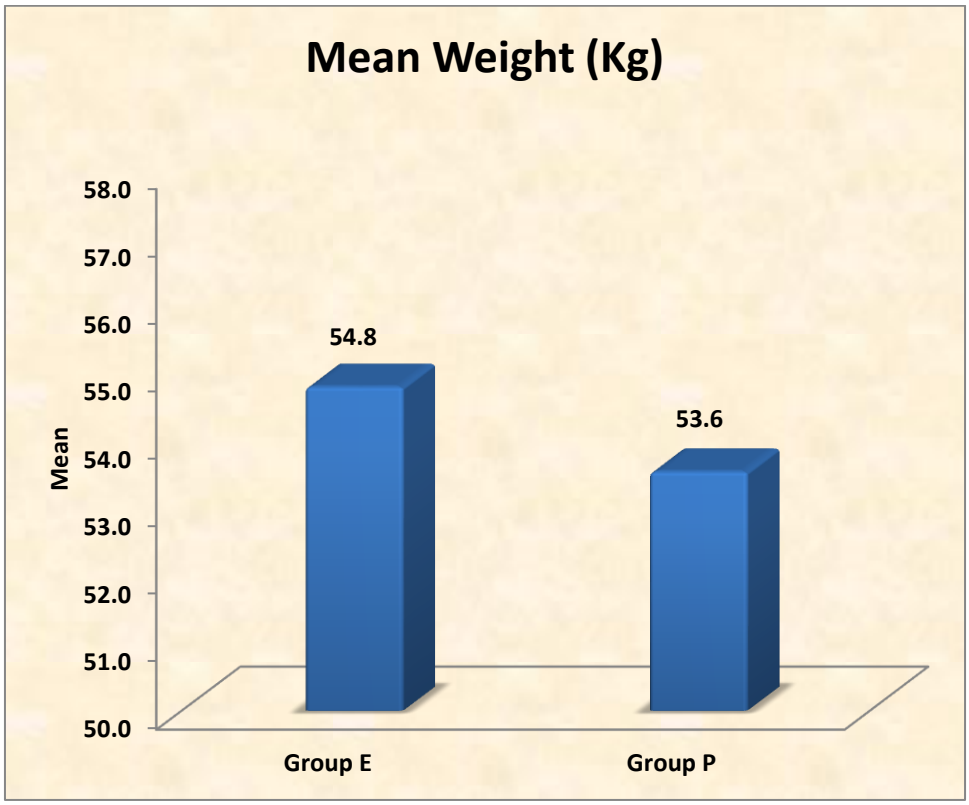


Table 5 shows mean weight distribution among study population where in mean weight for group E was 54.8 and for group P 53.6.

Table 6: Mean Heart rate between the study groups after intubation

HR	Group E (mmHg)				Group P (mmHg)				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
BASAL	68	120	89.9	13.2	66	112	90.8	12.0	0.757
Post intubations									
3MIN	74	118	92.1	10.0	61	102	78.1	9.0	0.000*
5MIN	68	110	89.7	10.2	72	116	94.8	8.1	0.014*
10MIN	75	105	88.0	7.9	73	97	85.5	7.1	0.132

*significantly different at 5% level of significance

Figure 10: Mean Heart rate between the study groups

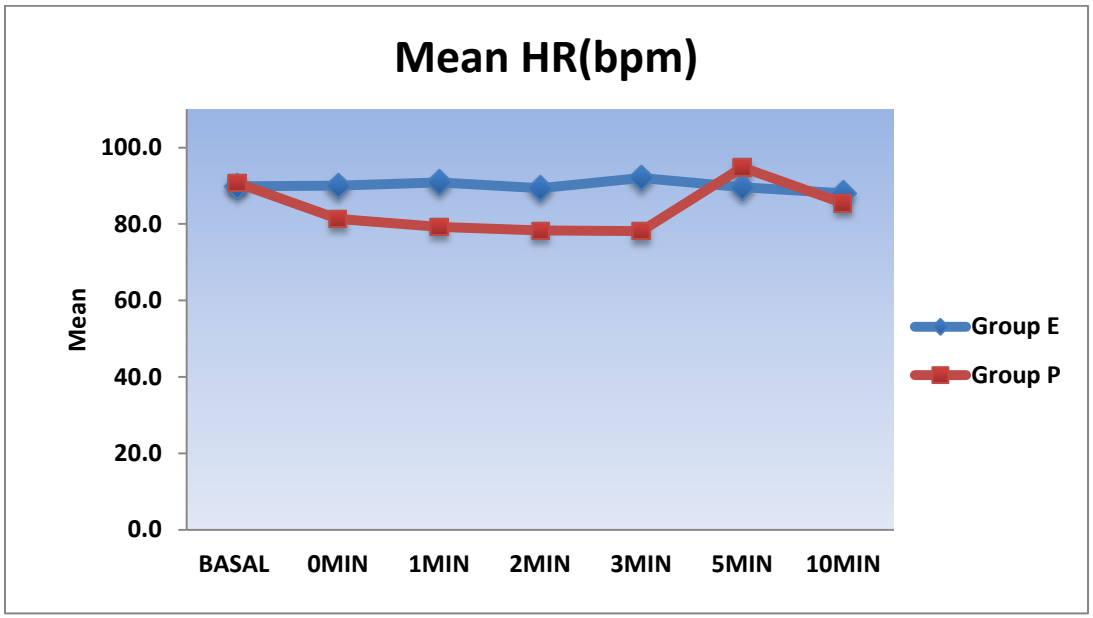


Table 4 shows shows changes in mean heart rate, in above table 3rd minute is the time of intubation and 5th minute is considered as second minute and 10min as 5th minute after intubation.

It shows that Group E patients the basal MHR in beats in beats per minute was 89.8 followed by 92.1 at intubation and 89.7 at 2min and 88 in 5min

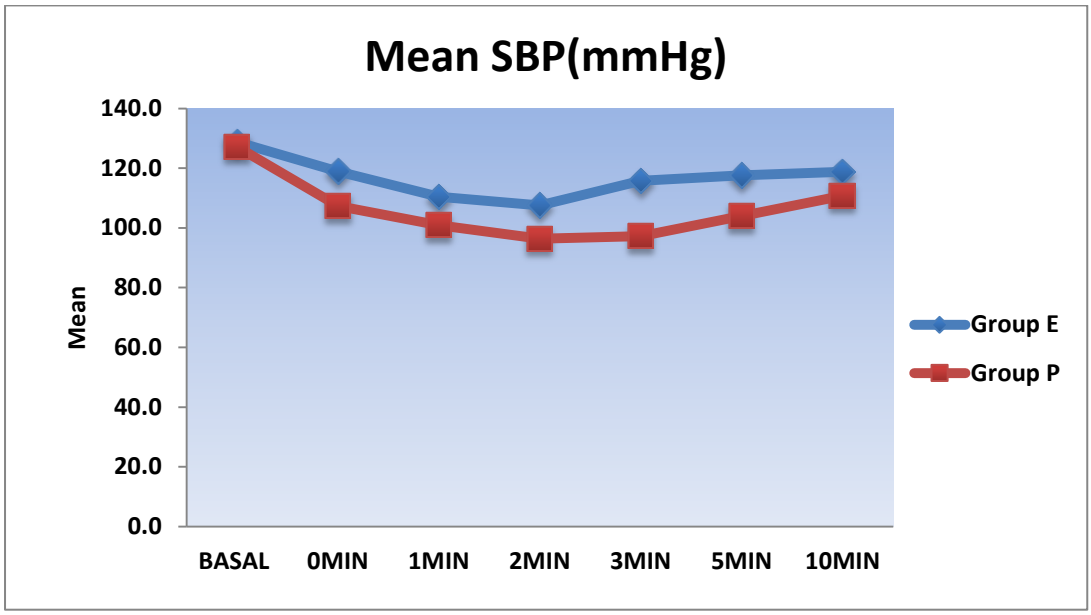
Among group P the basal MHR in beats per minute was 90.8, followed 78.1 at intubation and 94.8 at 2 min and 84.5 at 5min.

Table 7: Mean Systolic Blood Pressure (SBP) between the study groups after intubation

SBP	Group E (mmHg)				Group P (mmHg)				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
BASAL	107	148	128.8	10.6	107	149	127.1	10.5	0.479
Post intubations									
3MIN	93	135	115.8	10.4	85	111	97.2	6.4	<0.001*
5MIN	100	145	117.6	9.9	93	116	104.0	5.4	<0.001*
10MIN	102	148	118.8	8.4	100	126	110.8	5.3	<0.001*

*significantly different at 5% level of significance

Figure 11: Mean Systolic Blood Pressure (SBP) between the study groups



In group E the basal value of SBP was 128.8mmHg, 2min following intubation the SBP decreased to 117.6mmHg and 118.8mmHg at 5min.

In group P the basal value of SBP was 127.1mmHg, 2min following intubation the SBP decreased to 104mmHg and 110.8mmHg at 5min.

