COMPARISON OF SEVOFLURANE WITH PROPOFOL FOR LARYNGEAL MASK AIRWAY INSERTION IN ADULTS.

Dissertation submitted

in partial fulfilment of the requirements

for award of the degree

M.D. (Anaesthesiology)

BranchX

GOVT. KILPAUK MEDICAL COLLEGE

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

APRIL 2016

CERTIFICATE

This is to certify that this dissertation entitled "**COMPARISON OF SEVOFLURANE WITH PROPOFOL FOR LARYNGEAL MASK AIRWAY INSERTION IN ADULTS"** submitted by **Dr. THAMIL SELVI B.S** in partialfulfillment for the award of the degree of Doctor of Medicine in Anaesthesiology by The Tamil NaduDr.M.G.R. Medical University, Chennai is a bonafide work done by her at GOVERNMENT KILPAUK MEDICAL COLLEGE, CHENNAI during the academic year 2014-2016.

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CERTIFICATE

This is to certify that the dissertation entitled"**COMPARISON OF SEVOFLURANE WITH PROPOFOL FOR LARYNGEAL MASK AIRWAY INSERTION IN ADULTS"** submitted by **Dr. THAMIL SELVI B.S** in partial fulfillment for the award of the degree of Doctor of Medicine in Anaesthesiology for the April 2016 examination by the Tamil Nadu Dr. M.G.R. Medical University, Chennai , this is a bonafide original research work done by her in the department of Anaesthesiology, Govt. kilpauk Medical College, under my guidance and supervision.

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DECLARATION

I, **Dr. THAMIL SELVI B.S** solemnly declare that this dissertation, entitled "**COMPARISON OF SEVOFLURANE WITH PROPOFOL FOR LARYNGEAL MASK AIRWAY INSERTION IN ADULTS"**, has been prepared by me, under the expert guidance and supervision of Prof.Dr.T.Murugan, M.D.,D.A, HOD, Department of Anaesthesiology,Prof.Dr. M. Bhavani, M.D, Department of Anaesthesiology, Government Kilpauk Medical College and Hospital,Chennai and submitted in partial fulfilment of the regulations for the award of the degree M.D.(Anaesthesiology) by The Tamil Nadu Dr. M.G.R. Medical University and the examination to be held in April 2016.

This study was conducted at Government Kilpauk Medical College Hospital, Chennai. I have not submitted this dissertation previously to any university for the award of any degree or diploma.

Place: Chennai (DR.B.S.THAMILSELVI)
Date:

ACKNOWLEDGEMENT

I wish to express my sincere thanks to Prof.Dr.Narayanababu MD.,DCHDean, Government of Kilpauk Medical College, Chennai for having kindlypermitted me to utilize the facilities of the college for the conduct of the study.

I am grateful to the Professor and Head of the Department of Anesthesiology, Govt. Kilpauk Medical College, Prof. Dr. T.Murugan., M.D.,D.A., Prof. Dr. M. Bhavani, M.D. for their motivation, valuable suggestions, expert guidance, adviceand for providing all necessary arrangement for conducting the study.

I also express my sincere gratitude to all other Professors of Anaesthesiology,KMCH, Prof. Dr.R.Kundhavidevi, MD., DA.,Prof.Dr.ValliSathyamoorthy, M.D., D.A.,Prof. Dr. A.Chandrasekar, M.D., , for their constant motivation, encouragement and valuable suggestions.

I thank all the Assistant Professors and tutors of Anesthesiology KMCH and GRH for their keen interest and support without which this study would not have been possible.

I am thankful to the Institutional Ethical Committee for their guidance and approval of the study.

I also thank my entire colleague Postgraduates for supporting me throughout the study.

I also thank Mr. A. Guganesan for supporting me throughout the study.

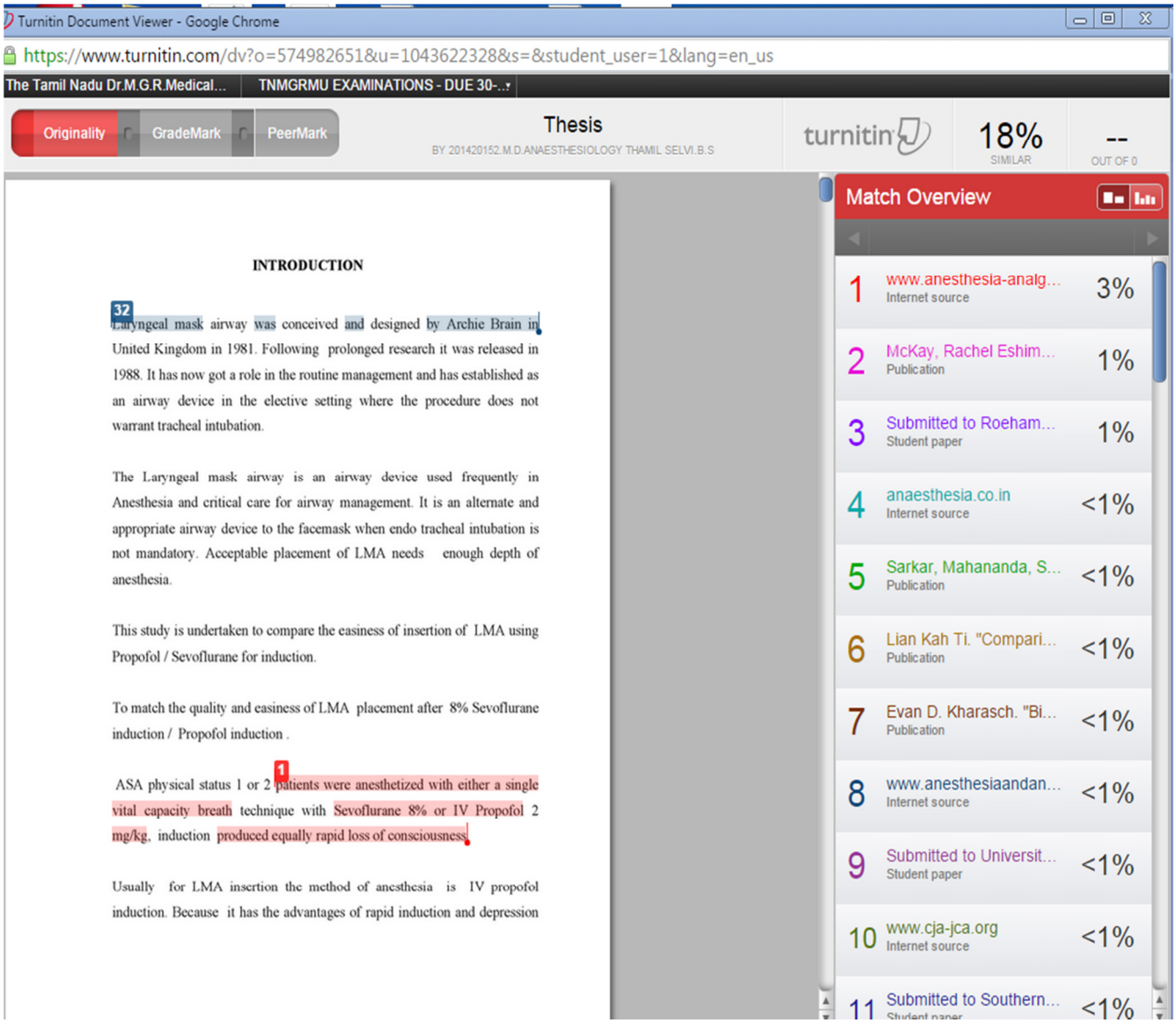
I also thank the theatre personnel for their co-operation and assistance.

I wish to thank all the patients whose willingness and patience made this study possible.

I finally thank God Almighty for His blessings in successfully completing the study.

CONTENTS

PLAGIARISM REPORT



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Author:
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Assignment title:
TNMGRMU EXAMINATIONS

Submission title:
Thesis

File name:
BST Thesis Protocol revised.docx

File size:
624.21K

Page count:
79

Word count:
11610

Character count:
67902

Submission date:
01-Oct-2015 08:34 IST

Submission ID:
574982651

« Page 1 »

INTRODUCTION

Laryngeal mask airway was conceived and designed by Archie Brain in United Kingdom in 1981. Following prolonged research it was released in 1988. It has now got a role in the routine management and has established as an airway device in the elective setting where the procedure does not warrant tracheal intubation.

The Laryngeal mask airway is an airway device used frequently in Anesthesia and critical care for airway management. It is an alternate and appropriate airway device to the facemask when endo tracheal intubation is not mandatory. Acceptable placement of LMA needs enough depth of anesthesia.

This study is undertaken to compare the easiness of insertion of LMA using Propofol / Sevoflurane for induction.

To match the quality and easiness of LMA placement after 8% Sevoflurane induction / Propofol induction.

ASA physical status 1 or 2 patients were anesthetized with either a single vital capacity breath technique with Sevoflurane 8% or IV Propofol 2 mg/kg, induction produced equally rapid loss of consciousness.

Usually for LMA insertion the method of anesthesia is IV propofol induction. Because it has the advantages of rapid induction and depression

Return to assignment list

Comparison of Sevoflurane with Propofol for Laryngeal Mask Airway Insertion in Adults

Abstract

We conducted a prospective, randomized, controlled trial to compare the quality and ease of laryngeal mask airway (LMA) insertion after either rapid inhaled sevoflurane or IV propofol induction of anesthesia.

Placement of the LMA under inhalational anesthesia is not performed universally in adult patients. A famous method of anesthesia for Laryngeal Mask airway placement is with use of intravenous propofol, it has the benefits of inducing anesthesia quickly and depressing reflexes of upper airway. On the other hand, propofol is not ideal agent, it is associated with many side effects like apnea, pain on injection and hypotension. Recently, single VCB technique induction of inhalational sevoflurane is used as an alternate method to intravenous induction of propofol in adult patients. Sevoflurane induction method is quick, with greater acceptance, better hemodynamic profiles and slight excitatory phenomena. Sevoflurane can be used for both maintenance and induction of anesthesia. It made the

conversion period easier. Hence, we compared sevoflurane inhaled induction and propofol IV induction.

After getting the Institutional Ethical Committee approval ,eighty adult patients of American society of Anesthesiologists Physical status 1 &2 of either sex undergoing minor surgical procedures are allocated randomly in to 2 groups, Group A (propofol induction) and Group B (sevoflurane induction).

LMA was inserted more rapidly in propofol group than in patients with sevoflurane group (53.88s vs 80.15s) .There was a greater incidence of difficulty in mouth opening initially in sevoflurane group. Once mouth was possible, the degree of attenuation of laryngeal reflexes was similar. The incidence of complications related to LMA insertion , especially apnoea was more frequent in the propofol group. There were two failures of LMA insertion in sevoflurane group. Both groups had stable hemodynamic parameters. However , prolonged jaw tightness after the sevoflurane inhalational induction may delay LMA insertion.

This study shows no significant difference between the two groups based on the demographic variables. The time to LMA insertion in sevoflurane group was significantly different from propofol.(p value <0.05)

Successful initial mouth opening in sevoflurane group was significantly different from Propofol group.(p value <0.05).The hemodynamic responses were significantly different from Sevoflurane (P value less than 0.05). There was no statistical difference between the two groups in number of attempts for Laryngeal Mask Airway insertion.

We concluded that, even though extended jaw muscle tightness can delay LMA placement in patients with sevoflurane inhalational induction, it can be compared favorably with intravenous induction of propofol .

Key Words:

Propofol, Sevoflurane, LMA Insertion, Jaw relaxation.

INTRODUCTION

Laryngeal mask airway was conceived and designed by Archie Brain in United Kingdom in 1981[13]. Following prolonged research it was released in 1988. It has now got a role in the routine management and has established as an airway device in the elective setting where the procedure does not warrant tracheal intubation.

The Laryngeal mask airway is an airway device used frequently in Anesthesia and critical care for airway management[1]. It is an alternate and appropriate airway device to the facemask when endo tracheal intubation is not mandatory. Acceptable placement of LMA needs enough depth of anesthesia.

This study is undertaken to compare the easiness of insertion of LMA using Propofol / Sevoflurane for induction.

To match the quality and easiness of LMA placement after 8% Sevoflurane induction / Propofol induction[4] .

ASA physical status 1 or 2 patients were anesthetized with either a single vital capacity breath technique with Sevoflurane 8% or IV Propofol 2 mg/kg, induction produced equally rapid loss of consciousness.

Usually for LMA insertion the method of anesthesia is IV propofol induction[14]. Because it has the advantages of rapid induction and depression

of reflexes of upper airway. On the other hand, propofol is not best; it is related with severe side effects like reduction in BP, pain during injection, and apnoea.

In recent times, inhalational induction with sevoflurane using single VCB technique has been used[3,4]. It is an alternate method to intravenous induction in adult patients. This method is rapid, with greater acceptancy, slight excitatory phenomena and better hemodynamic profiles. Laryngeal Mask Airway placement is more rapid after VCB induction using 8% of sevoflurane[19,20] This makes the sevoflurane a sole drug for both maintenance & induction of anesthesia. It will make conversion period easier.

Hence, this study is conducted to compare the consistency, excellence, and time to LMA insertion in adults after using sevoflurane induction and propofol induction

AIM OF THE STUDY

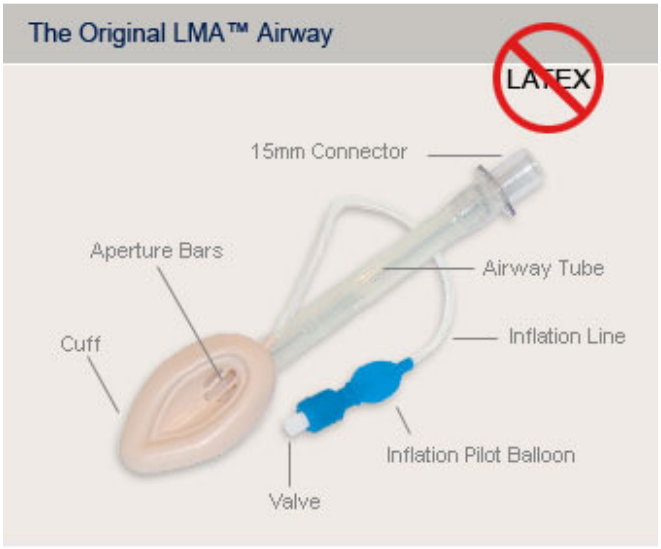
AIM OF THE STUDY

In our study we compare the easiness of Laryngeal Mask Airway insertion using sevoflurane inhalational technique and Propofol intravenous induction technique in patients undergoing elective minor surgical procedures. The following parameters are compared

1. Jaw Relaxation
2. Easiness of insertion
3. Patient movement
4. Coughing, Gagging
5. Laryngospasm
6. Hemodynamic parameters

CLASSIC LMA

CLASSIC LMA



The Classic LMA introduced in the United Kingdom in 1988 and in the United States in 1992. It is a substitutive device to the face mask. The Classic LMA is used in nearly every hospital and has the wide range of sizes from neonates to large adults. The Classic Laryngeal Mask Airway is suitable for elective and day care procedures. It is mostly used in spontaneously breathing patients, but can be used with assisted ventilation up to 20 cm H20.

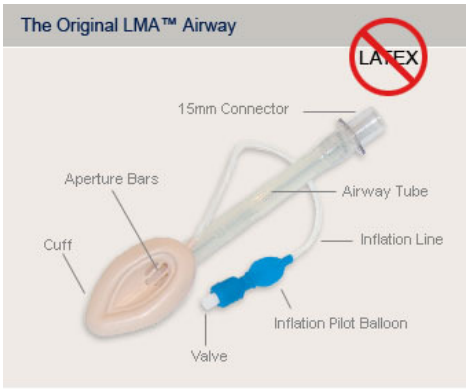
Even though it is called the "routine-use" Laryngeal Mask Airway, it is also used effectively in emergency situations, in patients with difficult airways, adult and peadiatric resuscitation. Laryngeal Mask Airway Unique is a disposable type of Classic Laryngeal Mask Airway.

The LMA is commonly used in position of a face mask or endotracheal tubes during administration of an anesthesia, to provide ventilation and passage of endotracheal tube in a patient with difficult airway, and to support ventilation during FOB.