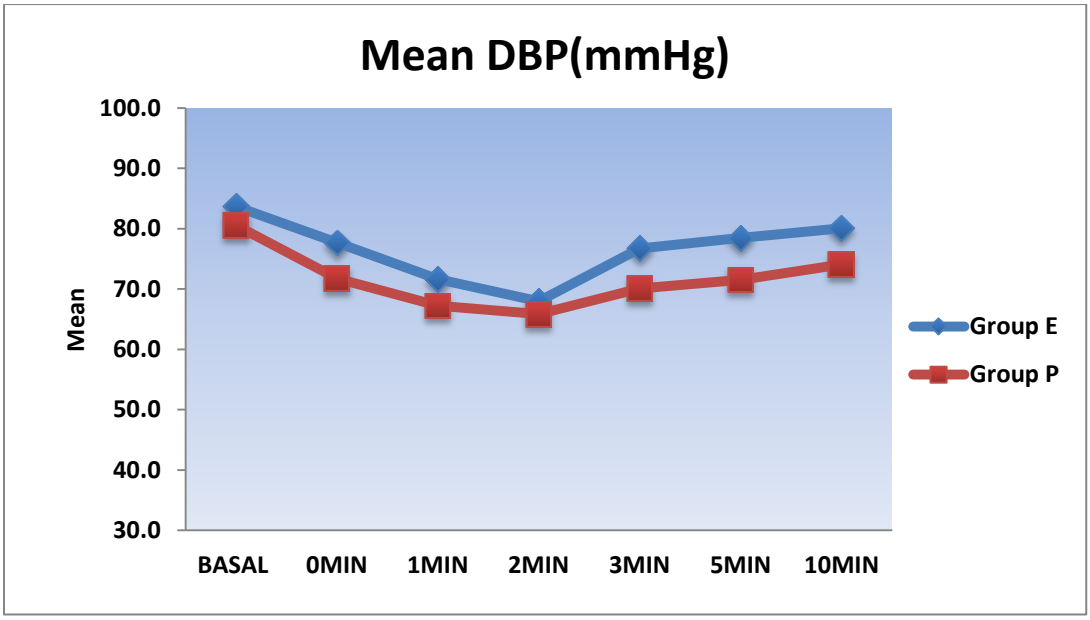
The decrease in SBP in group P was statistically significant compared to decrease in SBP in group E at 2min ($p<0.001$) and remained significant even upto 5min post intubation.

Table 8: Mean Diastolic Blood Pressure (DBP) between the study groups after intubation

DBP	Group E (mmHg)				Group P (mmHg)				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
BASAL	70	108	83.7	8.5	56	99	80.6	9.1	0.112
Post intubations									
3MIN	63	95	76.8	10.0	58	91	70.1	7.7	<0.001*
5MIN	56	99	78.5	10.5	52	89	71.5	8.0	<0.001*
10MIN	61	98	80.1	8.6	60	96	74.1	7.2	<0.001*

*significantly different at 5% level of significance

Figure 12: Mean Diastolic Blood Pressure (DBP) between the study groups



In group E the basal value of DBP was 83.7mmHg, 2min following intubation the DBP decreased to 78.5mmHg and 80.1mmHg at 5min.

In group P the basal value of DBP was 80.6mmHg, 2min following intubation the SBP decreased to 71.5mmHg and 74.1mmHg at min.

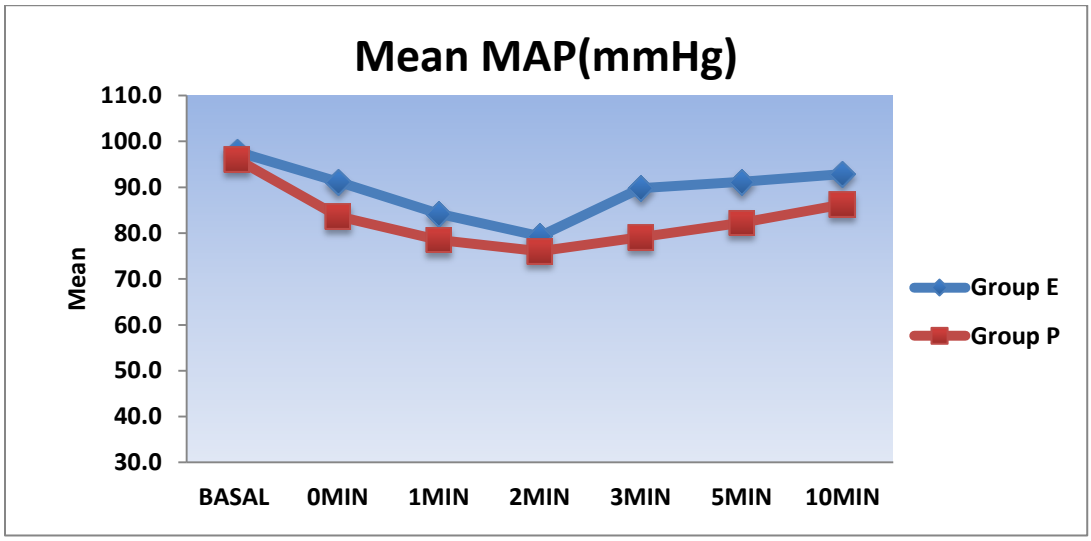
Statistical evaluation between the groups showed that the decrease in DBP observed in both groups was statistically significant ($p<0.001$) at intubation post intubation 2min and 5min.

Table 9: Mean Arterial Pressure (MAP) between the study groups after intubation

MAP	Group E (mmHg)				Group P (mmHg)				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
BASAL	84	114	97.8	7.4	74	116	96.1	8.8	0.361
Post intubations									
3MIN	69	107	89.8	9.8	67	95	79.2	6.3	<0.001*
5MIN	72	110	91.2	9.4	67	98	82.3	6.3	<0.001*
10MIN	79	110	92.9	7.8	77	106	86.3	6.0	<0.001*

*significantly different at 5% level of significance

Figure 13: Mean Arterial Pressure (MAP) between the study groups



In group E the basal value of MAP was 97.8mmHg, 2min following intubation the MAP decreased to 91.2mmHg and 92.9mmHg at 5min.

In group P the basal value of MAP was 96.1mmHg, 2min following intubation the MAP decreased to 82.3mmHg and 86.3mmHg at 5min.

Statistical evaluation between the groups showed that the decrease in MAP observed in both groups was statistically significant ($p<0.001$) at intubation post intubation 2min and 5min.

Table 10: Mean Heart rate between the study groups after induction

HR	Group E (bpm)				Group P (bpm)				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
BASAL HR	68	120	89.9	13.2	66	112	90.8	12.0	0.757
INDUCTION	65	120	90.1	11.5	62	99	81.4	9.1	0.000*
1MIN	70	117	90.9	10.7	61	96	79.2	8.7	0.000*
2MIN	69	116	89.4	11.7	60	91	78.4	8.1	0.000*

*significantly different at 5% level of significance

The change in mean heart rate between the group E and group P during first and second minute immediately after induction were statistically significant (p<0.000).

Table 11: Mean Systolic Blood Pressure (SBP) between the study groups after induction

SBP	Group E (mmHg)				Group (mmHg) P				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
BASAL	107	148	128.8	10.6	107	149	127.1	10.5	0.479
INDUCTION	98	143	118.9	10.0	90	121	107.4	6.9	<0.001*
1MIN	94	137	110.5	10.7	90	110	100.9	5.4	<0.001*
2MIN	91	136	107.5	10.6	84	109	96.4	5.5	<0.001*

*significantly different at 5% level of significance

In group E basal mean SBP was 128.8mmHg. One and two minutes of induction it was 110.5mmHg and 107.5mmHg respectively. It shows a fall in SBP of 21.3mmHg

In group P basal mean SBP was 127.1mmHg. one and two minutes of induction it was 100.9mmHg and 96.4mmHg respectively. It shows a fall in SBP of 30.7mmHg

This shows a more decrease in SBP in group P when compared to group E. The change in mean SBP between the group during first and second minute immediately after induction were statistically significant (p<0.001).

Table 12: Mean Diastolic Blood Pressure (DBP) between the study groups after induction

DBP	Group E (mmHg)				Group P (mmHg)				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
BASAL	70	108	83.7	8.5	56	99	80.6	9.1	0.112
INDUCTION	58	95	77.7	10.1	55	93	71.7	8.6	0.006*
1MIN	54	94	71.7	10.0	45	88	67.3	9.6	0.047*
2MIN	50	85	68.0	8.9	51	79	65.9	8.7	0.291

*significantly different at 5% level of significance

In group E basal DBP was 83.7mmHg. one and two minutes of induction it was 71.7mmHg and 68mmHg respectively. It shows a fall in DBP of 15.7mmHg

In group P basal DBP was 80.6mmHg. one and two minutes of induction it was 67.3mmHg and 65.9mmHg respectively. It shows a fall in DBP of 14.7mmHg

This shows decrease in DBP in group P when compared to group E. The change in mean DBP between the group at induction($p<0.006$) and during first minute immediately after induction were statistically significant ($p<0.047$).

Table 13: Mean Arterial Pressure (MAP) between the study groups after induction

MAP	Group E (mmHg)				Group P (mmHg)				p value
	Min	Max	Mean	SD	Min	Max	Mean	SD	
BASAL HR	84	114	97.8	7.4	74	116	96.1	8.8	0.361
INDUCTION	71	120	91.2	11.6	68	101	83.7	7.5	0.001*
1MIN	65	109	84.3	11.3	60	93	78.5	7.5	0.009*
2MIN	64	98	79.3	9.1	63	87	76.1	7.0	0.080

*significantly different at 5% level of significance

In group E basal MAP was 97.8mmHg. one and two minutes of induction it was 84.3mmHg and 79.3mmHg respectively. It shows a fall in DBP of 18.5mmHg

In group P basal MAP was 96.1mmHg. one and two minutes of induction it was 78.5mmHg and 76.1mmHg respectively. It shows a fall in DBP of 20mmHg

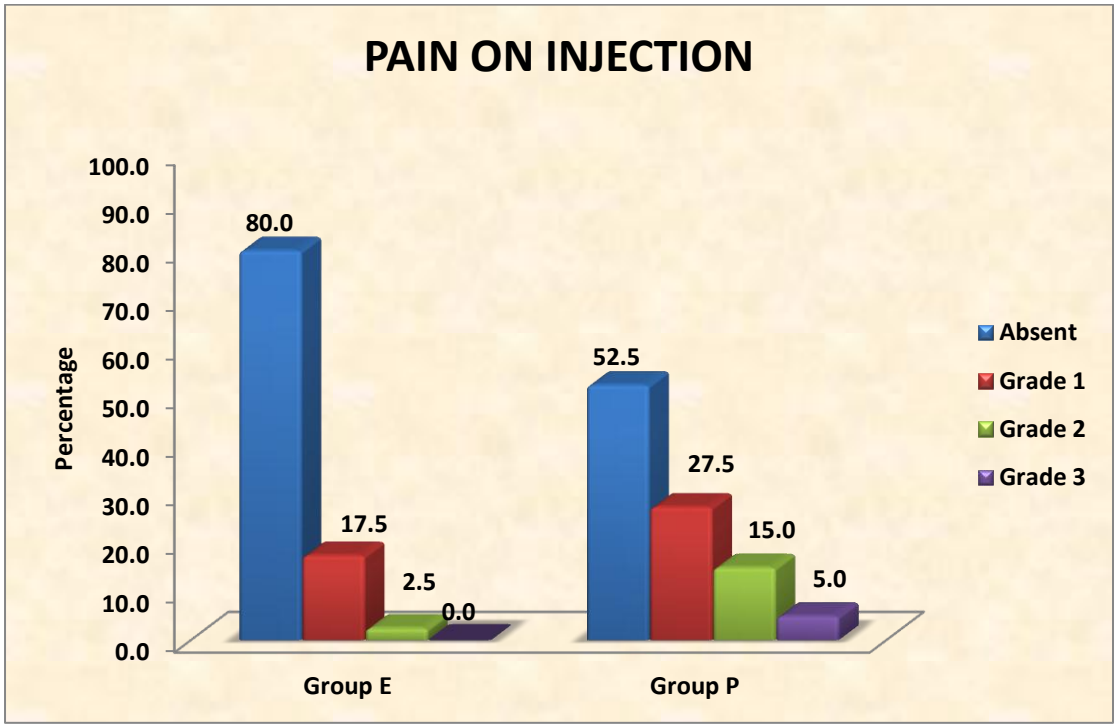
This shows decrease in MAP in group P when compared to group E. The change in mean MAP between the group at induction($p<0.001$) and during first minute immediately after induction were statistically significant ($p<0.009$).

Table 14: Distribution of pain on injection between the study groups

PAIN ON INJECTION	Group E		Group P		p value
	N	Percent	N	Percent	
Absent	32	80	21	52.5	0.032*
Grade I	7	17.5	11	27.5	
Grade II	1	2.5	6	15	
Grade III	0	0	2	5	
Total	40	100	40	100	

*significantly associated at 5% level of significance

Figure 14: Distribution of pain on injection between the study groups



Among forty patients in group E, 7 patients had grade I pain, 1 patient had grade II pain on injection.

In group P 11 patients had grade I, 6 patients grade II and 2 patients grade III pain on injection respectively

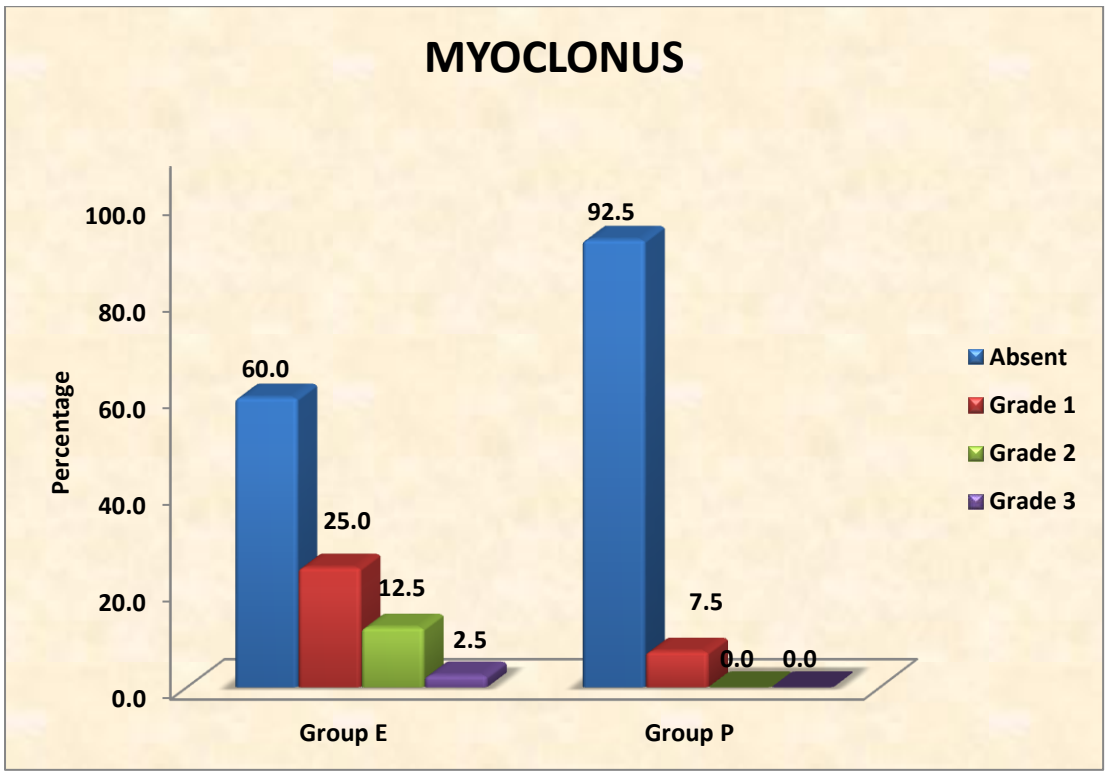
Pain on injection among the both groups were statistically significant ($p<0.032$)

Table 15: Incidence of Myoclonus between the study groups

MYOCLONUS	Group E		Group P		p value
	N	Percent	N	Percent	
Absent	24	60	37	92.5	
Grade 1	10	25	3	7.5	
Grade 2	5	12.5	0	0	0.005*
Grade 3	1	2.5	0	0	
Total	40	100	40	100	

*significantly associated at 5% level of significance

Figure 15: Incidence of Myoclonus



Among forty patients in group E 10 patients developed grade I myoclonus, grade II and grade III in 5 and 1 patients respectively.

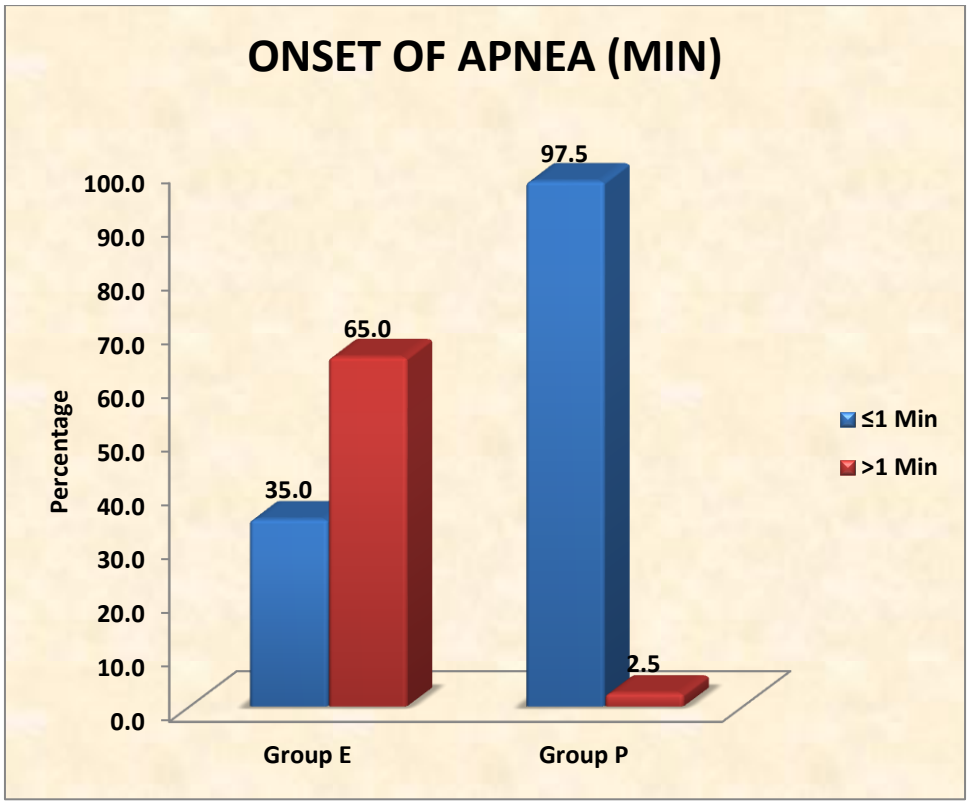
Among forty patients in group P 3 patients developed grade I myoclonus,