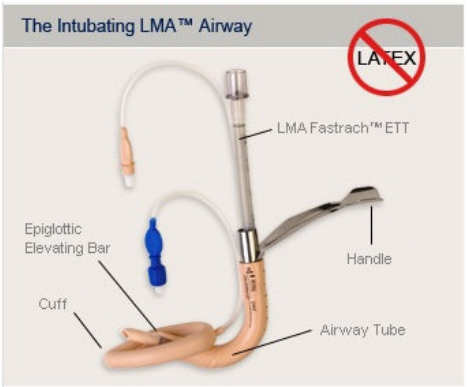
4 types of LMA s are routinely used:

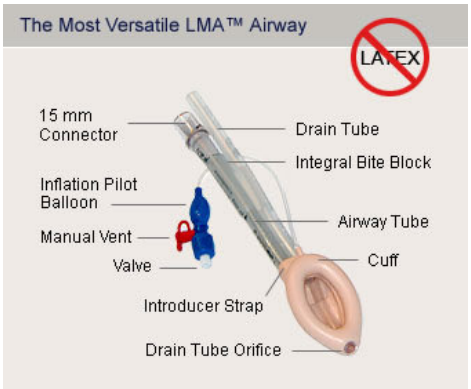
Disposable LMA

Classic LMA



Proseal LMA





Classic Laryngeal Mask Airway

Disposable Laryngeal Mask Airway

Proseal Laryngeal Mask Airway

Fastrach LMA-Intubating LMA

An LMA consists of a wide bore tube whose proximal end connects to a breathing circuit with a standard 15-mm connector and whose distal end is connected to an elliptical cuff that can be inflated through a pilot tube. The deflated cuff is lubricated and inserted blindly in to the hypopharynx so once inflated the cuff forms a low pressure seal around the entrance to the glottis. This requires an anesthetic depth slightly greater than required for the insertion of oral airway.

A correctly placed cuff is bordered superiorly by the base of tongue, laterally by the pyriform sinuses and inferiorly by the upper esophageal sphincter. The LMA partially protects the larynx from pharyngeal secretions but not gastric regurgitation and it should remain in place until the patient has regained airway reflexes.

Advantages;

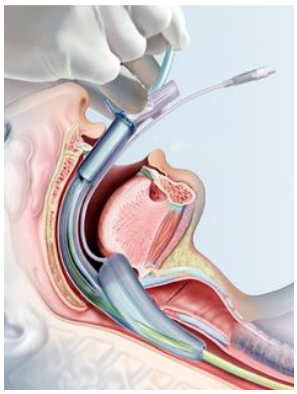
Compared with face mask

- Better seal in bearded patients
- Hands-free operation
- Less facial nerve and eye trauma
- Less cumbersome in ENT operations
- Less operating room pollution
- Often easier to maintain airway

Compared with endotracheal intubation

- Useful in difficult airways
- Compare to endotracheal intubation ,it is minimally invasive
- Less Laryngospasm & bronchospasm
- Minimal dental & laryngeal trauma
- Extensive neck mobility is not necessary
- Risk of Endo bronchial and esophageal intubation is not there
- Does not require muscle relaxation
- Airway tube is clear so that any obstruction can be seen

For successful insertion of LMA depends on following features:



Choose the appropriate size and check for leaks

The foremost edge of the deflated cuff must be wrinkle free and facing away from the aperture

Lubricate the back side of the cuff

Ensure adequate depth of anesthesia before insertion

Place patient head in sniffing position

Use index finger to guide the cuff along the hard palate and down in to the hypopharynx until an increased resistance is felt. The longitudinal black line should always be pointing directly cephalad.

Inflate with the correct amount of air

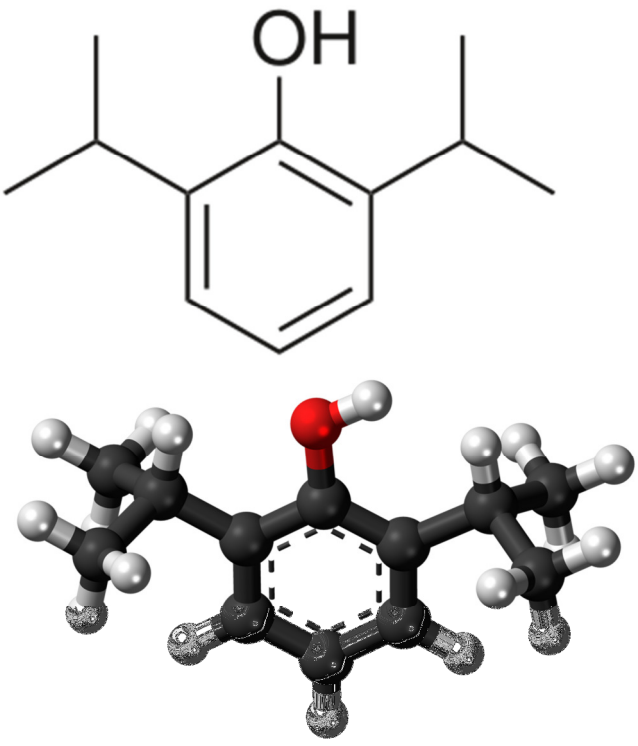
Obstruction after insertion is usually due to down folded epiglottis or transient laryngospasm.

Avoid pharyngeal suction, cuff deflation or LMA removal until the patient is awake.

LMA Size	Patient	Weight(Kg)	Cuff Volume
1	Neonates	<5kg	4ml
1 ½	Infant	5-10kg	4 to 7ml
2	Child	10-20kg	Up to 10ml
2 ½	Child	20-30kg	Up to 15ml
3	Small Adult	30-50kg	Up to 30ml
4	Normal	50-70kg	Up to 40ml
5	Large Adult	70-100kg	Up to 40ml
6	Large Adult	>100kg	Up to 50ml

PROPOFOL

Propofol



Chemical Structure of Propofol

Propofol is a 2,6-diisopropylphenol that is administered intravenously as 1 percent solution in an aqueous solution of ten percent soybean oil, 2.25percent glycerol, and 1.2 percent egg phosphatide. This drug is chemically distinct from other intravenous hypnotic drugs. Rapid IV injection of propofol, 1.5 to 2.5 milligram per kilogram(<15 seconds), yields unconsciousness within thirty seconds. Arousing is very quick and complete than other IV anesthesia induction drugs. The return of consciousness is more rapid, and the most important advantages of propofol are slight residual CNS effects.

Commercial Preparations

Propofol is an insoluble drug that needs a lipid vehicle for emulsification. Current formulations of propofol use soybean oil as the oil phase and egg lecithin act as the emulsifying agent that is made of long chain triglycerides. Diprivan and generic propofol differ with respect to the preservatives used and pH of the formulation.

Mixing of lidocaine with propofol may result in coalescence of oil droplets, which may pose the risk of pulmonary embolism.

A low-lipid emulsion of propofol (Ampofol) contains 5% soybean oil and 0.6% egg lecithin but does not require a preservative or microbial growth retardant. This formulation is equipotent to Diprivan but is associated with a higher incidence of pain on injection.

Mechanism of Action

Propofol is reasonably selective modulator of γ- Amino Butyric Acid (GABAA) receptors, although it has activity at glycine receptors. Propofol is assumed to exert its sedative-hypnotic effects through a Gamma Amino Butyric Acid receptor interaction. Gamma Amino Butyric Acid is the primary inhibitory neuro transmitter in the brain. GABA receptor increases the transmembrane chloride channel conductance that leads to postsynaptic cell membrane hyperpolarization and postsynaptic neuronal function. The propofol interaction with specific components of Gamma Amino Butyric Acid A receptors decreases the inhibitory neurotransmitter dissociation, GABA from the receptor, thereby increasing the GABA-activated opening of the chloride channel duration which results in hyperpolarization of cell membranes.

In contrast to volatile anesthetics, spinal motor neuron excitability, as measured by H reflexes, is not altered by propofol, suggesting that

immobility during propofol anesthesia is not caused by drug-induced spinal cord depression.

Pharmacokinetics

Propofol clearance from the plasma exceeds hepatic blood flow, emphasize that tissue uptake , as well as hepatic oxidative metabolism by cytochrome P450, is vital in drug removal from the plasma .Liver metabolism is quick and wide, results in inactive, metabolites of gucoronic acid excreted by the kidneys. Propofol forms 4-hydroxypropofol by ring hydroxylation by cytochrome P450. The hypnotic activity of 4-hydroxypropofol is about 1/3 of propofol. Other conjugates are inactive. A lesser amount of 0.3% of a dose is excreted unchanged in urine. The elimination half-time is 0.5 to 1.5 hours, but the context-sensitive half-time for propofol infusions lasting up to eight hours is less than 40 minutes. The context-sensitive half-time of propofol is minimally influenced by the length of the administration because of metabolic clearance is rapid. However, when used as a sedative for prolonged intensive care unit (ICU) care, the context-sensitive half-time is highly relevant and should be considered. Propofol, has a short effect-site equilibration time, that the special effects on the brain occur quickly after intravenous administration.

The fact that total clearance of propofol exceeds hepatic blood flow consistent with other than hepatic clearance (lung uptake and first-pass elimination, kidney excretion) of propofol. Pulmonary uptake of propofol is significant and influences the initial availability of propofol. Glucoronidation is the major metabolic pathway for propofol and uridine 5′-diphospho-glucuronosyltransferase isoforms are expressed in the kidneys and brain.

Even though the rapid clearance of propofol by metabolism, in patients with cirrhosis of the liver no impaired elimination. Plasma concentrations of propofol at the time of awakening are similar in alcoholic and normal patients. Clearance of propofol does not influenced by renal dysfunction, despite the observation that nearly three-fourths of propofol metabolites are eliminated in urine in the first 24 hours. Plasma clearance of propofol decreased in old age patients compared with younger ones. Propofol can be administered by continuous infusion because of rapid clearance of this drug without an excessive cumulative effect. Propofol crosses the placenta readily, and cleared from the neonatal circulation.

Clinical Uses

Propofol can be used as the induction agent of choice for many forms of anesthesia, especially in day care procedures. Continuous IV infusion of

propofol, a common method for producing IV “conscious” sedation or as part of a total IV anesthetic. Administration of propofol as a by infusion of continuous technique may be used for patients in the Intensive Care Unit.

Induction of Anesthesia

The induction dose of propofol is 1.5 to 2.5 milligram per kilogram intravenous, blood levels of two to six micro grams per milliliter produced unconsciousness depending on associated medications and the patient’s age. Children require higher induction doses of propofol on a milligram per kilogram basis, presumably reflecting a greater volume of central distribution and clearance rate is high. Elderly patients have decreased clearance rate so they need lower induction dose (25% to 50% decrease) and increased pharmacodynamic activity. Awakening typically occurs at plasma propofol concentrations of 1.0 to 1.5 µg/mL. The characteristic of propofol is complete awakening without residual CNS effects, that makes the propofol as the induction agent of choice.

Intravenous Sedation

Propofol has short context- sensitive half time, combined with the short effect-site equilibration time, makes suitable and titratable drug for IV sedation. No remaining sedation and lower occurance of vomiting makes propofol well suited for ambulatory conscious sedation techniques. The

conscious sedation dose of 25 to 100 microgram per kilogram per min intravenous produces minimal pain-relieving and amnesic things. In selected patients, midazolam or an opioid may be added to propofol for continuous IV sedation. When recovery from conscious sedation, there is sense of wellbeing also. Patient-controlled analgesia delivery system set to deliver 0.7 mg/kg doses of propofol with a 3-minute lockout period has been used as an alternative to continuous IV sedation techniques. Propofol has emerged as the agent of choice for sedation for brief gastrointestinal endoscopy procedures.

Propofol is administered as a sedative drug during mechanical ventilation in the ICU patients and also postoperative patients (cardiac surgery, neurosurgery), head injury patients. Propofol gives control of stress responses and has anti epileptic and amnesia properties. After cardiac surgery, propofol sedation appears to modulate postoperative hemodynamic responses by decreasing the incidence and severity of tachycardia and hypertension. Increasing metabolic acidosis, lipemic plasma, bradycardia, and progressive myocardial failure has been described, particularly in children who were sedated with propofol during management of acute respiratory failure in the ICU.

Maintenance of Anesthesia

The ideal dose of propofol for maintaining anesthesia is 100 to 300 microgram per kilogram per minute intravenously mostly combined with a shorter -acting opioid. General anesthesia with propofol is usually associated with minimal post operative queasiness and vomiting, and awakening is quick, with lesser remaining sedative side effects.

Antiemetic Effects

The incidence of postoperative nausea and vomiting is decreased when propofol is administered, regardless of the anesthetic technique. Propofol (10 to 15 milligram IV) used in the postanesthesia care for treating nausea and vomiting, particularly if it is not of vagal origin. Propofol is generally efficacious in treating postoperative nausea and vomiting at plasma concentrations that do not produce significant sedation. Simulations indicate that antiemetic plasma concentrations of propofol are achieved by a single IV dose of 10 mg followed by 10 μg/kg/minute. Propofol is also used to prevent chemotherapy-induced nausea and vomiting. Propofol is more effective than ondansetron in preventing postoperative nausea and vomiting. Propofol uniformly depresses CNS structures, including subcortical centers. Most drugs of known antiemetic efficacy exert this effect via subcortical structures, and it is possible that propofol modulates subcortical pathways to

inhibit nausea and vomiting or produces a direct depressant effect on the vomiting center.

Antipruritic Effects

Propofol, 10 mg intravenously, is efficient in the management of pruritus related with regional opioids and cholestasis. The mechanism effect may be related to the drug's ability to depress spinal cord activity. In this regard, there is evidence that intrathecal opioids produce pruritus by segmental excitation within the spinal cord.

Anticonvulsant Activity

Propol has anticonvulsant properties, by presynaptic and postsynaptic inhibition of chloride ion channels mediated by GABA. In this view, propofol in doses of greater than 1 mg/kg IV decreases seizure duration 35% to 45% in patients undergoing electroconvulsive therapy.

Attenuation of Bronchoconstriction

Propofol decreases the prevalence of wheezing after induction of anesthesia and tracheal intubation in healthy and asthmatic patients However, a newer formulation of propofol uses metabisulfite as a preservative. Metabisulfite may cause bronchoconstriction in asthmatic patients. Propofol having metabisulfite produces airway resistance in patients with history of smoking following tracheal intubation than ethylene

diamino tetra acetic acid (EDTA). Therefore, the preservative used for propofol can have effects on its ability to attenuate bronchoconstriction.

Effects on Organ Systems

CNS

Propofol reduces (CMRO2), cerebral blood flow, and intracranial pressure. Propofol does not raise intracranial pressure in patients with space -occupying lesions to produce sedation. However, large dose of propofol reduces blood pressure and cerebral perfusion pressure. Cerebrovascular autoregulation in response to changes in systemic blood pressure and reactivity of the cerebral blood flow to changes in PaCO2 are not affected by propofol. Propofol produces cortical electroencephalographic (EEG) changes that are similar to those of thiopental, including the ability of high doses to produce burst suppression. Propofol does not interfere with the adequacy of electrocorticographic recordings during awake craniotomy performed for the management of refractory epilepsy, provided administration is discontinued at least 15 minutes before recordings.

Cardiovascular System

Propofol produces decreases in systemic BP, these decreases in BP is due to reduced cardiac output and systemic vascular resistance. The relaxation of vascular smooth muscle produced by propofol is primarily due

to inhibition of sympathetic vasoconstrictor nerve activity. A negative inotropic effect of propofol produces negative inotropic activity by reducing intracellular calcium availability and inhibition of trans-sarcolemmal calcium influx. Stimulation produced by direct laryngoscopy and intubation of the trachea reverses the blood pressure effects of propofol. Propofol also effectively blunts the hypertensive response to placement of a laryngeal mask airway.

Propofol effects on blood pressure are increased in patients of hypotension, old age persons and compromised left ventricular function. To prevent this exaggerated effect, adequate hydration must be insisted before rapid IV administration of propofol. Even though there is blood pressure reduction, no change in pulse rate. However, asystole and bradycardia have observed after induction of propofol, results in occasional suggestion that anti cholinergic agents be administer while parasympathetic stimulation is possible to occur in association with administration of propofol.