P value<0.005, which shows significance

Table 16: Distribution of Onset of Apnea between the study groups

ONSET OF APNEA	Group E		Group P	
	N	Percent	N	Percent
≤1 Min	14	35	39	97.5
>1 Min	26	65	1	2.5
Total	40	100	40	100

Figure 16: Distribution of Onset of Apnea between the study groups

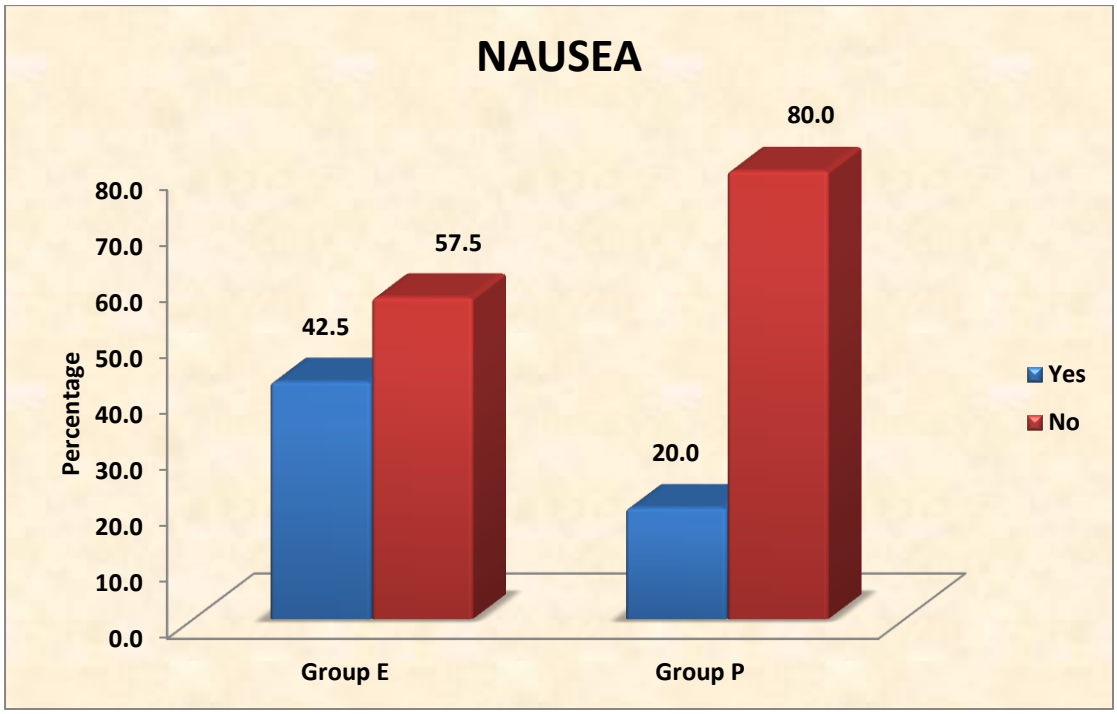


In group E 14 out of 40 patients had apnea in the first minute of induction, whereas in group P 39 patients had apnea during first minute.

Table 17: Incidence of Nausea between the study groups

NAUSEA	Group E		Group P	
	N	Percent	N	Percent
No	23	57.5	32	80
Yes	17	42.5	8	20
Total	40	100	40	100

Figure 17: Incidence of Nausea between the study groups

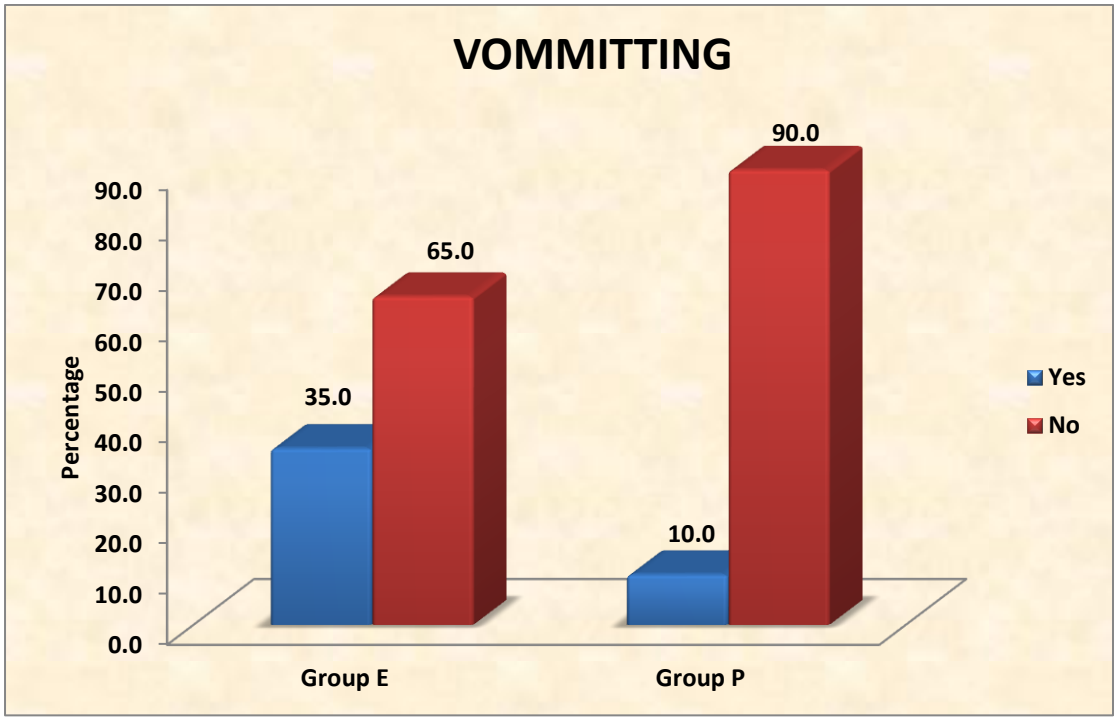


In group E 17 patients out of 40 had nausea post operatively as compared 8 patients in group P.

Table 18: Incidence of Vomiting between the study groups

Vomiting	Group E		Group P	
	N	Percent	N	Percent
No	26	65	36	90
Yes	14	35	4	10
Total	40	100	40	100

Figure 18: Incidence of Vomiting between the study groups



In group E 14 patients out of 40 had vomiting post operatively as compared 4 patients in group P.

DISCUSSION

Hypotension is known to occur with Propofol induction due to reduction of sympathetic activity causing vasodilatation, Direct effect on intracellular calcium mobilization, inhibition of prostaglandin synthesis in endothelial cells etc., are the causative factors.[11] Sudden hypotension has deleterious effects on maintaining the circulation to vital organs in conditions like Ischemic heart disease, valvular heart disease, systemic hypertension and shock. The hemodynamic stability observed with Etomidate may be due partly to its unique lack of effect on the sympathetic nervous system and on baroreceptor function[1,2].

In patients with valvular heart disease, pulmonary artery and pulmonary capillary wedge pressure also are reduced, implying the resultant decrease in pressure is due to a decrease in preload and after load. Although the decrease in systemic pressure after an induction dose of Propofol is due to vasodilation, the direct myocardial depressant effects of Propofol are more controversial[11].

The cardiovascular effects of Propofol have been evaluated after its use for induction and for maintenance of anaesthesia .The most prominent effect of Propofol is a decrease in arterial blood pressure during induction of anaesthesia.[11] Heart rate does not change significantly after an induction dose of Propofol. Propofol either may reset or may inhibit the baroreflex, reducing the tachycardic response to hypotension.[2] The most common side effect during induction of anaesthesia is hypotension, which is augmented by the concomitant administration of opioids.

The properties of Etomidate include hemodynamic stability, minimal respiratory depression, cerebral protection, and pharmacokinetics enabling rapid recovery after either a single dose or a continuous infusion.

Induction with Etomidate produces a brief period of hyperventilation, sometimes followed by a similarly brief period of apnea.[11] Apnea after induction with Propofol is common. The incidence of apnea is greater when compared to Etomidate.[30]

Gooding JM 1979 gave 0.3 mg/kg of Etomidate to cardiac patients for noncardiac surgery resulted in almost no change in heart rate, MAP, mean pulmonary artery pressure, pulmonary capillary wedge pressure, central venous pressure, stroke volume, cardiac index, and pulmonary and systemic vascular resistance[39].

Propofol also possesses significant antiemetic activity at low (sub hypnotic) doses[23,24]. This effect can be achieved by a 10- to 20-mg loading dose followed by infusion at 10μg/kg/min.

Myoclonus occur but less frequently with Propofol than after Etomidate[25]. Pain on injection is less than or equal to that with Etomidate[25,26].

The present study was carried out to evaluate the hemodynamic stability of the two drugs and to assess their side effects.

We have studied 80 of ASA grade I and II patients of both sexes between 18-60 years of age posted for elective surgeries. They were allocated into two groups of 40 each.

The two groups were designated as,

Group E: received Etomidate (0.3 mg / kg)

Group P: received Propofol. (2.5 mg / kg)

ANALYSIS OF DATA WITHIN THE GROUP

Heart rate changes

Present study shows the changes in mean heart rate, where it was seen that among group E, It shows that Group E patients the basal MHR in beats in beats per minute was 89.8 followed by 92.1 at intubation and 89.7 at 2min and 88 in 5min.

Among group P the basal MHR in beats per minute was 90.8, followed 78.1 at intubation and 94.8 at 2 min and 84.5 at 5min.

Statistical evaluation between the groups showed that the change in MHR observed in both the groups were statistically significant($p<0.05$)

Crido *et al* (1979) studied hemodynamic characteristics of 36 patients on induction with Etomidate, there was a reduction in cardiac out put, stroke volume and arterial pressure and compensatory increase in heart rate.[40]

John M. Gooding *et al* (1979) studied on 11 patients of ASA I-III the effects of Etomidate on cardiovascular system. They observed only 10% increase in heart rate.[39]

A. Gauss (1991) noticed the increase in the heart rate (HR) after Propofol injection but not with Etomidate[41]. John M. Gooding did not find significant change after induction with Etomidate it increased from 69 bpm before induction to 70 after induction.[41]

Giese JL, Stockham RJ, Stanley TH, *et al* (1985)noted the hemodynamic changes and side effects of anaesthesia induction with Etomidate or Thiopental in 83 ASA class I or 11 patients. Patients were randomly assigned to one of 12 groups

according to pretreatment drug (Fentanyl 100 mcg, or normal saline intravenously), induction agent (Etomidate 0.4 mg/kg, or Thiopental 4 mg/kg), and maintenance anesthetic technique (isofiurane-Oxygen, Isoflurane-Nitrous oxide-Oxygen, or Fentanyl-nitrousoxide Oxygen). There were significant increases in heart rate in all groups, especially after tracheal intubation. These increases were attenuated but not eliminated by Fentanyl pretreatment.[44]

Supriya Aggarwal *et al* (2016) conducted study to compare Propofol and Etomidate for their effect on hemodynamics and various adverse effects like myoclonus, pain on injection and apnea on patients in general anaesthesia in 100 ASA I and II of aged between 18-60 years. Patients in Etomidate(0.3mg/kg) group showed little change in mean arterial pressure (MAP) and heart rate (HR) compared to Propofol(2mg/kg) ($p > 0.05$) from baseline value.[70]

The results obtained in our study were similar to those obtained by above the studies.

BLOOD PRESSURE CHANGES

Changes in Mean Systolic Blood Pressure

Present study shows the changes in mean systolic blood pressure, where it was seen that among group E basal mean SBP was 128.8mmHg. One and two minutes of induction it was 110.5mmHg and 107.5mmHg respectively. It shows a fall in SBP of 21.3mmHg.

In group P basal mean SBP was 127.1mmHg. One and two minutes of induction it was 100.9mmHg and 96.4mmHg respectively. It shows a fall in SBP of 30.7mmHg.

This shows a more decrease in SBP in group P when compared to group E. The change in mean SBP between the group during first and second minute immediately after induction were statistically significant ($p<0.001$).

In group E the basal value of SBP was 128.8mmHg, 2min following intubation the SBP decreased to 117.6mmHg and 118.8mmHg at 5min.

In group P the basal value of SBP was 127.1mmHg, 2min following intubation the SBP decreased to 104mmHg and 110.8mmHg at 5min.

The decrease in SBP in group P was statistically significant compared to decrease in SBP in group E at 2min ($p<0.001$) and remained significant even upto 5min post intubation.

Changes in Mean Diastolic Blood Pressure

Present study shows the changes in mean diastolic blood pressure, where it was seen that among group E basal mean DBP was 83.7mmHg. one and two minutes of induction it was 71.7mmHg and 68mmHg respectively. It shows a fall in DBP of 15.7mmHg

In group P basal mean DBP was 80.6mmHg. one and two minutes of induction it was 67.3mmHg and 65.9mmHg respectively. It shows a fall in DBP of 14.7mmHg

This shows decrease in DBP in group P when compared to group E. The change in mean DBP between the group at induction($p<0.006$) and during first minute immediately after induction were statistically significant ($p<0.047$).

In group E the basal value of mean DBP was 83.7mmHg, 2min following intubation the DBP decreased to 78.5mmHg and 80.1mmHg at 5min.

In group P the basal value of mean DBP was 80.6mmHg, 2min following intubation the SBP decreased to 71.5mmHg and 74.1mmHg at min.

Statistical evaluation between the groups showed that the decrease in DBP observed in both groups was statistically significant ($p<0.001$) at intubation post intubation 2min and 5min.

Changes in Mean Arterial Blood Pressure

In group E basal MAP was 97.8mmHg. one and two minutes of induction it was 84.3mmHg and 79.3mmHg respectively. It shows a fall in DBP of 18.5mmHg

In group P basal MAP was 96.1mmHg. one and two minutes of induction it was 78.5mmHg and 76.1mmHg respectively. It shows a fall in DBP of 20mmHg

This shows decrease in MAP in group P when compared to group E. The change in mean MAP between the group at induction($p<0.001$) and during first minute immediately after induction were statistically significant ($p<0.009$).

In group E the basal value of MAP was 97.8mmHg, 2min following intubation the MAP decreased to 91.2mmHg and 92.9mmHg at 5min.

In group P the basal value of MAP was 96.1mmHg, 2min following intubation the MAP decreased to 82.3mmHg and 86.3mmHg at 5min.

Statistical evaluation between the groups showed that the decrease in MAP observed in both groups was statistically significant ($p<0.001$) at intubation post intubation 2min and 5min.

A.Gauss (1991) noticed the change in SBP by 1 mm Hg, DBP by 1mmHg with Etomidate and SBP decreased by 13 mmHg, DBP by 4 mmHg in Propofol group.[41]

Thomas Brussel (1992) found no change in SBP, 1 mm Hg decrease in DBP, no change in MAP with Etomidate and 20 mmHg decrease in SBP, 15 mmHg decrease in DBP, 16 mmHg decrease in MAP with Propofol.[55]

A. Criado (1980) noticed 18 mmHg decrease in SBP, 10 mmHg decrease in MAP and 6 mmHg decrease in DBP after induction with Etomidate.[40]

Saricaoglu *et al* (2011) were compared Etomidate-lipuro and Propofol and 50%, (1:1) admixture of these agents at induction with special reference to injection pain, hemodynamic changes, and myoclonus. They noticed that the hemodynamic (systolic, diastolic and mean blood pressures, heart rate) changes were minimal in group PE than other two groups (P = 0.017). [62]

Supriya Aggarwal *et al* (2016) conducted study to compare Propofol and Etomidate for their effect on hemodynamics and various adverse effects like myoclonus, pain on injection and apnea on patients in general anaesthesia in 100 ASA I and II of aged between 18-60 years and they found that Patients in Etomidate(0.3mg/kg) group showed little change in mean arterial pressure (MAP) and heart rate HR) compared to Propofol(2mg/kg) ($p > 0.05$) from baseline value.

A study by J G Reves *et al* showed that the cardiovascular effects of Propofol have been evaluated after its use for induction and for maintenance of anaesthesia. The most prominent effect of Propofol is decrease in arterial blood pressure during induction of anaesthesia.[11]

Incidence of Myoclonus

Among forty patients in group E 10 patients developed grade I myoclonus, grade II and grade III in 5 and 1 patients respectively.

Among forty patients in group P 3 patients developed grade I myoclonus ($p < 0.005$).

Giese JL, Stockham RJ, Stanley TH, *et al* (1985) noted the hemodynamic changes and side effects of anaesthesia induction with Etomidate or Thiopental in 83 ASA class I or 11 patients. Patients were randomly assigned to one of 12 groups according to pretreatment drug (Fentanyl 100 mcg, or normal saline intravenously), induction agent (Etomidate 0.4 mg/kg, or Thiopental 4 mg/kg), and maintenance anesthetic technique (isofiurane-Oxygen, Isoflurane-Nitrous oxide-Oxygen, or Fentanyl-nitrousoxide Oxygen). Patients in whom anaesthesia was induced with Etomidate had a greater incidenc myoclonus. Fentanyl pretreatment significantly decreased the incidence myoclonus

Saricaoglu *et al* (2011) were studied to compare Etomidate-lipuro and Propofol and 50%, (1:1) admixture of these agents with reference to myoclonus. The intensity of myoclonus was more in the group E (76.3%). Myoclonus was not observed in group PE and group P.[62]

A double blind study conducted by Lars Huter *et al* (2007) on 80 patients using low dose Midazolam for reducing the incidence of myoclonus by Etomidate. In 40 patients who received Midazolam along with Etomidate 2 patients developed myoclonus whereas 10 patients developed myoclonus in Etomidate group. So he concluded that Midazolam 0.015mg/kg as premedication is effective in reducing myoclonus.[51]

Kahlon A.Singh, Gupta Ruchi, Aujla K.Singh, Bindra T.Kaur (2014) did a study to know the efficacy of Lignocaine versus Midazolam in controlling Etomidate induced myoclonus on 75 ASA grade I and II patients. They found that incidence of myoclonus was 76% in control group, 28% in Midazolam group and 44% in Lignocaine group and concluded that both Midazolam and Lignocaine were effective in reducing the incidence and severity of myoclonus.[66]

Ebru Kelsaka (2006) found reduction in incidence of myoclonus with remifentanil premedication with Etomidate. Moderate myoclonus was found in 2 patients among 30 patients.[53]

Supriya Aggarwal *et al* (2016) studied to compare Propofol and Etomidate for their effect on hemodynamics and various adverse effects like myoclonus, pain on injection and apnea on patients in general anaesthesia in 100 ASA I and II of aged between 18-60 years. They found that myoclonus activity was higher in Etomidate group.[70]

Pain on injection

Among forty patients in group E, 7 patients had grade I pain, 1 patient had grade II pain on injection.