Propofol may reduce sympathetic nervous system activity to a larger extent than para sympathetic nervous system activity, results in a majority of para sympathetic activity. Propofol drug does not alter sinoatrial or atrioventricular node function in normal patients or in patients with Wolff-

Parkinson-White syndrome, thus making it an acceptable drug to administer during ablative procedures.

Bradycardia-Related Death

Profound bradycardia and asystole after administration of propofol have been described in healthy adult patients, despite prophylactic anti cholinergics. The risk has been estimated to be 1.4 in 100,000. Propofol anesthesia, compared with other anesthetics, increases the incidence of the oculocardiac reflex in pediatric strabismus surgery, despite prior administration of anti cholinergics.

Heart rate responses to IV administration of atropine are attenuated in patients receiving propofol compared with awake patients, this decreased responsiveness to atropine cannot be effectively overcome by larger doses of atropine suggesting that propofol may induce suppression of sympathetic nervous system activity. Treatment of propofol-induced bradycardia may require treatment with a direct β agonist such as isoproterenol.

Lungs

Propofol produces ventilatory depression, with apnea occurs in 25% to 35% patients after anesthesia induction. Opioids enhance ventilatory depressive effect of propofol. A maintenance dose of propofol infusion reduces breath rate and tidal volume. The ventilatory response to arterial

hypoxemia is also decreased by propofol due to an effect at the central chemo receptors. Propofol maintains the effect of Hypoxic Pulmonary Vasoconstriction.

Liver and Kidney Function

Propofol is not normally affect Liver and kidney function as reflected by measurements of liver transaminase enzymes or creatinine concentrations. Prolonged infusions of propofol have been associated with hepatic cellular injury accompanied by lactic acidosis, brady arrhythmias, and rhabdomyolysis as part of the propofol infusion syndrome. Presence of phenols causes excretion of green urine, reflecting the presence of phenols that indicates long term administration of propofol.

Intraocular Pressure

Laparoscopic surgery is associated with increased intraocular pressure and some consider laparoscopic surgery with the head down position a risk in the presence of preexisting ocular hypertension. In this regard, propofol is associated with significant decreases in intraocular pressure that occur immediately after induction of anesthesia and are sustained during tracheal intubation. Total IV anesthesia with propofol for laparoscopic surgery was associated with lower intraocular pressures.

Other Side Effects

Side effects of propofol may reflect the parent drug or actions attributed to the oil-in-water emulsion formulation. For example, some of the side effects of propofol (bradycardia, pain during injection, risk of infection, increased triglyceride levels with prolonged administration, potential risk for pulmonary embolism) are due to in huge part to the lipid emulsion formulation.

Allergic Reactions

The phenyl nucleus and di isopropyl side chain are allergic components of propofol. The diisopropyl radical, is present in most of dermatologic preparations. Likewise, the phenol nucleus is common to many drugs. Indeed, anaphylaxis to propofol during the first exposure to this drug has been observed, especially in patients with a history of other drug allergies, often to neuromuscular blocking drugs. Propofol-induced bronchoconstriction has been described in patients with allergy histories.

Lactic Acidosis

Lactic acidosis is known as propofol infusion syndrome has been described in adult and pediatric patients receiving long term large-dose infusions of propofol (greater than 75 microgram per kilogram per minute) for more than 24 hours. Severe, refractory, bradycardia in children in the

ICU has been observed with long-term propofol sedation. Unexpected tachycardia occurring during propofol anesthesia should prompt laboratory evaluation for possible metabolic acidosis. Measurement of arterial blood gases and serum lactate concentrations is recommended. Metabolic acidosis in its early stages is reversible with discontinuation of propofol administration.

The differential diagnosis when propofol-induced lactic acidosis is suspected includes hyperchloremic metabolic acidosis associated with large volume infusions of 0.9% saline and metabolic acidosis associated with excessive generation of organic acids, such as lactate and ketones (diabetic acidosis, release of a tourniquet). Measurement of the anion gap and individual measurements of anions and organic acids will differentiate hyperchloremic metabolic acidosis from lactic acidosis.

Pro convulsion Activity

The greater number of propofol-induced "seizures" at the time of induction and emergence from anesthesia reveal spontaneous excitatory movements of subcortical source. These responses are not thought to be due to cortical epileptic activity.

Abuse Potential

Extreme dreaming activity, sentimental behavior, and hallucinations have reported in the recovery from low-dose infusions of propofol. Addiction to virtually all opioids and hypnotics, including propofol, has been described.

Bacterial Growth

Propofol robustly supports the expansion of Escherichia coli bacteria & Pseudomonas aeruginosa. Clusters of postoperative surgical infections manifesting as temperature elevations have been attributed to extrinsic contamination of propofol. For this reason, it is recommended that (a) an aseptic technique be used in handling propofol as reflected by disinfecting the ampule neck surface or vial rubber stopper with 70% isopropyl alcohol; (b) the contents of the ampule containing propofol should be withdrawn into a sterile syringe immediately after opening and administered promptly; and (c) the contents of an opened ampule must be discarded if they are not used within 6 hours. In the ICU, the tubing and any unused portion of propofol must be discarded after 12 hours.

Antioxidant Properties

Propofol has potent antioxidant properties that resemble those of the endogenous antioxidant vitamin E. Like vitamin E, propofol contains a

phenolic hydroxyl group that scavenges free radicals and inhibits lipid peroxidation. A neuroprotective effect of propofol may be at least partially related to the antioxidant potential of propofol's phenol ring structure. For example, propofol reacts with lipid peroxyl radicals and thus inhibits lipid peroxidation by forming relatively stable propofol phenoxyl radicals.

Reintroduction of molecular oxygen into formerly ischemic tissues (removal of an aortic cross-clamp) can damage partially injured cells. O2 leads to the production of free O2 radicals, which react with poly unsaturated fatty acids of cell membranes resulting in disruption of cell membranes. Cardiac cell injury can cause postischemic dysfunction, cardiac stunning, and reperfusion cardiac dysrhythmias. Propofol strongly attenuates lipid peroxidation in CABG.

Pain on Injection

Pain on injection is the most commonly reported adverse event associated with propofol administration to awaked patients. This unpleasant side effect of propofol occurs in less than 10% of patients when the drug is injected into a large vein rather than a dorsum vein on the hand. Preceding the propofol with (using the same injection site as for propofol) 1% lidocaine or by prior administration of a potent short-acting opioid decreases the incidence of discomfort experienced by the patient. The incidence of

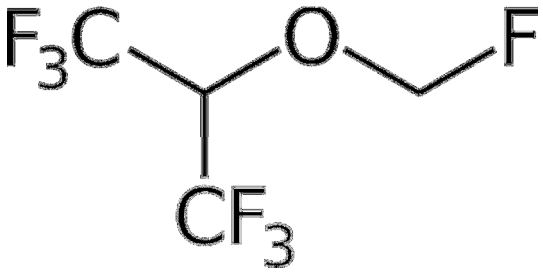
thrombosis or phlebitis is usually less than 1%. Changing the composition of the carrier fat emulsion for propofol to long and medium chain triglycerides decreases the incidence of pain on injection.

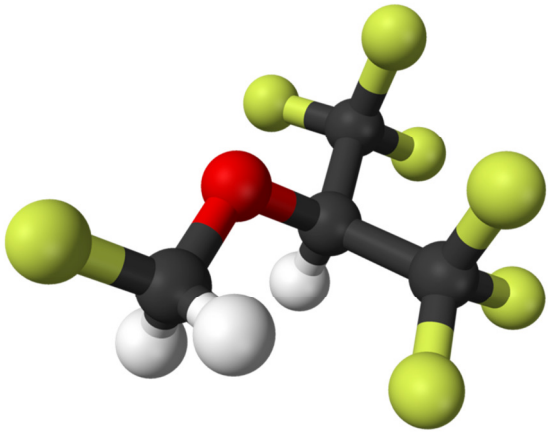
Miscellaneous Effects

Propofol can be administered in patients of hereditary copro porphyria. Because it will not trigger malignant hyperthermia.

SEVOFLURANE

Sevoflurane





Chemical structure of Sevoflurane

Sevoflurane is a fluorinated methyl isopropyl ether. The sevoflurane vapor pressure resembles that of halothane and isoflurane, permitting delivery of this anesthetic via a conventional unheated vaporizer. The solubility of sevoflurane (blood:gas partition coefficient 0.69) mimics that of desflurane, ensuring punctual anesthesia induction and recovery after anesthesia discontinuation. Compared with isoflurane, recovery from sevoflurane anesthesia is 3 to 4 minutes faster and the difference is magnified in longer duration surgical procedures (>3 hours) Sevoflurane is non pungent, has lesser odor, produces bronchodilation parallel in degree to isoflurane, and produces the smallest amount degree of airway irritation amongst the presently available inhaled anesthetics. For these reasons, sevoflurane, like halothane, is acceptable for inhalation induction of anesthesia.

Sevoflurane may be 100-fold more vulnerable to metabolism than desflurane, with an estimated 3% to 5% of the dose undergoing biodegradation. The resulting metabolites include inorganic fluoride (plasma concentrations exceed those that occur after enflurane) and hexafluoroisopropanol. The chemical structure of sevoflurane is such that it cannot undergo metabolism to an acyl halide. Sevoflurane metabolism does not result in the formation of trifluoroacetylated liver proteins and therefore

cannot stimulate the formation of antitrifluoroacetylated protein antibodies. In this regard, sevoflurane differs from halothane, enflurane, isoflurane, and desflurane, all of which are metabolized to reactive acyl halide intermediates with the potential to produce hepatotoxicity as well as cross-sensitivity between drugs. Sevoflurane is the least likely volatile anesthetic to form carbon monoxide on exposure to carbon dioxide absorbents. In contrast to other volatile anesthetics, sevoflurane breaks down in the presence of the strong bases present in carbon dioxide absorbents to form compounds that are toxic in animals The principal degradation product is fluoromethyl-2, 2-difluoro-1-(trifluoromethyl) vinyl-ether (compound A). Compound A is dose dependent renotoxin in rats, causing renal proximal tubular injury.Eventhough this finding is a concern, the levels of these compounds (principally compound A) that occur during administration of sevoflurane to patients are far below speculated toxic levels, even when total gas flows are 1 L per minute.

Pharmacokinetics of Volatile agents

The pharmacokinetics of inhaled anesthetics described by (1) Absorption from alveoli into pulmonary capillary blood, (2) Distribution of drug (3) metabolism of drug, (4) Excretion of drug mostly through pulmonary system. The pharmacokinetics of volatile anesthetics may be

influenced by aging, reflecting decreases in lean body mass and increases in body fat. The volume of distribution (Vd) of the central compartment (plasma volume) is smaller, whereas the apparent Vd (steady state) for these drugs in the elderly is larger, especially for those anesthetics most soluble in fat. In addition, impaired pulmonary gas exchange may decrease anesthetic clearance with age. Furthermore, reduced cardiac output in the elderly decreases tissue perfusion, increases time constants, and may be associated with an altered regional distribution of anesthetics. Opposite effects on the pharmacokinetics of inhaled anesthetics might be expected in the very young.

A sequence of partial pressure gradients starting from the anesthesia machine serve to drive the volatile anesthetic crosswise different levels (alveoli, capillaries, cell membranes) to sites of action in the Central Nervous System. The primary purpose of volatile is to attain a steady & best possible partial pressure of brain of the volatile anesthetic drug.

The CNS and other tissues equilibrate with the partial pressures of volatile anesthetics deliver to them by arterial blood (Pa). Likewise, the alveolar partial pressures (PA) equilibrate with arterial blood of anesthetics. This emphasizes that the PA of inhaled anesthetics mimics the partial pressure of brain (PBRAIN) at steady state. This is the reason that PA is

used as an index of (1) intensity, (2) recovery from anesthesia, and (3) MAC value. This is essential to identify that the same partial pressure exists in both phases that means the equilibration. Equilibration is not denoted equal opportunity of concentrations in 2 bio phases.

Understanding these factors that conclude the Partial pressure of alveoli and thus the Partial pressure of brain allows control of the volatile anesthetics delivered to brain so as to sustain a steady and optimal anesthesia depth. This relationship is applicable because volatile anesthetics are only minimally metabolized and as such are excreted from the lung. The availability of an "online" readout of end-tidal partial pressure, which at equilibrium matches brain partial pressure, makes volatile anesthetic dosing easier than intravenous anesthetic dosing.

Determinants of Partial Pressure of Alveoli

The partial pressure of alveoli and ultimately the partial pressure of brain of inhaled anesthetics are determined by input into the alveoli minus uptake the drug from the alveoli into the arterial blood. Input of anesthetics into alveoli depends on the (a) inhaled partial pressure (PI), (b) ventilation of alveoli, (c) characteristics of the anesthetia delivery. Uptake of volatile anesthetics from the alveoli into the pulmonary capillary blood depends on (a) anesthetic solubility in tissues, (b) CO and (c) A-v differences (A-vD).

Inhaled Partial pressure

A high PI delivered from anesthesia machine is required in the initial management of anesthetic. A high initial enter offsets the force of uptake, accelerating anesthesia induction as reflected by the speed of rise in the PA and thus the PBRAIN. By time, as uptake into the blood reduces, the PI should reduce to equal the reduced uptake of anesthesia and therefore maintain a regular and optimal PBRAIN. If the PI is maintained regular with time, the PA and PBRAIN will increase progressively as uptake diminishes.

Concentration Effect

The impact of PI on the speed of increase of the PA of an inhaled anesthetic is called as the concentration effect. The concentration effect states that the higher the PI, the more rapidly the PA approaches the PI. The higher PI provides anesthetic molecule input to offset uptake and thus speeds the rate at which the PA increases.

The concentration effect results from (a) a concentrating effect and (b) an augmentation of tracheal inflow. The concentrating effect reflects absorption of the inhaled anesthetic in a lesser lung volume due to uptake of all gases in the lung. At the same time, anesthetic input via tracheal inflow is increased to fill the space (void) produced by uptake of gases.

Second-Gas Effect

The second-gas effect reflects the capability of large-volume uptake of first gas to speed up the rate of increase of the alveolar partial pressure of a concomitantly administered "companion" gas. For instance, the primary huge uptake of N2O increases the uptakes of accompanied (second) gases such as oxygen and inhaled anesthetics. This augmented uptake of the 2nd gas reflects raised tracheal flow of all the volatile gases (1st and 2nd gases) and more absorption of 2nd gas or gases in a lesser lung volume (concentrating effect) due to large uptake of initial gas volume.

Alveolar Ventilation

Raised ventilation of alveoli like inspired pressure, promote entry of anesthetics to counteract the uptake. Total effect is quick speed of raise in the PA toward the PI and thus induction of anesthesia. In addition to the increased input, the decreased PaCO2 produced by hyperventilation of the lungs decreases cerebral blood flow. Possibly, the impact of augmented entry on the speed of rise of PA would be counteracting by reduced anesthesia delivery of drug to the Central Nervous System. Decreased alveolar ventilation decreases input and thus slows the establishment of a PA and PBRAIN necessary for the induction of anesthesia. The larger the ventilation of alveoli to functional residual capacity ratio, the more fast is the

speed of increase in the alveolar partial pressure. In neonates, this ratio is approximately 5:1 compared with only 1.5:1 in adults, reflecting the greater metabolic rate in neonates compared with adults. As a result, the rate of increase of PA toward the PI and thus the induction of anesthesia is more rapid in neonates than in adults

Impact of Solubility

The impact of changes in ventilation of alveoli on the speed of raise in the alveolar partial pressure toward the PI depends on the anesthetic solubility in blood. For example, changes in alveolar ventilation influence the speed of increase of the alveolar partial pressure of a soluble anesthetic (halothane, isoflurane) more than a poorly soluble anesthetic (nitrous oxide, desflurane, sevoflurane). Indeed, the speed of increase in the alveolar partial pressure of nitrous oxide is rapid regardless of alveolar ventilation. This occurs because uptake of nitrous oxide is limited because of its poor solubility in blood. Conversely, uptake of a more blood-soluble anesthetic is larger, and increasing alveolar ventilation will accelerate the rate at which the PA of the soluble anesthetic approaches the PI. This emphasizes that changing from spontaneous breathing to mechanical (controlled) ventilation of the lungs, which also is likely to be associated with increased alveolar

ventilation, will probably increase the depth of anesthesia (PA) produced by a more blood-soluble anesthetic.