In group P 11 patients had grade I, 6 patients grade II and 2 patients grade III pain on injection respectively ($p<0.032$).

A.W.Doenicke *et al* (1999) compared the pain on injection between Etomidate in propylene glycol and Etomidate-lipuro with medium chain fatty acids. Nine out often patients reported moderate to severe pain on injection where as one of ten patients had pain. Hence they came out with conclusion that Etomidate-lipuro causes less pain on injection.[49]

Y.Nyman *et al* (2006) studied 110 pediatric patients aged 2-16 years for incidence of injection pain on induction using four point scale. A significantly lower incidence of injection pain was found in the Etomidate-lipuro group as compared to Propofol-lidocaine group.[50]

Saricaoglu *et al* (2011) were studied to compare Etomidate-lipuro and Propofol and 50%, (1:1) admixture of these agents with reference to injection pain. They found that there were no injection pain in group PE as the incidence were (83.8%) in group P and in (63.2%) group E. [62]

Mohammadreza safavi *et al* (2014) studied to comapare the magnesium sulphate and lignocaine pretreatment for prevention of pain on Etomidate induction on 135 patients undergoing elective surgeries under general anaesthesia. They found that 60% patients in control group had pain on injection was assessed by a four point scale. They found that 60% patients in control group had pain on injection and 22.2% and 40% in Lignocaine and Magnesium Sulphate group respectively and concluded that both the drugs are comparably effective in reducing Etomidate induced pain.[66]

Supriya Aggarwal *et al* (2016) conducted study to compare Propofol and Etomidate for their effect on hemodynamics and various adverse effects like myoclonus, pain on injection and apnea on patients in general anaesthesia in 100 ASA I and II of aged between 18-60 years. They found that pain on injection was more in Propofol group.[70]

Onset of apnea

In group E 14 out of 40 patients had apnea in the first minute of induction, whereas in group P 39 patients had apnea during first minute.

Supriya Aggarwal *et al* (2016) conducted study to compare Propofol and Etomidate for their effect on hemodynamics and various adverse effects like myoclonus, pain on injection and apnea on patients in general anaesthesia in 100 ASA I and II of aged between 18-60 years. The episodes of apnea were transient and not associated with ant fall in Oxygen saturation.[70]

J.S.C.McCollum (1986) noticed 11% of patients after Propofol and none of the patients receiving Etomidate.[56]

John M study with Etomidate induction showed transient apnea in 16%. Among forty patients in group I, 12 had mild pain on injection, In group II 15 patients had mild pain, moderate and severe pain in 3 and 1 patients respectively.[34]

Incidence of Nausea

Present study shows the incidence of nausea, where in group E 17 patients out of 40 had nausea post operatively as compared 8 patients in group P.

Incidence of vomiting

Present study shows the incidence of vomiting, where in group E 14 patients out of 40 had vomiting post operatively as compared 4 patients in group P.

In a double blind randomized study, M.St pierre (2000) studied the incidence and severity of post-operative nausea and vomiting was investigated with Etomidate and Propofol. He noted nausea in 17 patients, vomiting in 13 patients in Etomidate

group of 80 patients and nausea in 17 patients and vomiting in 5 patients in Propofol group of 80 patients. They concluded that Etomidate does not increase nausea during early post-operative period.[47]

A study by Borgeat A *et al* (1992) showed Propofol possesses significant antiemetic activity at low doses. This effect can be achieved by a 10-20mg loding dose followed by infusion at 10mcg/kg/min.[23]

CONCLUSION

- Patients induced with Propofol had significant decrease in systolic, diastolic blood pressure and mean arterial pressures at 10minute after induction compared to Etomidate.
- This characteristic indicates that Etomidate maintained hemodynamic stability.
- Heart rate changes were significant between the two groups.
- Incidence of apnea and pain on injection were more with Propofol group, however, Etomidate caused more of myoclonus than Propofol.
- Post operative Nausea and vomiting were more in Etomidate group compared to Propofol group.
- So Etomidate is better inducing agent than Propofol with regard to cardiovascular stability.

SUMMARY

The present study entitled "**COMPARATIVE STUDY OF ETOMIDATE AND PROPOFOL FOR INDUCTION OF GENERAL ANAESTHESIA**" was carried out at BLDE University Shri. B. M. Patil Medical College, Hospital And Research Centre, Vijayapur, from December 2014 to June 2016. The study population consisted of 80 patients divided in two groups.

1. Group E– Received Etomidate (0.3mg/kg)
2. Group P– Received Propofol (2.5mg/kg)

The demographic changes such as Age, Sex, Weight, were comparable in all the groups.

Statistical evaluation between both the groups sowed that the increase in heart rate was significant after intubation ($p<0.05$). raise in heart rate was more in group P.

The SBP in group E and group P decreased by 21.3mmHg and 30.7mmHg respectively at the end of second minute of induction.

The DBP in group E and group P decreased by 15.7 mm Hg and 14.7 mm Hg respectively at the end of second minute of induction.

The MAP in group E and group P decreased by 18.5mmHg and 20 mmHg respectively at the end of second minute of induction.

The maximum decrease in SBP, MAP and DBP is seen in group P compared to group E. group E is more hemodynamically stable compared to group P.

The apnea occurred in 14 out of 40 patients in group E and 39 out of 40 patients in group P patients.

Pain on injection was more in group P, 11 patients had grade I, 6 patients grade II and 2 patients grade III pain on injection respectively, where as in group E 7 patients had grade I pain, 1 patient had grade II pain on injection.

Among forty patients in group E, 10 patients developed grade I myoclonus, grade II and grade III in 5 and 1 patients respectively.

Among forty patients in group P, 3 patients developed grade I myoclonus.

Among forty patients in group E, 17 patients had nausea post operatively as compared 8 patients in group P.

Among forty patients in group E 14 patients out of 40 had vomiting post operatively as compared 4 patients in group P.

BIBLOGRAPHY

1. Stoelting Robert and Simon C.Hiller. Pharmacology and Physiology in Anesthetic practice. 4th edition. Philadelphia: Lippincott Williams and Wilkins publishers.,2006,159-160.
2. Ebert TJ, Muzi M, Berens R, *et al*. Sympathetic responses to induction of anaesthesia in humans with Propofol or Etomidate.*Anesthesiology* 1992;76:725-733.
3. Larsen R, Rathgeber J, Bagdahn A, *et al*. Effects of Propofol on cardiovascular dynamics and coronary blood flow in geriatric patients. A comparison with Etomidate. *Anaesthesia* 1988; 43(Suppl):25-31.
4. Van Aken H, Meinshausen E, Prien T, *et al*. The influence of Fentanyl and tracheal intubation on the hemodynamic effects of anaesthesia induction with Propofol/N2O in humans. *Anesthesiology* 1988; 68:157-163.
5. Stoelting, Roberta L. Hinges, Katherine E. Marschall. Stoelting's Anaesthesia and Co-existing Disease. 5th Edition. Philadelphia: Churchill Livingstone, 2009.
6. R. Carlos, S. Innerarity. Effect of Premedication on Etomidate Anaesthesia. *British Journal of Anaesthesia*. 1979;51:1159.
7. Guyton, Arthur C, Hall, John. Guyton and Hall Textbook of Medical Physiology. 10th ed. Philadelphia: Elsevier Saunders; 2006.
8. William F Ganong, Kim E Barrett. Ganong Review of Medical Physiology. 22nd ed. Newyork: McGraw-Hill Medical; 2005.
9. Mehernoor F. Wathca, Paul F. White, Postoperative Nausea and Vomiting. *Anesthesiology* 1992;77:162-184.

10. Tong J. Gan; Mechanism underlying Postoperative Nausea and Vomiting and Neurotransmitter Receptor Antagonist Based Pharmacology: *CNS Drugs* 2007;21(10):813-833.
11. .J. G. Reves, Peter Glass, David A. Lubarsky. In: Ronald D Miller, editors. Miller's Anaesthesia. 7th ed. Philadelphia: *Churchill Livingstone Elsevier;* 2005. P.719-769.
12. Hiroshi Ohmiza, Shinju Obara, Hiroshi Iwama. Mechanism of injection pain with long and long- medium chain triglyceride emulsive Propofol. *Canadian Journal of anaesthesia* 2005;52:595-599.
13. Kuipers JA, Boer F, Olieman W, *et al*. First-pass lung uptake and pulmonary clearance of Propofol: Assessment with a recirculatory indocyanine green pharmacokinetic model. *Anesthesiology* 1999; 91:1780-1787.
14. Kay NH, Sear JW, Uppington J, *et al*. Disposition of Propofol in patients undergoing surgery: A comparison in men and women. *British Journal of Anaesthesia* 1986; 58:1075-1079.
15. Veroli P, O'Kelly B, Bertrand F, *et al*. Extrahepatic metabolism of Propofol in man during the anhepatic phase of orthotopic liver transplantation. *British Journal of Anaesthesia* 1992; 68:183-186.
16. Kirkpatrick T, Cockshott ID, Douglas EJ, Nimmo WS. Pharmacokinetics of Propofol (diprivan) in elderly patients. *British Journal of Anaesthesia* 1988; 60:146-150.
17. Shafer A, Doze VA, Shafer SL, White PF. Pharmacokinetics and pharmacodynamics of Propofol infusions during general anaesthesia. *Anesthesiology* 1988; 69:348-356.