Anesthetic Breathing System

Breathing system of anesthesia characteristics manipulate the speed of raise of PA are (1) External breathing system volume, (2) Volatile anesthetic solubility , solubility in the plastic and rubber parts of the breathing system, and (3) entry of gas from anesthetic delivery system. Anesthetic delivery system volume acts like a barrier to sluggish down the achievement of the alveolar partial pressure. Large gas flow (5 to 10 L per minute) from the anesthetic delivery system reverse buffer effect. Volatile agent solubility in the components of the breathing system of anesthesia primarily reduces speed at which the PA increases. At termination of the anesthesia administration, however, reverse of the partial pressure slope in breathing system of anesthesia resulted in elution of the anesthetic, which slow down the speed at the PA decreases.

Solubility

The drug solubility in tissue and blood is reflected by the partition coefficient. A partition coefficient is allocation share describes how volatile anesthetic allocates itself among 2 phases at the equilibrium (partial pressures equal in both phases). For example, a blood: gas partition

coefficient of 0.5 denotes that the concentration of volatile anesthetic in blood is half that present in the alveolar gases when partial pressures of the anesthetic in these 2 phases is indistinguishable. Similarly, a brain: blood partition coefficient of 2 indicates a concentration of anesthetic in the brain is twice that in the blood when partial pressures of anesthetic are indistinguishable at both sites.

Partition coefficients may be thought of as reflecting the relative capacity of each phase to accept anesthetic. Partition coefficients are indirectly proportional to temperature that is gas solubility in the liquid is decreased while temperature of liquid increases.

Coefficients of Blood –Gas partition

Speed of raise of the PA toward the PI (maintained constant by mechanical ventilation of the lungs) is inversely related to the solubility of the drug in blood. Based on their blood: gas partition coefficients, inhaled anesthetics are categorized traditionally as soluble, intermediately soluble, and poorly soluble. Blood could be measured the pharmacologically inactive reservoir, the size is determined by drug solubility in blood. While blood: gas partition coefficient is elevated, a huge quantity of anesthetic should dissolved in blood earlier than the arterial pressure equilibrates with alveolar partial pressure. For example, the high blood solubility of methoxyflurane

slows the rate at which the PA and Pa increase relative to the PI, and the induction of anesthesia is slow. The impact of large solubility of blood on the speed of augment of Pa can be counterbalance to few degrees by escalating the inspired partial pressure above that required for maintenance of anesthesia. This is termed the overpressure technique and may be used to speed the induction of anesthesia, recognizing that sustained delivery of a high PI will result in an anesthetic overdose.

When blood solubility is small, least amounts of inhaled anesthetic must dissolved prior to equilibration is achieved; therefore, the speed of raise of PA and Pa, and thus onset-of-drug effects such as the induction of anesthesia, are rapid. For example, the inhalation of a constant PI of nitrous oxide, desflurane, or sevoflurane for about 10 minutes results in a PA that is ≥80% of the PI. Use of an overpressure technique with sevoflurane is more readily accepted by patients because this anesthetic is less pungent than desflurane. Indeed, one or more vital capacity breaths of high concentrations of sevoflurane (7% with 66% nitrous oxide) may result in loss of the eyelash reflex.

Coefficients of Tissue- Blood partition

The Tissue:blood partition coefficients conclude uptake of anesthetic into tissues and the time taken for equilibration of tissues with the partial

pressure of arterial blood. This time for equilibration could be estimated by calculate the time constant (quantity of volatile agent dissolved in tissue divided by tissue blood flow) for every tissue. One time constant on an exponential curve represents 63% equilibration. Three time constants are equivalent to 95% equilibration. For volatile anesthetics, equilibration between the Pa and PBRAIN depends on the anesthetic's blood solubility and requires 5 to 15 minutes (three time constants). Fat has an enormous capacity to hold anesthetic, and this characteristic, combined with low blood flow to this tissue, prolongs the time required to narrow anesthetic partial pressure differences between arterial blood and fat. For example, equilibration of fat with isoflurane (three time constants) based on this drug's fat:blood partition coefficient and an assumed fat blood flow of 2 to 3 mL per minute per 100 g fat is estimated to be 25 to 46 hours. Fasting before elective operations results in transport of fat to the liver, this could increase anesthetic uptake by this organ and modestly slow the rate of increase in the PA of a volatile anesthetics during anesthesia induction.

Anesthesia recovery

Anesthesia recovery is depicted by the rate of decrease in the PBRAIN as reflected by the PA the rate of washout of anesthetic from the brain should be rapid because inhaled anesthetics are not highly soluble in

brain and the brain receives a large fraction of the cardiac output. Although similarities exist between the rate of induction and recovery, as reflected by changes in the PA of the inhaled anesthetic, there are important differences between the two events. In contrast to induction of anesthesia, which may be accelerated by the concentration effect, it is not possible to speed the decrease in PA by this mechanism.

Furthermore, at the conclusion of every anesthetic, the concentration of the volatile agent in tissues depending highly on solubility of the inhaled drug and the duration of its administration. This contrasts with tissue concentrations of zero at the initiation of induction of anesthesia. The failure of certain tissues to reach equilibrium with the PA of the inhaled anesthetic during maintenance of anesthesia means that the rate of decrease of the PA during recovery from anesthesia will be more rapid than the speed of increase of the alveolar partial pressure at the time of anesthesia induction. . Indeed, even after a prolonged anesthetic, skeletal muscles probably, and fat almost certainly, will not have equilibrated with the PA of the inhaled anesthetic.

Thus, when the PI of an anesthetic is abruptly decreased to zero at the conclusion of an anesthetic, these tissues initially cannot contribute to the transfer of drug back to blood for delivery to the liver for metabolism or to

the lungs for exhalation. As long as gradients exist between the Pa and tissues, the tissues will continue to take up anesthetic. Thus, during recovery from anesthesia, the continued passage of anesthetic from blood to tissues, such as fat, acts to speed the rate of decrease in the PA of that anesthetic. Continued tissue uptake of anesthetic will depend on the solubility of the inhaled anesthetic and the duration of anesthesia, with the impact being most important with soluble anesthetics. For soluble anesthetics recovery from anesthesia is prolonged, which is directly proportional to duration of drug delivery, whereas for poorly soluble anesthetics like sevoflurane recovery time is less.

Context-Sensitive Half-Time

Elimination of volatile anesthetics depends on length of anesthesia administration and blood-gas solubility of the volatile anesthetic. As with propofol, this is possible to apply computer simulation to conclude context-sensitive half-time for inhaled agents. In this regard, time needs for fifty percent reduce in anesthetic concentration of sevoflurane is < five min. and this not rise considerably with escalating length of anesthesia. It would seem, it is the indication of the primary phase excretion which is a role of alveolar ventilation.

Pharmacodynamics --Inhaled Anesthetics

Minimal Alveolar Concentration

MAC of an inhaled anesthetic is defined as that concentration at 1 atmosphere that prevents skeletal muscle movement in response to a supramaximal painful stimulus (surgical skin incision) in 50% of patients. MAC is an anesthetic 50% effective dose (ED50). Immobility produced by inhaled anesthetics as measured by MAC is mediated principally by effects of these drugs on the spinal cord and only a minor component of immobility results from cerebral effects.

Central Nervous System Effects

Cerebral metabolic oxygen requirements are decreased in parallel with drug-induced decreases in cerebral activity. Drug-induced increases in cerebral blood flow may increase intracranial pressure (ICP) in patients with space-occupying lesions. The effects of desflurane and sevoflurane on the CNS do not differentiate these inhaled anesthetics from the older inhaled drugs.

Seizure Activity

Sevoflurane like isoflurane, do not produce evidence of convulsive activity on the EEG either at deep levels of anesthesia or in the presence of hypocapnia or auditory stimulation. Nevertheless, there are reports of

pediatric patients with epilepsy and otherwise healthy adults who developed EEG evidence of seizure activity during sevoflurane anesthesia. Sevoflurane can suppress convulsive activity induced with lidocaine.

Circulatory Effects

Drug-induced circulatory effects manifest as changes in heart rate, blood pressure, stroke volume, cardiac output, systemic vascular resistance, right atrial pressure cardiac rhythm, and coronary blood flow. Circulatory changes of inhaled anesthetics may be dissimilar in the presence of (a) mechanical ventilation of the lungs compared with spontaneous breathing, (b) preexisting cardiac disorders, or (c) indirect and directly acting drugs on the heart. The circulatory effects of mechanism are diverse often reflect the special effects of volatile anesthetics on (a)cardiac contractility, (b) peripheral vascular resistance, and (c) activity of autonomic nervous system.

Mean Arterial Pressure.

Sevoflurane produce dose-dependent decreases in mean arterial pressure. Sevoflurane increases heart rate only at concentrations of >1.5 MAC, whereas isoflurane and desflurane tend to increase heart rate at lower concentrations.

Cardiac Output and Stroke Volume

Sevoflurane, produces dose-dependent decreases in cardiac output. Sevoflurane did decrease cardiac output at 1 and 1.5 MAC, but at 2 MAC cardiac output had recovered to nearly awake values.

Mechanisms of Circulatory Effects

There is no known single mechanism that explains the cardiovascular depressant effects of volatile anesthetics, just as there is none for the neurobehavioral effects. Proposed mechanisms include (a) direct myocardial depression, (b) inhibition of CNS sympathetic activity, (c) peripheral autonomic ganglion blockade, (d) attenuation of carotid sinus reflex, (e) reduced cyclic adenosine monophosphate, (f) reduced catecholamine release and (g) reduction of calcium ions influx in the course of slow channels. Indeed, negative inotropic, vasodilating, and depressant effects on the sinoatrial node produced by volatile anesthetics are similar to the effects produced by calcium entry blockers.

Ventilation Effects

Inhaled anesthetics produce dose-dependent and drug-specific effects on the (a) pattern of breathing, (b) ventilatory response to carbon dioxide, (c) ventilatory response to arterial hypoxemia, and (d) airway resistance. The

PaO2 predictably declines during administration of inhaled anesthetics in the absence of supplemental oxygen.

Sevoflurane Produce dose-dependent increases in the frequency of breathing. Tidal volume is decreased in association with anesthetic-induced increases in the frequency of breathing. The net effect of these changes is a rapid and shallow pattern of breathing during general anesthesia.

Ventilatory Response to Carbon Dioxide

Volatile anesthetics produce dose-dependent depression of ventilation characterized by reduced hypercarbic drive to the ventilation and increases in the PaCO2

Mechanism of Depression

The ventilatory depression associated with sevoflurane may result from a combination of central depression of medullary inspiratory neurons and depression of diaphragmatic function and contractility.

Ventilatory Response to Hypoxemia

Sevoflurane is useful during thoracic surgery as it is a potent bronchodilator, its low blood-gas solubility permits rapid adjustment of the depth of anesthesia, and effects on hypoxic pulmonary vasoconstriction are small.

Airway Resistance and Irritability

Sevoflurane decreases airway resistance as much or more than isoflurane. Sevoflurane have been administered without evidence of bronchospasm to patients with bronchial asthma.

Hepatic Effects

Drug Clearance

Volatile anesthetics may interfere with clearance of drugs from the plasma as a result of decreases in hepatic blood flow or inhibition of drug-metabolizing enzymes.

Sevoflurane

Compound A, a product of sevoflurane interaction with carbon dioxide absorbents, is hepatotoxic in animals, but the concentration present in the anesthesia breathing circuit is far below the toxic level in animals.

Renal Effects

Volatile anesthetics produce similar dose-related decreases in renal blood flow, glomerular filtration rate, and urine output.

Vinyl Halide Nephrotoxicity

Potassium and sodium hydroxide containing co2 absorbents react with sevoflurane and eliminate hydrogen fluoride from its isopropyl moiety to form breakdown products. The degradation product produced in greatest

amounts is fluoromethyl-2,2-difluro-1-(trifluoromethyl) vinyl ether (compound A). During closed-circuit anesthesia with sevoflurane administered to patients undergoing operations lasting longer than 5 hours, the average concentration of compound A in the anesthesia circuit was <20 ppm and no evidence of renal dysfunction occurred based on measurements of blood urea nitrogen and plasma creatinine concentrations. Higher concentrations of compound A occurred in the presence of Baralyme (no longer clinically available) probably as a result of higher absorbent temperatures compared with soda lime. Similarly, carbon dioxide production increases the absorbent temperature and thus the production of compound A. Probenecid is a selective inhibitor of organic anion transport and pretreatment with this drug prevents compound A–induced renal injury in rats and may provide similar protection in humans.

Malignant Hyperthermia

All volatile anesthetics including desflurane and sevoflurane could cause malignant hyperthermia in genetically vulnerable patients even in absence of concomitant administration of succinylcholine.

Sevoflurane Vaporizer

REVIEW

OF

LITERATURE

Review of literature

- Propofol is an intravenous induction agent which has a rapid onset of action with good relaxation properties. It is administered as a 1% solution. Administration of 1.5-2.5 mg/kg intravenously produces unconsciousness within 30 seconds. The rapid induction and rapid return of consciousness with minimal residual effects are the most important advantages of propofol.
- The action of propofol is mediated by enhancing γ-aminobutyric acid (GABA)–induced chloride current through binding to the β-subunit of $GABA_A$ receptor leading to depressant action on the Central Nervous System.
- Propofol reduces systolic blood pressure by depression of the baroreceptor reflexes. Propofol produces dose dependent depression of ventilation with apnea in 25%-35% of patients. Propofol blunts the hypertensive response during insertion of laryngeal mask airway.
- Propofol has the advantages of anti-emetic, anti-convulsant and amnestic properties also. The common adverse effects are apnea, hypotension and pain on injection.

- Laryngeal mask airway (LMA) insertion is rapid in patients with Propofol induction. It has the jaw muscles relaxant effect and it prevents the laryngeal reflexes during placement of the Laryngeal mask airway.
- Sevoflurane is an inhalational anesthetic agent. With a blood gas partition co-efficient of 0.69% and minimum alveolar concentration of 2.1,it ensures rapid induction and rapid recovery after discontinuation of anesthesia.
- Sevoflurane causes least degree of airway irritation amongst the other volatile anesthetics and has smooth conversion to maintenance phase without apnea. It is also considered safe in adult patients because of lack of nephrotoxicity and minimal metabolism.
- Sevoflurane associated with delayed jaw muscle relaxation and may take a longer time for insertion of laryngeal mask airway. On the other hand, it has better hemodynamic profile and can be used in high risk patients.
- **TiLK, Chow Mark YH et.al** conducted a study titled "Comparison of sevoflurane with propofol for laryngeal mask airway insertion in adults"The study population consisted of 76 un premedicated American society of Anaesthesiologists physical status1/,2 patients who are anaesthetized with either sevoflurane 8% by using single vital breath capacity or i.v induction of propofol 3mg/.kg. They noted that

sevoflurane induces quick loss of consciousness (40.5±13.9s vs. 37.7±9.9s: p value greater than 0.05) but laryngeal mask airway was inserted more quickly in patients with propofol induction (74±29s vs. 127±35s: p value less than 0.01) and needs lesser attempts than the sevoflurane group. Both the groups have stable haemodynamic properties & noble patient gratification. They concluded that the sevoflurane 8% VCB induction compares favourably with i.v induction of Laryngeal mask airway placement in adults, although sustained jaw tightness may delay LMA placement.[1]

- **Molloy ME, Buggy DJ et.al (1999) :** conducted a study titled "Propofol or sevoflurane for laryngeal mask airway insertion". The study population consisted of eighty eight patients of American Society of Anesthesiologists I or II underwent general anaesthesia for the elective surgeries allocated into 2 groups. Patients in Propofol group (n=44) received 2.5mg/kg propofol intravenously and in Sevoflurane group (n=44) received sevoflurane 8% in N2O 50% and O2 50%. LMA placement is attempted at 1 min interval from loss of eyelash reflex. The mean time to successful laryngeal mask airway placement is 1.3 minutes in propofol group, 2.2 minutes in Sevoflurane group. They noted that complications were similar in both groups. They concluded that,

modified vital capacity breath inhalational induction with sevoflurane 8% is efficient for laryngeal mask airway placement in many cases, but it takes longer time than the propofol.[2]

- **Philip BK, Lambard L et.al (1999):** conducted a study titled "Comparison of vital capacity induction with sevoflurane to intravenous induction with propofol for adult ambulatory anaesthesia". In this study there were fifty six patients allocated randomly to receive either 8% sevoflurane in 75% Nitrous Oxide/Oxygen from already primed circuit (VC group n=32 patients) or propofol 2mg/kg bolus (IV group n=24) and time to induction, loss of consciousness and side effects are monitored. In the VC group patients, 59% have lost responsiveness in one breath taking 39±3s. All Vital capacity patients finished the induction and all measures, induction time are appreciably shorter time for Vital capacity group than intravenous group. They concluded that Vital capacity induction with sevoflurane is an satisfactory alternative to propofol intravenous induction of general anaesthesia for the adult ambulatory anaesthesia.[3]
- **V Priya, JVDivatia et.al (2002) :** conducted a study titled "A comparison of propofol vs sevoflurane for laryngeal mask airway insertion". Fifty female patients of American Society of Anesthesiologists grade I/II are randomly allocated into 2 groups (n=25

in every group)- Group S (inhalational sevoflurane) and group P (intravenous propofol). Group P received i.v propofol mean dosage 2.5mg/kg and group S 8% sevoflurane in 50% Nitrous Oxide and 50% Oxygen for 30s. After loss of eyelash reflex laryngeal mask airway insertion was excellent in group P (64%) than in group S(32%). 72% of patients in group P had complete jaw opening when compared to 44% of group S. Hence they concluded that, propofol is better than sevoflurane for laryngeal mask airway insertion.

- **Kati I, Demirel CB et.al (2003):** conducted a study titled "Comparison of propofol and sevoflurane for laryngeal mask airway insertion". In this study hundred patients aged between 20 to 40 years are randomly assigned into two groups. Group 1 received propofol (2.5mg.kg $^{-1}$ i.v) for induction and the group 2 received sevoflurane 6% (50% Nitrous Oxide+50% Oxygen) by the tidal volume technique of inhalational anesthesia. In both the groups, insertion of appropriate sized laryngeal mask airway was attempted. Laryngeal mask airway placement time is found to significantly lengthier in the sevoflurane group than in the propofol group.[5]
- **GuiQian S, GuoHua Z et.al (2007):** conducted a study titled "Comparison of propofol and sevoflurane for laryngeal mask airway

insertion in elderly patients". The study population consisted of ninety patients aged 60 years or more. They were induced either with i.v propofol or with sevoflurane 8% using the vital capacity breath or tidal volume breath technique. Laryngeal mask airway was inserted most rapidly with propofol (89 ±28s), less with sevoflurane 8% using the vital capacity breath (163±34s) and least with tidal volume breath (205±44s) techniques.[6]

- In the above studies, time taken for insertion of LMA was taken from loss of eye lash reflex. As a result of this, Sevoflurane induction using tidal volume technique would not produce sufficient systemic concentration in a short duration. So in this study, by adding 30 seconds to the loss of eyelash reflex, the ease of insertion of LMA is studied.

MATERIALS

AND

METHODOLOGY

METHODOLOGY

Study Design:

A prospective, randomized, controlled trial. This study conducted in Govt. Kilpauk Medical College and hospital Chennai.

Study setting population:

After getting approval from the Institutional Ethical Committee and informed written consent, eighty adult patients under American Society of Anesthesiologists physical status 1 and 2 of either sex undergoing elective minor surgical procedures were enrolled for this study.

Inclusion Criteria:

1. Elective - minor surgical procedures.
2. Males and females.
3. ASA physical status 1 – 2
4. Age above 18 years and below 50 years.
5. Patients with valid informed consent.

Exclusion Criteria:

1. Patients not satisfying inclusion criteria.
2. Patients with cardiac disease

3. Patients with allergic to inhaled anesthetics and propofol.
4. Known case of malignant hyperthermia or suspected genetic propensity.
5. Smokers (greater than or equal to twenty cigarettes per day).
6. Patients who are unconscious or severely ill.

MATERIALS:

1. Boyles machine with circle CO2 absorber circuit.
2. Volatile anaesthetic drug –Sevoflurane with vaporizer.
3. Propofol.
4. Classic Laryngeal Mask Airway size 3 and 4.

Resuscitation kit should be kept ready- Approximate size Endo tracheal tubes, Airways, Suction apparatus.

Patients in both the groups were IV cannulated with 18 G venflon. Monitors connected are NIBP, ECG, and Pulse Oxymetry. Premedicated with Inj. Glycopyrrolate 0.2mg i.v., Inj. Fentanyl 2microgram per Kilogram, Inj. Ranitidine 50 mg i.v., Inj. Ondensetron 0.1mg /kg. Then preoxygenated for 3 minutes with 100% O2.

Propofol group:

Patients in the propofol group were preoxygenated with 100 percent oxygen for three minutes and anesthetized using propofol 2 milligram per kilogram IV, given over a period of thirty seconds. 30seconds after the achievement of induction (i.e., sixty seconds after the start of propofol), jaw relaxation was assessed and, if achievable, Laryngeal Mask Airway placement was attempted. If not possible, attempts were repeated every thirty seconds upto a max. 4 attempts, every time preceded by intravenous boluses of propofol about 0.5 milligram per kilogram . Once the Laryngeal Mask Airway was inserted, all the patients were given sevoflurane four percent in 67% N2O in O2 at a rate of three litres/minute of fresh gas flow for three minutes. Then the sevoflurane concentration was reduced to two percent for volatile agent conservation. NIBP, ECG, SPO2 readings were recorded for five minutes in one minute interval. Any failure of placement, defined as failure to insert the LMA after 4 attempts, they were rescued with suxamethonium twenty five milligrams intravenously. Unless the patient suffered from O2 desaturation, controlled breaths were not given. The decision of not to ventilate the patients manually between Laryngeal Mask Airway insertions was proposed to avoid eliminating high PCO2 drive, as it could lengthen the time of apnoea. The existence of difficulties correlated to

induction and placement of the Laryngeal Mask Airway were noted, like excitatory movement or withdrawal from pain, gagging, coughing, apnoea and laryngospasm . At the end of the surgery, the existence of blood on the Laryngeal Mask Airway was noted.

Sevoflurane group

A closed circuit with circle absorber for CO2, with a 2-Litre breathing bag was used. The closed circuit was primed with eight percent sevoflurane in a 2:1 of N2O to O2 for one minute at a rate of six liters per minute of fresh gas flow. Then the patients asked to take a deep breath and then expire to residual volume. The face mask with primed closed circuit was positioned confidently over the face of the patient. The patients were taught to inspire a vital capacity breath and asked to hold it as long as possible. Loss of conscious ness was established by testing the eyelash reflex. Duration of vital capacity breath-hold was noted and 90s after the induction, the jaw relaxation was assessed. 90s was selected because it signifies the time at which all patients finished their Vital capacity breath. If jaw relaxation was not possible, attempts were repeated each thirty seconds upto a max. 4 attempts. An attempt of opening of mouth was considered as an attempt at placement of Laryngeal Mask airway. At this time, anesthesia sustained with sevoflurane 8% and N2O 67% in O2. Once jaw relaxation

was possible, Laryngeal Mask Airway insertion was tried, and the extent of diminution of reflexes of larynx were assessed.

If Laryngeal Mask Airway was inserted easily it classified as full; placement was associated with coughing, gagging, or patient movement, it classified as partial; when Laryngeal Mask Airway placement was not possible, it classified as poor. Three or four sizes of Laryngeal Mask Airway were used according to weight of the patient.

The following parameters were observed

During induction:

- Laryngospasm
- Cough
- Involuntary movement

During Laryngeal mask airway insertion

- Gagging
- Coughing
- Involuntary movement
- Apnea

Quality of Laryngeal Mask Airway Insertion

- Successful LMA insertion
- Time to LMA insertion (s)
- Number of Attempts (n)
- Successful initial mouth opening
- Presence of blood on LMA
- Hemodynamic parameters

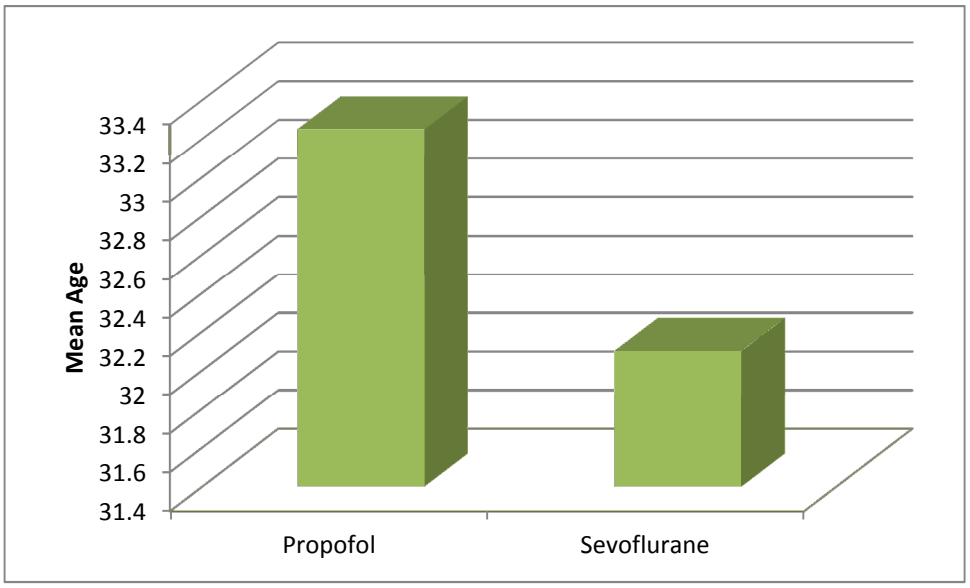
OBSERVATIONS AND RESULTS

OBSERVATION & RESULTS

Eighty patients of either sex in ASA 1 &2 status undergoing elective minor procedures were studied. The data was analysed with SPSS software version 19.1 P values less than 0.05 were considered statistically significant. Demographic data, the time taken for LMA insertion and hemodynamic variables among the groups were analysed with unpaired student t test . Chi-square analysis was used for comparing gender and number of attempts for insertion.

Comparison of age between Propofol and Sevoflurane group

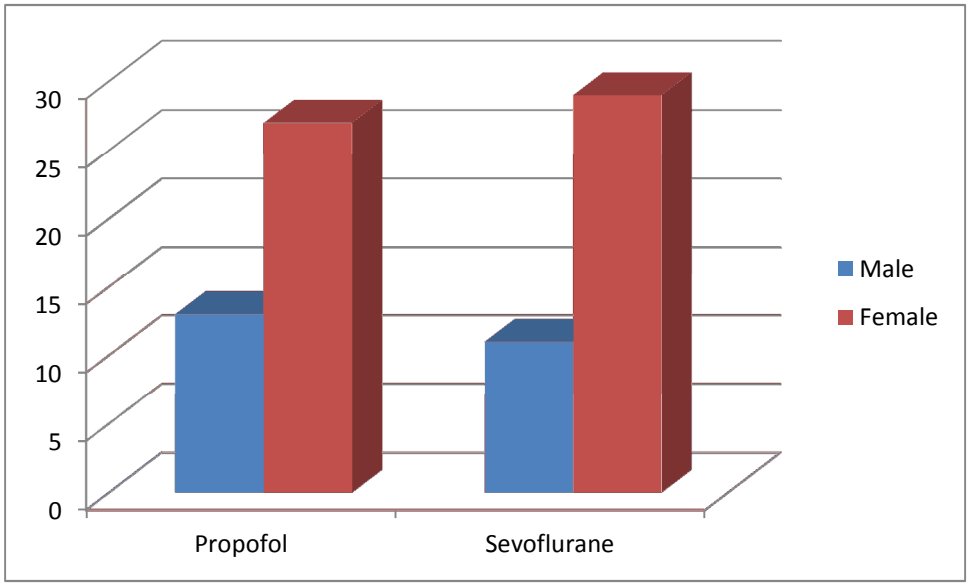
Group	Mean	SE	T stat	P value
Propofol	33.25	1.3	0.640	0.524
Sevoflurane	32.10	1.2		



The mean age in both groups was around 33 years. Both groups were equivalent with regard to age and there was no statistically signifigant difference between the two groups.

Comparison of Gender between Propofol and Sevoflurane group

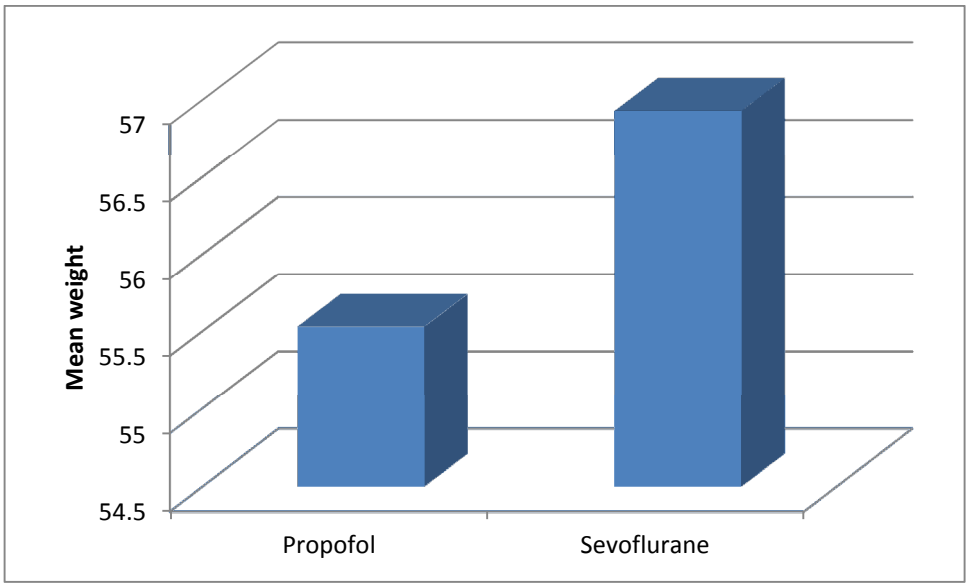
Group	Gender		Chisquare	P value
	Male	Female		
Propofol	13	27	0.24	0.626
Sevoflurane	11	29		



The gender distribution was comparable in both groups without any statistically significant variation in distribution.

Comparison of weight between Propofol and Sevoflurane group

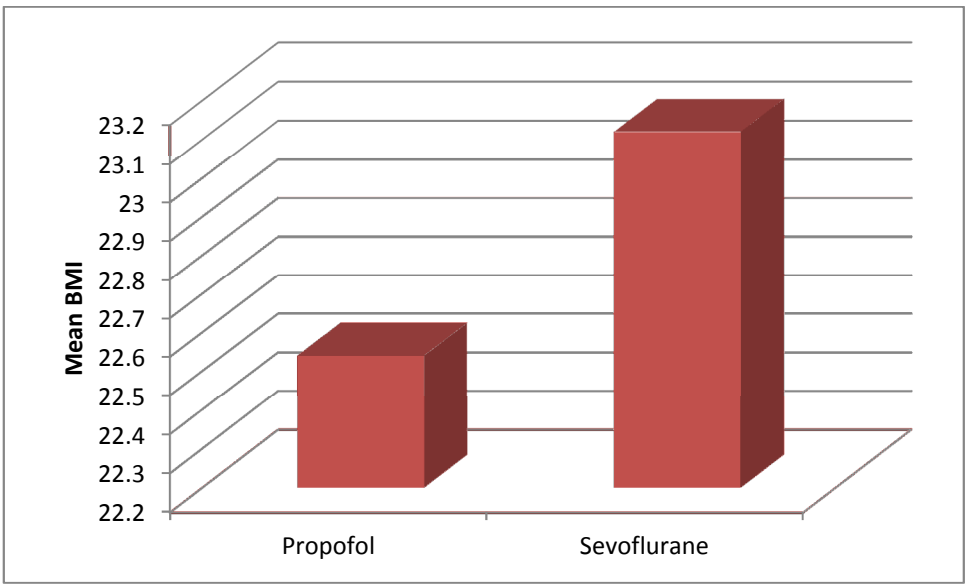
Group	Mean	SE	T stat	P value
Propofol	55.53	1.4	-0.786	0.434
Sevoflurane	56.93	1.1		



The mean weight in both the groups was around 56 kgs. Both the groups were comparable with regard to weight. There was no statistically significant difference between the two groups in terms of weight.