18. Taylor MB, Grounds RM, Mulrooney PD, Morgan M. Ventilatory effects of Propofol during induction of anaesthesia: Comparison with thiopentone. *Anaesthesia* 1986; 41:816-820.

19. Conti G, Dell'Utri D, Vilardi V, *et al*. Propofol induces bronchodilation in mechanically ventilated chronic obstructive pulmonary disease (COPD) patients. *Acta Anaesthesiol Scand* 1993; 37:105-109.

20. Larsen R, Rathgeber J, Bagdahn A, *et al*. Effects of Propofol on cardiovascular dynamics and coronary blood flow in geriatric patients: Acomparison with Etomidate. *Anaesthesia* 1988; 43(Suppl):25-31.

21. Yushi U. Adachi, Maiko Sotomoto, Hideyuki Higuchi *et al*. Fentanyl attenuates the hemodynamic response to Endotracheal Intubation more than the response to Laryngoscopy. *Anaesthesia Analgesia* 2002;95:233-7.

22. Stephan H, Sonntag H, Schenk HD, *et al*. Effects of Propofol on cardiovascular dynamics, myocardial blood flow and myocardial metabolism in patients with coronary artery disease. *British Journal of Anaesthesia* 1986; 58:969-975.

23. Borgeat A, Wilder-Smith OH, Saiah M, Rifat K. Subhypnotic doses of Propofol relieve pruritus induced by epidural and intrathecal morphine. *Anesthesiology* 1992; 76:510-512.

24. Gan TJ, Glass PS, Howell ST, *et al*. Determination of plasma concentrations of Propofol associated with 50% reduction in postoperative nausea. *Anesthesiology* 1997; 87:779-784.

25. Mirakhur RK. Induction characteristics of Propofol in children: Comparison with thiopentone. *Anaesthesia* 1988; 43:593-598.

26. Doenicke A, Roizen MF, Nebauer AE, *et al*. A comparison of two formulations for Etomidate, 2-hydroxypropyl-beta-cyclodextrin (HPCD) and propylene glycol. *Anaesthesia Analgesia* 1994; 79:933-939.

27. Shuichi yokota, Toru Komatsu, Yoko Komura *et al*. Pretreatment with Topical 60% Lidocaine Tape reduced pain on injection of Propofol. *Anaesthesia Analgesia* 1997;85:672-4.

28. King SY, Davis FM, Wells JE, *et al*. Lidocaine for the prevention of pain due to injection of Propofol. *Anaesthesia Analgesia* 1992;74: 246-9.

29. Kyunghwa Kwak, Hoysun Chung, Choonhak Lim *et al*. A combination of Lidocaine (Lignocaine) and Remifentanil reduces pain during Propofol injection. *Clinical drug Investigation* 2007;27(7):493-497.

30. Turtle MJ, Cullen P, Prys-Roberts C, *et al*. Dose requirements of Propofol by infusion during Nitrous oxide anaesthesia in man, II: Patients premedicated with lorazepam. *British Journal of Anaesthesia* 1987; 59:283-287.

31. Doenicke A, Kugler. N. Vollmann *et al*. Etomidate in a new solution promoter clinical experimental investigations of toleration by the veins and bioavailability. *Anaesthesist* 1990; 39:475-480.

32. Doenicke AW, Roizen MF, Rau J, *et al*. Pharmacokinetics and pharmacodynamics of Propofolin a new solvent. *Anaesthesia Analgesia* 1997;85: 1399–403.

33. Michael J. Van Hamma, M. M. Ghoneim, John J Ambre. Pharmacokinetics of Etomidate, a New Intravenous Anesthetic:*Anesthesiology* 1978;49:274-277.

34. Gooding JM, Corssen G: Etomidate: an ultrashort-acting nonbarbiturate agent for anaesthesia. *Anaesthesia Analgesia* 1976;55:286.

35. Robert J. Fragen, Nancy Caldwell, Edward A. Brunner. Clinical use of Etomidate for anaesthetic induction: A preliminary report. *Anaesthesia analgesia* 1976;55:730-731.

36. Dearden NM, McDowall DG. Comparison of Etomidate and althesin in the reduction of increased intracranial pressure after head injury. *British Journal of Anaesthesia* 1985; 57:361-368.

37. Cold GE, Eskesen V, Eriksen H, *et al*. CBF and CMRO2 duringcontinuous Etomidate infusion supplemented with N2O and Fentanyl in patients with supratentorial cerebral tumour: A dose-response study. *Acta Anaesthesiol Scand* 1985; 29:490-494.

38. Gooding JM, Weng JT, Smith RA, *et al*. Cardiovascular and pulmonary responses following Etomidate induction of anaesthesia in patients with demonstrated cardiac disease. *Anaesthesia Analgesia* 1979; 58:40-41.

39. Gooding JM, Corssen G: Effect of Etomidate on the cardiovascular system. *Anaesthesia Analgesia* 1977; 56:717-719.

40. Criado A, Maseda J, Navarro E, *et al*. Induction of anaesthesia with Etomidate: Haemodynamic study of 36 patients. *British Journal of Anaesthesia* 1980; 52:803-806.

41. A. Gauss, H. Heinrichand H. G. Wilder-Smith. Echocardiographic assessment of the haemodynamic effects of Propofol : a comparison with Etomidate and Thiopentone. *Anaesthesia* 1991;46:99-105.

42. Duthie DJ, Fraser R, Nimmo WS. Effect of induction of anaesthesia with Etomidate on corticosteroid synthesis in man. *British Journal of Anaesthesia* 1985; 57:156-159.

43. Fragen RJ, Caldwell N. Comparison of a new formulation of Etomidate with Thiopental—side effects and awakening times.*Anesthesiology* 1979; 50:242-244.

44. Giese JL, Stockham RJ, Stanley TH, *et al*. Etomidate versus Thiopental for induction of anaesthesia. *Anaesthesia Analgesia* 1985; 64:871-876.

45. Wells JK. Comparison of ICI 35868, Etomidate and methohexitone for day-case anaesthesia. *British Journal of Anaesthesia* 1985; 57:732-735.

46. Craig J, Cooper GM, Sear JW. Recovery from day-case anaesthesia: Comparison between methohexitone, Althesin and Etomidate. *British Journal of Anaesthesia* 1982; 54:447-451.

47. M. St Pierre. et. al. Does Etomidate increase post operative nausea? A double blind controlled comparision of Etomidate in lipid emulsion with Propofol for balanced anaesthesia. *European Journal of anaesthesiology* 2000;7:1-9.

48. Galloway PA, Nicoll JM, Leiman BC: Pain reduction with Etomidate injection. *Anaesthesia* 1982; 37:352-353.

49. Doenicke AW, Roizen MF, Hoernecke R, *et al*. Solvent for Etomidate may cause pain and adverse effects. *British Journal of Anaesthesia* 1999;83(3):464-466.

50. Y. Nyman. et. al. Etomidate-Lipuro is associated with considerably less injection pain in children compared with Propofol with added lidocaine. *British Journal of Anaesthesia* 2006;10:1093.

51. Huter L, Schreiber T, Gugel M, Schwarzkopf K. Low-dose intravenous midazolam reduces Etomidate-induced myoclonus: A prospective, randomized study in patients undergoing elective cardioversion. *Anaesthesia Analgesia* 2007; 105:1298-1302.

52. Doenicke AW, Roizen MF, Kugler J, *et al*. Reducing myoclonus after Etomidate. *Anesthesiology* 1999;90:113–9.

53. EbruKelsaka, Deniz Kara kaya, BinnurSarihasan *et al*. Remifentanil pretreatment reduces myoclonus after Etomidate. *Journal of clinical anaesthesia* 2006;18:83-86.

54. Doenicke AW, Roizen MF, Hoernecke R, *et al*: Haemolysis after Etomidate : comparision of propylene glycol and lipid formulations *British Journal of Anaesthesia* 1997;79:386-388.

55. Thomas Brussel, Josef L. Theissen, Gisili Vigfusson, P. Paul Lunkenheimer *et al*: Hemodynamic and cardiodynamic effects of Propofol and Etomidate: Negetive Inonotropic properties of Propofol. *Anaesthesia Analgesia* 1989;69:35-40.

56. J. S. C. McCollum and J. W. Dundee: Comparison of induction characteristics of four intravenous anaesthetic agents. *Anaesthesia* 1986;41:995-1000.

57. Schaeuble *et al* (2005). Comparision of Etomidate and Propofol for fibreoptic intubation as part of an airway management algorithm:a

prospective, randomizes, double-blind study. *Eur J Anaesthesiol*. 2005 Oct;22(10):762-7.

58. Sarkar M, Laussen P, Zurakowski D, Shukla A, Kussman B, Odegard K. Hemodynamic Responses to Etomidate on Induction of Anaesthesia in Pediatric Patients. *Anaesthesia & Analgesia*. 2005;101(3):645-650.

59. Kim T, Park I. Comparative Study of Brain Protection Effect between Thiopental and Etomidate Using Bispectral Index during Temporary Arterial Occlusion. *J Korean Neurosurg Soc*. 2011;50(6):497.

60. Morel J, Salard M, Castelain C, Bayon M, Lambert P, Vola M *et al*. Haemodynamic consequences of Etomidate administration in elective cardiac surgery: a randomized double-blinded study. *British Journal of Anaesthesia*. 2011;107(4):503-509.

61. Kalogridaki M[1], Souvatzis X, Mavrakis HE, Kanoupakis EM, Panteli A, Kasotaki S *et al*. Anaesthesia for cardioversion: a prospective randomised comparison of Propofol and Etomidate combined with Fentanyl. *J Korean Neurosurg Soc*. 2011 Dec;50(6):497-502.

62. Saricaoglu F, Uzun S, Arun O, Arun F, Aypar U. A clinical comparison of Etomidate-lipuro, Propofol and admixture at induction. *Saudi Journal of Anaesthesia*. 2011;5(1):62.

63. Pandey A, Makhija N, Chauhan S, Das S, Kiran U, Bisoi A *et al*. The Effects of Etomidate and Propofol Induction on Hemodynamic and Endocrine Response in Patients Undergoing Coronary Artery Bypass Graft Surgery on Cardiopulmonary Bypass. *World Journal of Cardiovascular Surgery*. 2012;02(03):48-52.

64. Masoudifar M, Beheshtian E. Comparison of Cardiovascular Response to Laryngoscopy and Tracheal Intubation After Induction of Anaesthesia by Propofol and Etomidate. *J Res Med Sci*. 2013 Oct; 18(10): 870–874.

65. Singh K, Ruchi G, Singh A, Kaur B. Efficacy of lignocaine versus midazolam in controlling Etomidate-induced myoclonus: a randomized placebo-controlled study. *Ain-Shams Journal of Anaesthesiology*. 2014;7(3):460.

66. Honarmand A, Iazdani A, Norian N, Payandeh M, Safavi M, Sahafi A *et al*. Magnesium sulfate versus Lidocaine pretreatment for prevention of pain on Etomidate injection: A randomized, double-blinded placebo controlled trial. *Journal of Research in Pharmacy Practice*. 2015;4(1):4.

67. Shah S, Chowdhury I, Bhargava A, Sabbharwal B. Comparison of hemodynamic effects of intravenous Etomidate versus Propofol during induction and intubation using entropy guided hypnosis levels. *J Anaesthesiol Clin Pharmacol*. 2015;31(2):180.

68. Kaushal R, Vatal A, Pathak R. Effect of Etomidate and Propofol induction on hemodynamic and endocrine response in patients undergoing coronary artery bypass grafting/mitral valve and aortic valve replacement surgery on cardiopulmonary bypass. *Annals of Cardiac Anaesthesia*. 2015;18(2):172.

69. Desai P, Kane D, Sarkar M. Cardioversion: What to choose? Etomidate or Propofol. *Annals of Cardiac Anaesthesia*. 2015;18(3):306.

70. Aggarwal S, Goyal V, Chaturvedi S, Mathur V, Baj B, Kumar A. A comparative study between Propofol and Etomidate in patients under general anaesthesia. *Brazilian Journal of Anesthesiology* (English Edition). 2016;66(3):237-241.

71. Meena K, *et al*. A Comparative Study of Effect of Propofol, Etomidate and Propofol Plus Etomidate Induction on Hemodynamic Response to Endotracheal Intubation: A RCT. *Journal of Anaesthesia & Clinical Research*. 2016;07(05).

ANNEXURES

ETHICAL CLEARANCE CERTIFICATE

Institutional Ethical Committee
BIJAPUR-586 103.
OUTWARD
No. 6014
Date 24/11/14
BLDE U's Shri B.M. Patil Medical College

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 22-11-2014 *at* 3-30 pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title "A Comparative Study of Etomidate and Propofol for Induction of General"

Name of P.G. student Dr. Santosh Kumar Halamath.
Dept of Anaesthesiology

Name of Guide/Co-investigator Dr Vijay Kumar. T.K. Professor.
Dept of Anaesthesiology

for

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR – 586103, KARNATAKA

TITLE OF THE PROJECT: **"COMPARATIVE STUDY OF ETOMIDATE AND PROPOFOL FOR INDUCTION OF GENERAL ANAESTHESIA"**

INVESTIGATOR : **Dr. SANTOSHKUMAR ALALAMATH**
Department Of Anaesthesiology

PG GUIDE : **Dr. VIJAYKUMAT T. K** **MD.,DA**
PROFESSOR
DEPT OF ANAESTHESIOLOGY
B.L.D.E. UNIVERSITY'S SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, SOLAPUR ROAD
VIJAYAPUR-586103

PURPOSE OF RESEARCH:

I have been informed that this study is **"COMPARATIVE STUDY OF ETOMIDATE AND PROPOFOL FOR INDUCTION OF GENERAL ANAESTHESIA".**I have been explained about the reason for doing this study and selecting me/my ward as a subject for this study. I have also been given free choice for either being included or not in the study.

PROCEDURE:

I understand that I will be doing **"COMPARATIVE STUDY OF ETOMIDATE AND PROPOFOL FOR INDUCTION OF GENERAL ANAESTHESIA".**

RISKS AND DISCOMFORTS:

I understand that I/my ward may experience some pain on injection, myoclonus , nausea and vomiting and I understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

I understand that my/my wards participation in this study will help in finding out **"COMPARATIVE STUDY OF ETOMIDATE AND PROPOFOL FOR INDUCTION OF GENERAL ANAESTHESIA".**

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time. **Dr.Santoshkumar Alalamath** is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me, and that a copy of this consent form will be given to me for keep for careful reading.

REFUSAL OR WITHDRAWL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that **Dr.Santoshkumar Alalamath** will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: Dr. Vijaykumar T.K (Guide)

Dr. Santoshkumar Alalamath (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that **Dr. Santoshkumar Alalamath** has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

PROFORMA

"COMPARATIVE STUDY OF ETOMIDATE AND PROPOFOL FOR INDUCTION OF GENERAL ANAESTHESIA"

Patient Name :	I.P. No:
Age :	Gender:
Date of Operation:	Occupation:
Address :	Anaesthesiologist:

Preanaesthetic evaluation

Chief Complaints

Past History

a. HTN / DM / Asthma / Epilepsy / Drug allergy

b. Drug therapy

c. Previous exposure to anaesthesia

Family history

General Physical Examination

Pallor / Icterus / Clubbing / Lymphadenopathy / Odema

P.R.: B.P.:

R.R.:

Musculoskeletal disorders

Jaw movements	Teeth:
Airway assessment:	Spine:

Systemic examination

R.S.	CNS
CVS	GIT

Investigations

Hb%:	Total count:
Differential count:	Bleeding time:
Clotting time:	PT:
aPTT:	INR:
Urine routine	
Any others	

Preoperative physical status: ASA Grade I II

Diagnosis:

Proposed surgery:

Preoperative baseline:

HR :	BP:

Monitors attached

Pulse oximeter	Non invasive blood pressure:
ECG	

Group: Group E / Group P

Premedication : Inj.Fentanyl 2mcg/kg, Inj Glycopyrrolate 0.2mg ,ten minutes before induction

Induction agent :

Group A: Etomidate 0.3 mg/kg given slowly over 45 seconds

Group B: Propofol 2.5 mg/kg given slowly over45 seconds

Observations:

1)

	Heart Rate/minute	**SBP and DBP(mmHg)**	**MAP(mmHg)**
Basal			
Induction			
1min			
2min			
3min			

2)

	Heart Rate/minute	**SBP and DBP(mmHg)**	**MAP(mmHg)**
Basal			
Post Intubation			
1min			
3min			
5min			
10min			

3) Pain on injection : Grade0/Grade 1/Grade2/Grade3

Grade 0 - No pain

Grade 1 - Verbal complain of pain

Grade 2 - Withdrawal of arm

Grade 3 – Both Verbal complain of pain and Withdrawal of arm

3) Onset of Apnea in seconds:

4) **Myoclonus Grading** : Absent/Mild/Moderate/Severe

Absent-No myoclonus

Mild – Short movement of body segment (a finger or shoulder).

Moderate -Slight movement of two different muscles or muscle groups of the body.

Severe-Intense clonic movements in two or more muscle groups of the body (fast abduction of a limb).

5) Post operative Nausea / Vomiting: Yes/ No

KEYWORDS TO MASTER CHART

SL.NO	–	Serial Number
HR	–	Heart rate
SBP	–	Systolic blood pressure\
DBP	–	Diastolic blood pressure
MAP	–	Mean arterial pressure
M	–	Male
F	–	Female
’	–	Minute
”	–	Seconds
Kg	–	Kilograms