Comparison of BMI between Propofol and Sevoflurane group

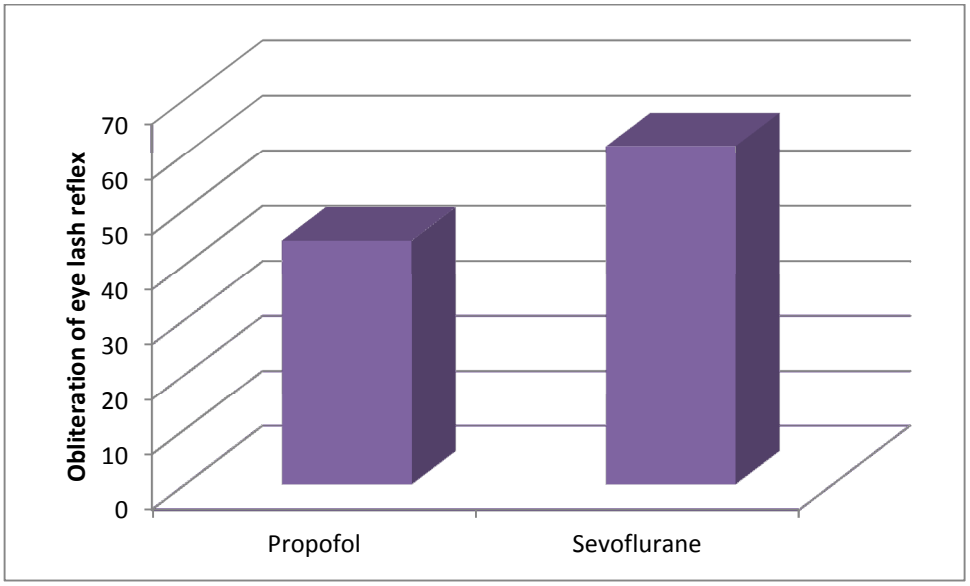
Group	Mean	SE	T stat	P value
Propofol	22.54	0.5	-0.982	0.329
Sevoflurane	23.12	0.4		



The mean Body Mass Index was around 22. There was no statistically significant difference between the two groups in terms of BMI and both the groups were comparable with regard to BMI.

Comparison of obliteration of eye lash reflex between Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	44.40	0.7	-13.977	0.0001
Sevoflurane	61.45	1.0		

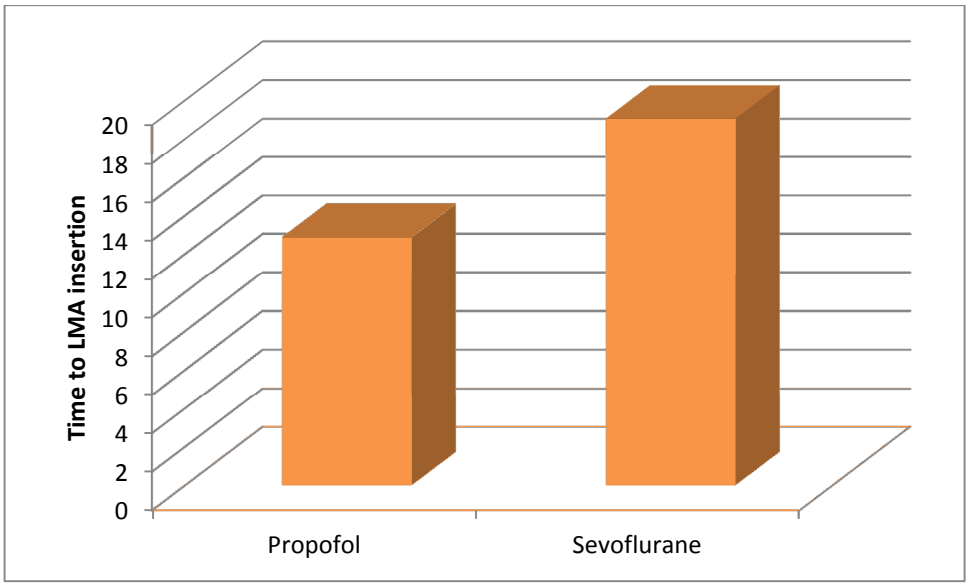


The maximum time for obliteration of eyelash reflex was 85secs in Sevoflurane group and 60secs in Propofol group. The minimum time for obliteration of eyelash reflex was 55secs in Sevoflurane group and 40secs in Propofol group.The mean time for obliteration of eyelash reflex was 44secs in Propofol group and 61secs in Sevoflurane group. The P value was statistically significant. i.e. 0.0001.

Comparison of Time to LMA insertion between

Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	12.88	0.6	-6.993	0.0001
Sevoflurane	19.05	0.6		

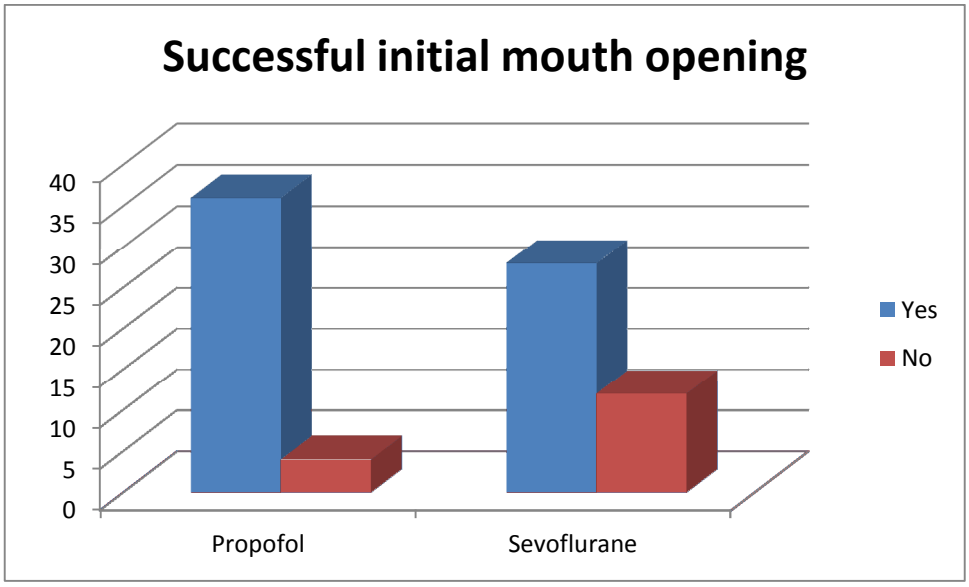


The maximum time for LMA insertion was 32secs in Sevoflurane group and 25secs in Propofol group. The minimum time for LMA insertion was 18secs in Sevoflurane group and 10secs in Propofol group.

The mean time for LMA insertion was 19.05secs in Sevoflurane group and 12.88secs in Propofol group. The P value was statistically significant i.e.0.0001.

Comparison of Successful initial mouth opening between Propofol and Sevoflurane group

Group	Successful initial mouth opening		Chisquare	P value
	Yes	NO		
Propofol	36	4	4.12	0.03
Sevoflurane	28	12		

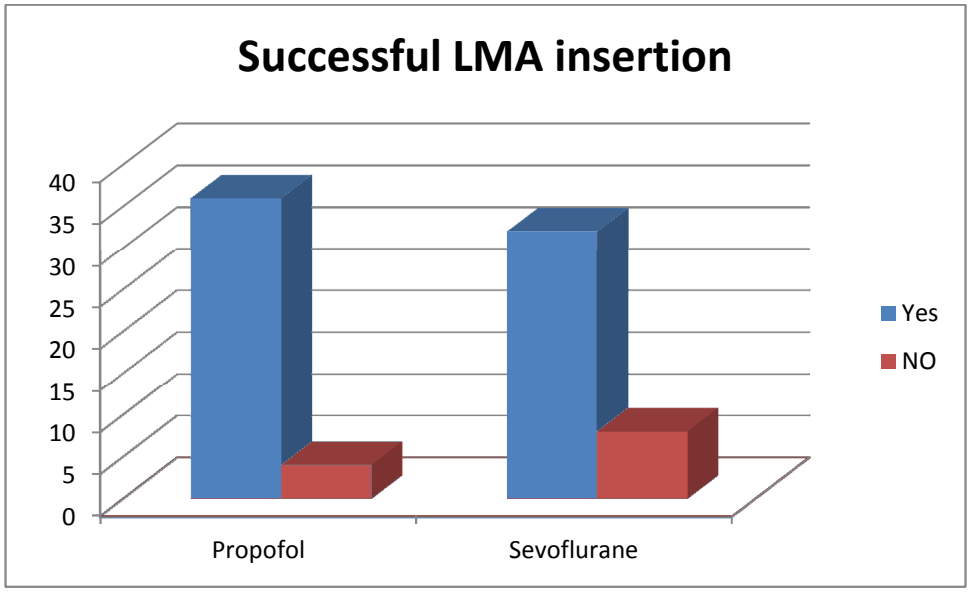


The successful initial mouth opening was more in the Propofol group compare to Sevoflurane group .The P value was statistically significant i.e. 0.03.

Comparison of Successful LMA Insertion between

Propofol and Sevoflurane group

Group	Successful LMA insertion		Chisquare	P value
	Yes	NO		
Propofol	36	4	1.57	0.210
Sevoflurane	32	8		

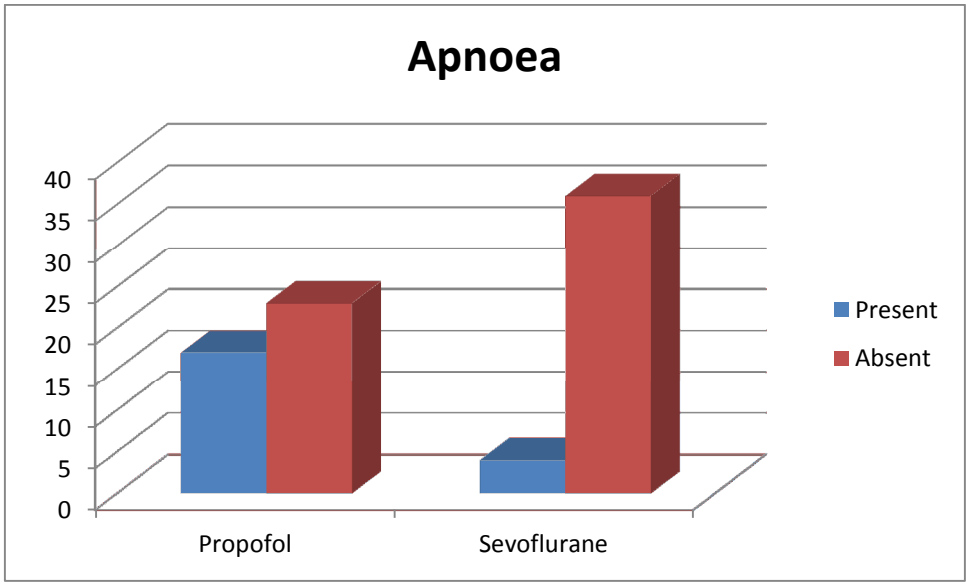


The successful LMA insertion was possible in both the groups without any statistically significant difference.

Comparison of occurrence of Apnoea between

Propofol and Sevoflurane group

Group	Apnoea		Chisquare	P value
	Present	Absent		
Propofol	17	23	10.91	0.0009
Sevoflurane	4	36		

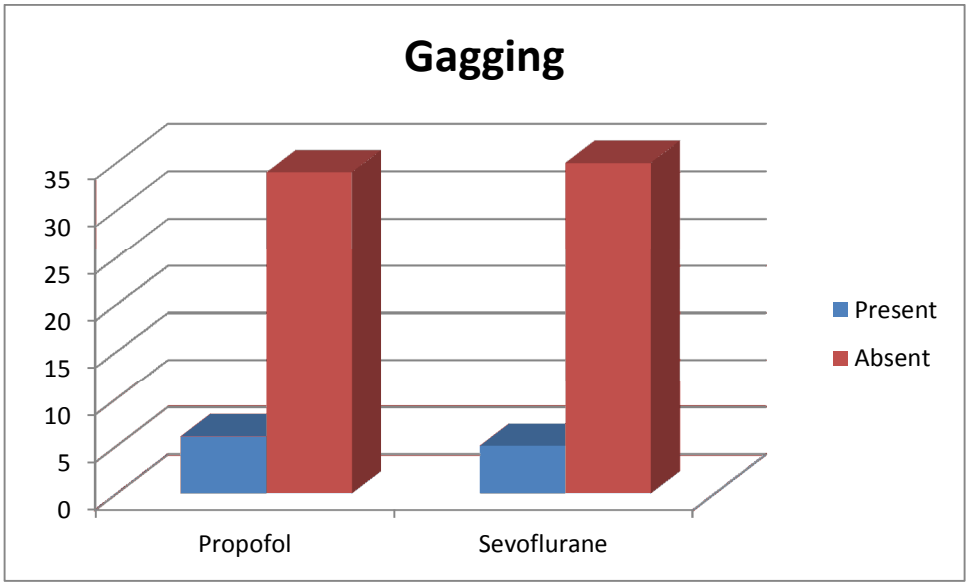


The occurance of Apnoea during induction were comparable in both groups with statistically important difference. The P value was 0.0009

Comparison of occurrence of Gagging between

Propofol and Sevoflurane group

Group	Gagging		Chisquare	P value
	Present	Absent		
Propofol	6	34	0.11	0.745
Sevoflurane	5	35		

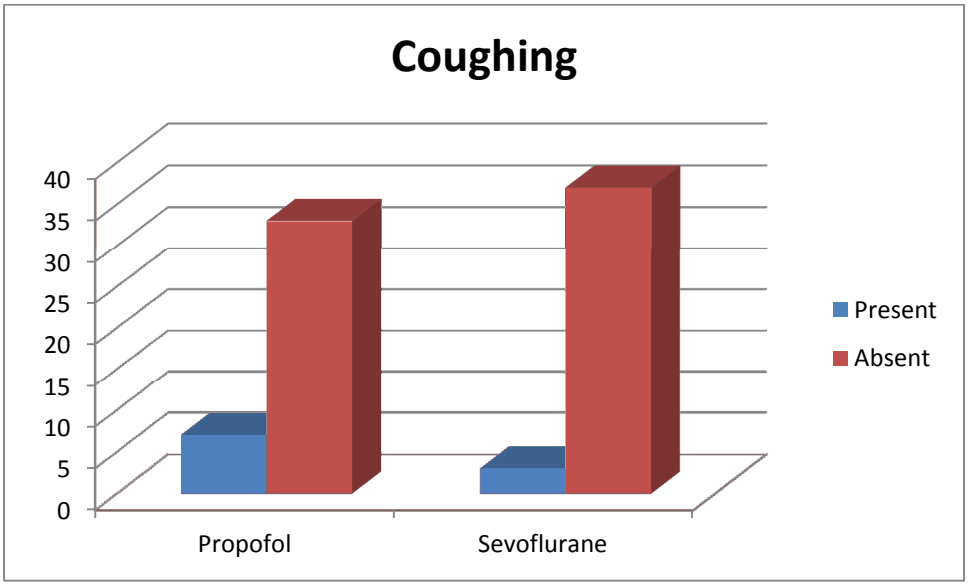


Occurrence of Gagging during LMA insertion was comparable in both the groups without any statistically significant difference.

Comparison of occurrence of Coughing between

Propofol and Sevoflurane group

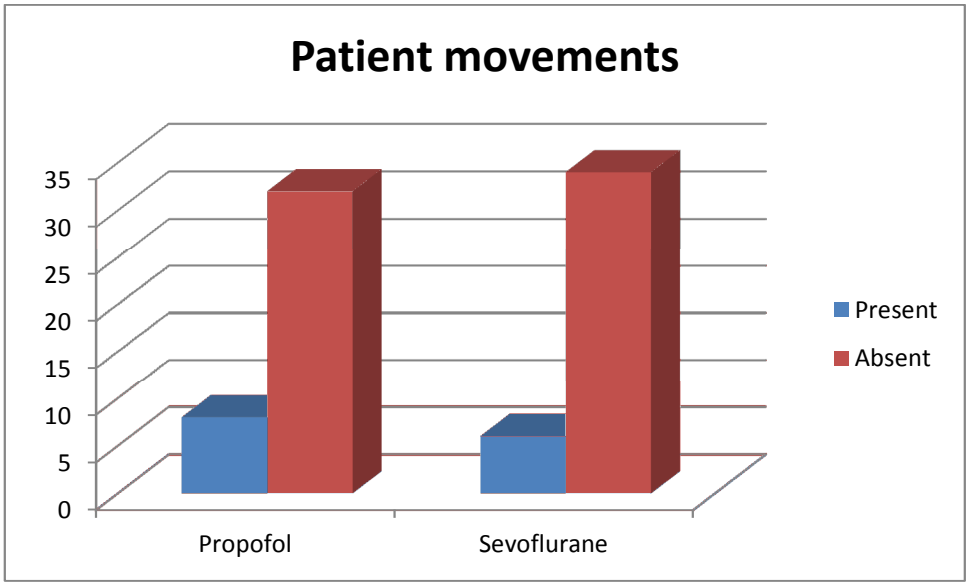
Group	Coughing		Chisquare	P value
	Present	Absent		
Propofol	7	33	1.83	0.176
Sevoflurane	3	37		



The occurrence of coughing during LMA insertion was comparable in both the groups without any statistically significant difference.

Comparison of occurrence of patient movements between Propofol and Sevoflurane group

Group	Patient movements		Chisquare	P value
	Present	Absent		
Propofol	8	32	0.35	0.556
Sevoflurane	6	34		

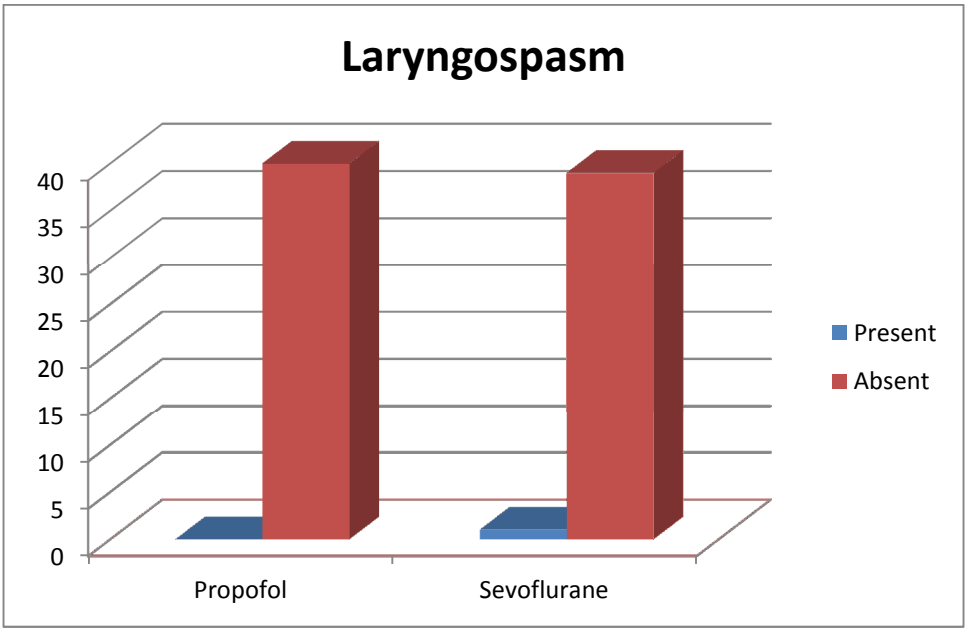


The occurrence of patient movements during LMA insertion was comparable in both the groups without any statistically significant difference.

Comparison of occurrence of Laryngospasm between

Propofol and Sevoflurane group

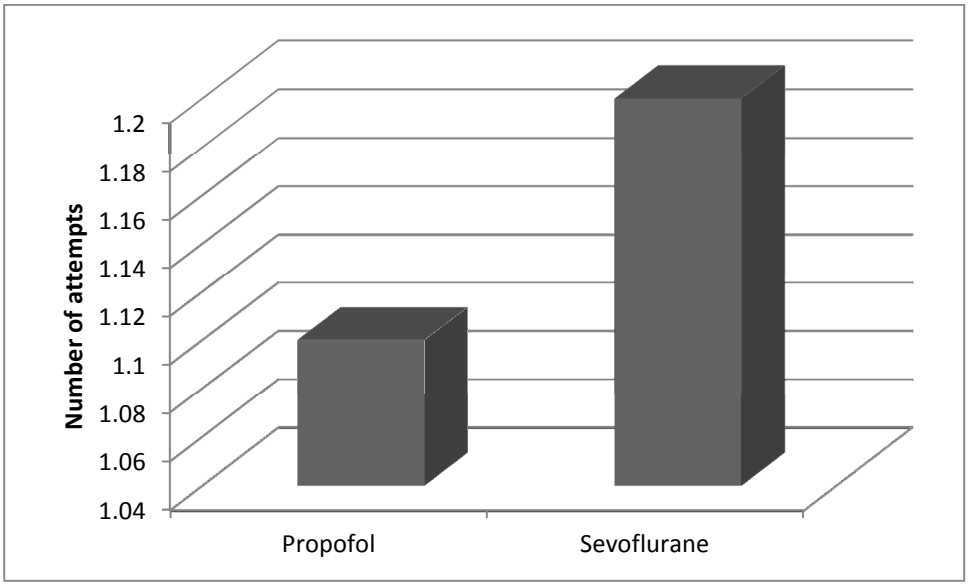
Group	Laryngospasm		Fisher test	P value
	Present	Absent		
Propofol	0	40	1.01	0.50
Sevoflurane	1	39		



The occurrence of Laryngospasm during induction was comparable in both the groups without any statistically significant difference.

Comparison of number of attempts between Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	1.1	0.04	-1.249	0.215
Sevoflurane	1.2	0.06		



The number of attempts for LMA insertion was comparable in both the groups without any statistically significant difference. The only thing was time to LMA insertion was prolonged in Sevoflurane group.

Comparison of pre induction PR between Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	82.40	0.8	-0.832	0.325
Sevoflurane	84.03	0.5		

Comparison of after induction PR between Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	78.15	0.8	-0.792	0.431
Sevoflurane	80.70	0.3		

Comparison of after LMA PR between Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	83.80	0.8	-1.103	0.119
Sevoflurane	85.75	0.3		

Comparison of one minute after LMA PR between

Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	86.48	0.9	-0.657	0.527
Sevoflurane	87.50	0.4		

Comparison of three minute after LMA PR between

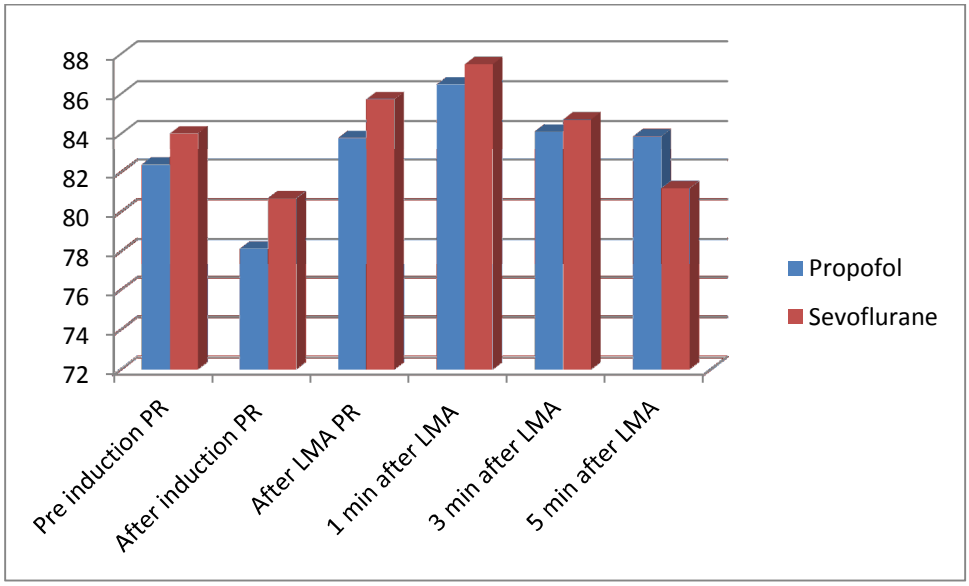
Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	84.13	0.8	-0.665	0.508
Sevoflurane	84.73	0.4		

Comparison of five minute after LMA PR between

Propofol and Sevoflurane group

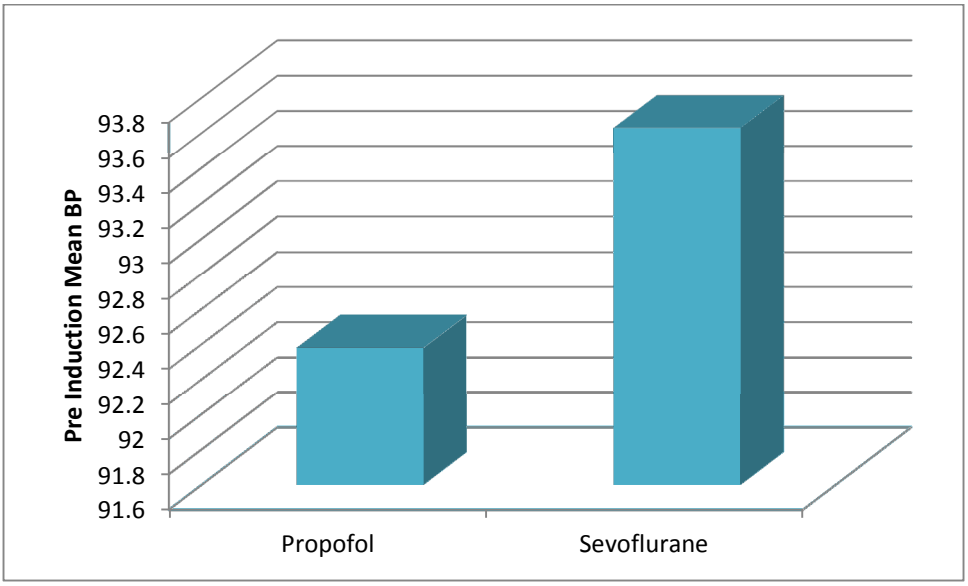
Group	Mean	SE	T stat	P value
Propofol	83.88	0.8	1.132	0.121
Sevoflurane	81.20	0.5		



Among the groups there were no significant disparity between the preinduction, after induction, immediately after insertion of LMA, one, three and five minutes post insertion heart rate.

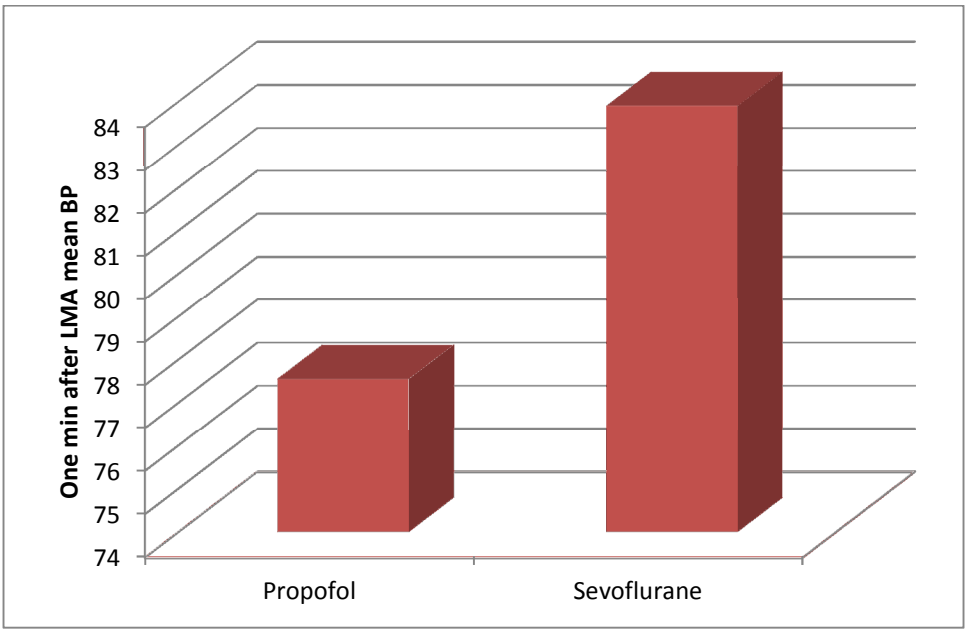
Comparison of pre induction mean BP between Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	92.38	0.8	-1.211	0.230
Sevoflurane	93.63	0.6		



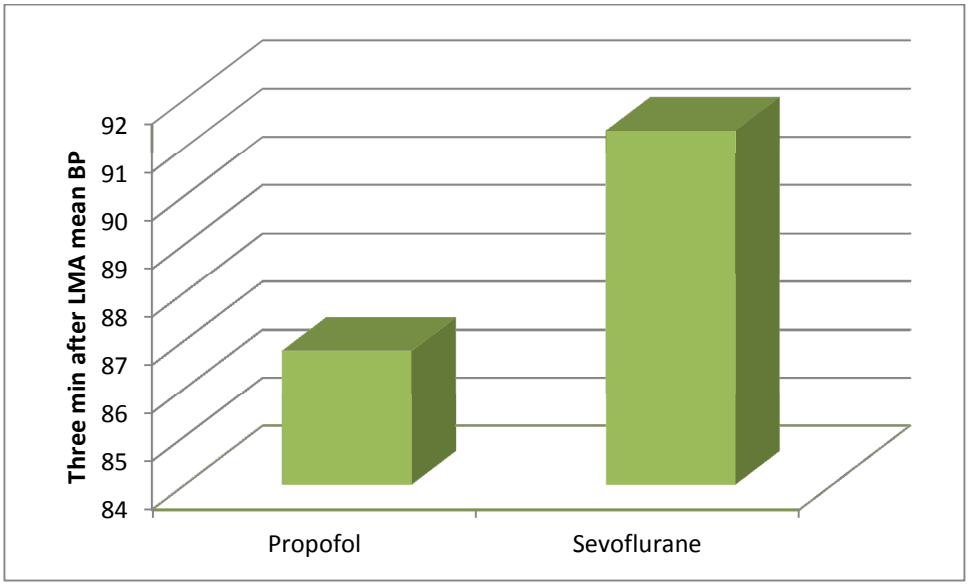
Comparison of one minute after LMA mean BP between Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	77.55	0.9	-6.430	0.0001
Sevoflurane	83.90	0.5		



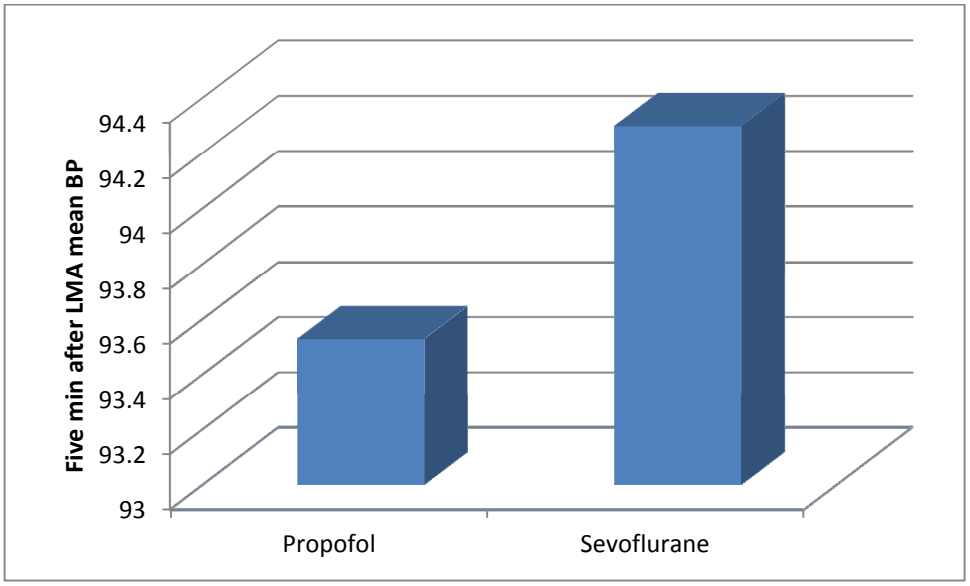
Comparison of three minute after LMA mean BP between Propofol and Sevoflurane group

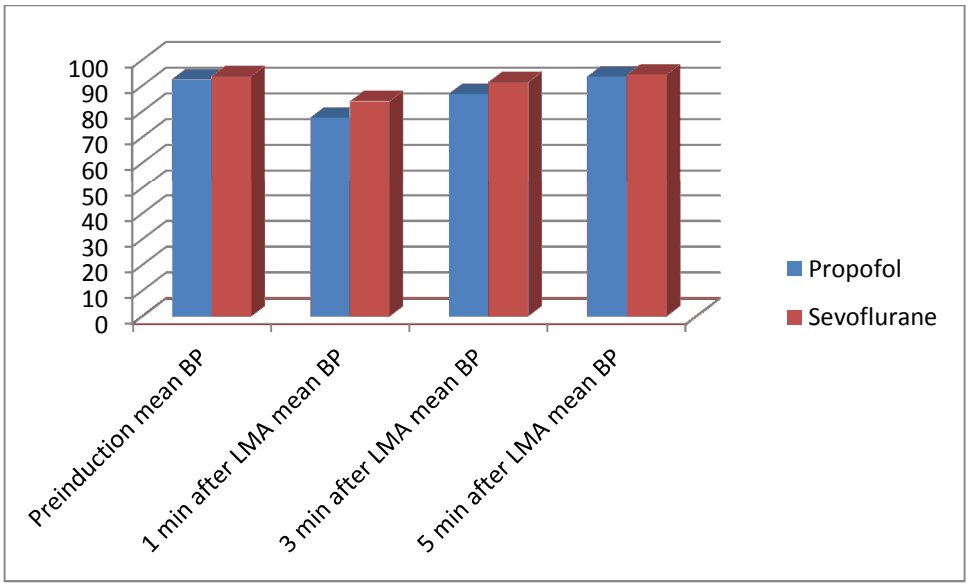
Group	Mean	SE	T stat	P value
Propofol	86.78	0.8	-4.653	0.0001
Sevoflurane	91.35	0.6		



Comparison of five minute after LMA mean BP between Propofol and Sevoflurane group

Group	Mean	SE	T stat	P value
Propofol	93.53	0.8	-0.806	0.423
Sevoflurane	94.30	0.6		





When compared between both groups , there was significant variation in the one, three minutes post insertion mean blood pressure. i.e. P value 0.0001.

DISCUSSION

DISCUSSION

In our study, we observed Sevoflurane single Vital capacity breath inhalational induction equates satisfactorily with propofol intravenous induction for placement of Laryngeal Mask Airway in adult patients[7,10]. Both sevoflurane and propofol fruitfully induced anesthesia in all patients in nearly forty seconds. The hemodynamic parameters were constant for both groups. Placement of Laryngeal Mask Airway after Propofol induction was achieved in all patients, paralleled with two failures in sevoflurane group. Both the induction methods were comparable and statistically insignificant. However, the time to successful Laryngeal Mask Airway insertion was prolonged in the Sevoflurane group.

Anesthetic induction and Laryngeal Mask Airway insertion by means of sevoflurane have so many benefits, like it allow a better conversion to maintenance phase without apnea[30,32]. The presence of apnoea needs the anesthesiologist to ventilate the patient manually. It nullified the benefit of anesthesiologist's free hands afforded by the Laryngeal Mask Airway. Sevoflurane prevents pain on injection that was present in propofol induction. There was also less hypotension with sevoflurane.

Hall et al[7]. Compared Laryngeal Mask Airway insertion by using 8% sevoflurane with single breath technique and intravenous propofol 3 milligram per kilogram. They showed that the adding of N2O in sevoflurane group increases speed of induction and safety, but they did not evaluate the easiness and excellence of Laryngeal Mask Airway placement at the most primitive opportunity. This resulted in slow times for Laryngeal Mask Airway insertion (109s and 146s for the propofol group and sevoflurane groups, respectively).

Our major problem about the excellence of Laryngeal Mask Airway insertion while using sevoflurane was inadequate mouth opening that resulted in two failures in our study. Interestingly, Muzi et al[5] reported inadequate jaw muscle relaxation after induction of sevoflurane, which resulted in failure to insert the Laryngeal Mask Airway in some patients.

Likewise, Hall et al[7] reported long time to jaw muscle relaxation with induction of sevoflurane compared with induction of propofol, even though they did not suggest any reasons for it. The possible explanation for the inadequate mouth opening in our patients was the lag time during which the concentration of sevoflurane in alveoli equilibrates with the concentration in brain, which leads to incomplete anesthesia during the primary attempt at insertion[21,27]. Furthermore, jaw muscle relaxation adequate for a jaw thrust

might be an indication of sufficient depth of anesthesia. However, Inomata and Nishikawa[9] dis agree the significance of this lag time. They argue that this was unlikely to be significant with sevoflurane induction because of its blood-gas partition coefficient is low.

Propofol is known to have a relaxant effect on jaw muscles, whereas inhaled anesthetics may cause accelerated muscle pitch and spasticity. Therefore, for a similar depth of anesthesia, there may be greater jaw muscle relaxation with propofol[16,17].

In contrast to the inadequate jaw muscle relaxation, there was superb lessening of laryngeal reflexes with sevoflurane[15.] This resulted in a lower incidence of distressing Laryngeal Mask Airway insertion in our patients. Although Laryngeal Mask Airway insertion is more intimately related with deglutition and only require repression of the less sensitive hypopharynx for successful insertion[13,14]. Stimulation of the anterior larynx can occur during placement. Therefore, reductions of the reflexes of larynx are necessary to lessen the occurrence of pulmonary complications during placement of Laryngeal Mask Airway. However, sevoflurane conserve reflexes of larynx at values up to 1.8 Minimum Alveolar Concentration. But in our study suggested that sevoflurane could lower reflexes of larynx at the higher Minimum Alveolar Concentration values achieved in our patients[18,20].

The disadvantage of our study was the anesthetic depth between two groups were not compared. However, it is not easy to evaluate the depth of anesthesia between volatile and intravenous anesthetics. Even though the plasma concentration of propofol correlated with depth of anesthesia, the correlation between Minimum Alveolar Concentration values and anesthetic depth of sevoflurane was not distinct obviously. This is because Minimum Alveolar Concentration refers to a state of balance, which was not achieved at some point in single Vital Capacity Breath technique induction. Moreover, the existence of a lag time between brain and alveolar concentrations can confound some attempted association.

We showed that the safety, excellence, and consistency of sevoflurane single Vital Capacity Breath induction makes it, an alternate method to intravenous induction of propofol for the placement of the Laryngeal Mask Airway in adult patients. Adjuvant drugs were not necessary in sevoflurane group. Sevoflurane Vital capacity breath technique results in equivalent complications and stable hemodynamic parameters during the induction and placement of Laryngeal Mask Airway. Sevoflurane produces a lesser frequency of apnoea and allows better conversion to the phase of maintenance[32]. On the other hand, it may results in a longer time to Laryngeal Mask Airway placement owing to lengthened jaw muscle tightness.

SUMMARY

SUMMARY

Placement of the LMA under inhalational anesthesia is not performed universally in adult patients. A famous method of anesthesia for Laryngeal Mask airway placement is with use of intravenous propofol, it has the benefits of inducing anesthesia quickly and depressing reflexes of upper airway. On the other hand, propofol is not ideal agent, it is associated with many side effects like apnea, pain on injection and hypotension. Recently, single VCB technique induction of inhalational sevoflurane is used as an alternate method to intravenous induction of propofol in adult patients. Sevoflurane induction method is quick, with greater acceptance, better hemodynamic profiles and slight excitatory phenomena. Sevoflurane can be used for both maintenance and induction of anesthesia. It made the conversion period easier. Hence, we compared sevoflurane inhaled induction and propofol IV induction.

After getting the Institutional Ethical Committee approval ,eighty adult patients of American society of Anesthesiologists Physical status 1 &2 of either sex undergoing minor surgical procedures are allocated randomly in to 2 groups, Group A (propofol induction) and Group B (sevoflurane induction).

The following parameters were observed:

- Jaw muscle relaxation
- Time to Laryngeal Mask Airway insertion.
- Number of attempts
- Patient Movement.
- Coughing, Gagging.
- Laryngospasm.
- Hemodynamic parameters.

This study shows no significant difference between the two groups based on the demographic variables. The time to LMA insertion in sevoflurane group was significantly different from propofol.(p value <0.05) Successful initial mouth opening in sevoflurane group was significantly different from Propofol group.(p value <0.05).The hemodynamic responses were significantly different from Sevoflurane (P value less than 0.05). There was no statistical difference between the two groups in number of attempts for Laryngeal Mask Airway insertion.

CONCLUSION

CONCLUSION

In our study we concluded that, inhalational induction by vital capacity breath technique using 8%Sevoflurane is an alternate to intravenous induction using Propofol for insertion of Laryngeal Mask Airway in adult patients. When compared to intravenous propofol induction, Sevoflurane Vital capacity breath technique had stable hemodynamic parameters and lesser complications. It allowed smooth conversion to maintenance phase and minimal occurrence of apnoea.

Even though extended jaw muscle tightness can delay LMA placement in patients with sevoflurane inhalational induction, it can be compared favorably with intravenous induction of propofol .

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ANNEXURE

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10

Protocol ID No. 15/02/15 Dt. 26/03/2015

CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "Comparison of Sevoflurane with propofol for Laryngeal Mask Airway Insertion in Adults"- For Project Work- submitted by Dr.B.S.Thamilselvi, MD (Anaesthesiology), Post Graduate Student, Govt Kilpauk Medical College, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

GOVT. KILPAUK MEDICAL COLLEGE * Kilpauk, Chennai-10. * SEP

25/03/2015.

INFORMATION TO PARTICIPANTS

Investigator:- Dr.B.S.Thamilselvi

Name of the Participant:-

Title: "Comparison of Sevoflurane with Propofol for Laryngeal Mask Airway Insertion in Adults".

You are invited to take part in this research study. We have got approval from the ICE. You are asked to participate because you satisfy the eligibility criteria.

Voluntary Participation:

Your participation in this research is entirely voluntarily. It's your choice whether to participate or not.

What is the purpose of this research?

Propofol is considered the drug of choice for the insertion of the laryngeal mask airway (LMA) during induction of anesthesia because of its depressant effect on airway reflexes. However, propofol has been associated with several adverse effects, including hypotension, apnea, pain on injection, and excitatory patient movement. Sevoflurane is a nonpungent inhaled anesthetic with a low blood gas solubility coefficient (0.69) and minimal respiratory irritant characteristics that make it suitable for inhaled induction of anesthesia and insertion of the LMA. Furthermore, sevoflurane, as compared with propofol, has the advantage of providing better hemodynamic stability and a smoother transition to the maintenance phase without a period of apnea.

Benefits:

Personally you won't be benefited in any way directly from the research. But by taking part in the research, you will be helping the scientific world to learn more about the drugs and parameters which are used in the study.

Possible Risks:

Adverse effects reported are

- During induction – apnea, laryngospasm,

- During LMA insertion – cough, gagging,

- Postoperatively – nausea, vomiting, sore throat

Right to Refuse or Withdraw:

You do not have to take part in the research if you do not wish to do so. You may also stop participating in the research at any time you choose. It is your choice and all of your rights will be respected.

You can ask me any more questions about any part of the research study, if you wish to. Do you have any questions?

1.

2.

3.

Date: Signature of the investigator.

Place:

Signature /Thumb impression
of the participant.

PATIENT CONSENT FORM

Study Detail: COMPARISON OF SEVOFLURANCE WITH PROPOFOL FOR LARYNGEAL MASK AIRWAY INSERTION IN ADULTS

Study center: GOVT. KILPAUK MEDICAL COLLEGE HOSPITAL, CHENNAI.

Patients Name :

Patients Age :

Identification Number :

Patient may check these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at anytime without giving reason, without my legal rights being affected. ☐

I Understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well – being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests. ☐

Signature/thumb Impression :

Patients Name and address:

Signature of investigator :

Study investigator's Name:

நோயாளி ஒப்புதல் படிவம்

ஆராய்ச்சியின் விவரம் :

ஆராய்ச்சி மையம் : அரசு கீழ்பாக்கம் மருத்துவக் கல்லூரி மருத்துவமனை

நோயாளியின் பெயர் : நோயாளியின் வயது:

பதிவு எண் :

நோயாளி கீழ்கண்டவற்றுள் கட்டங்களை (✓) செய்யவும்

1 மேற்குறிப்பிட்டுள்ள ஆராய்ச்சியின் நோக்கத்தையும் பயனையும் முழுவதுமாக புரிந்து கொண்டேன். மேலும் எனது அனைத்து சந்தேங்களையும் கேட்டு அதற்கான விளக்கங்களையும் தெளிவுபடுத்திக் கொண்டேன். ☐

2 மேலும் இந்த ஆராய்ச்சிக்கு எனது சொந்த விருப்பத்தின் பேரில் பங்கேற்கிநேன் என்றும், மேலும் எந்த நேரத்திலும் எவ்வித முன்றிவிப்பு மின்றி இந்த ஆராய்ச்சியிலிருந்து விலக முழுமையான உரிமை உள்ளதையும் இதற்கு எவ்வித சட்ட பிணைப்பும் இல்லை என்பதையும் அறிவேன். ☐

3 ஆராய்சியாளரோ, ஆராய்ச்சி உதவியாளரோ, ஆராய்ச்சி உபயத்தாரரோ, ஆராய்ச்சி பேராசிரியரோ, ஒழுங்குநெறி செயற்குழு உறுப்பினர்களோ எப்போது வேண்டுமானாலும் எனது அனுமதியின்றி எனது உள்நோயாளி மற்றும் புற நோயாளி பதிவுகளை இந்த ஆராய்ச்சிக்காகவோ அல்லது எதிர்கால பிறஆராய்ச்சிகளுக்காகவோ பயன்படுத்திக் கொள்ளலாம் என்றும் மேலும் இந்த நிபந்தனை நான் இவ்வராய்ச்சிலிருந்து தகும் என்றும் ஒப்புக்கொள்கிறேன். ஆயினும் எனது அடையாளம் சம்பந்தப்பட்ட எந்த பதிவகளும் (சட்டபூர்வமான தேவைகள் தவிர) வெளியிடப்படமாட்டது என்ற உறுதிமொழியின் பெயரில் இந்த ஆராய்ச்சிலிருந்து கிடைக்கப்பெறும் முடிவுகளை வெளியிட மறுப்பு தெறிவிக்கமாட்டேன் என்று உறுதியளிக்கிறேன். ☐

4. இந்த ஆராய்ச்சிஆச்ன வாயின் அருகில் வரும் சீழ் கட்டியை குறித்தது. அந்த நோயின் தன்மையையும், பின் விளைவுகளையும் பற்றியும், அறுவை சிகிச்சையின் போது கீறி எடுக்கப்படும் சீழை பரிசோதனைக்கு அனுப்பி கிருமியின் தன்மையையும் அதற்கு உகந்த மருந்தை பற்றியும் அறிய நடத்தும் ஆராய்ச்சி என்பதை மருத்துவர் மூலம் அறிந்து கொண்டேன். ☐
5. இந்த ஆராய்ச்சிக்கு நான் முழுமனதுடன் சம்மதிக்கின்றேன் என்றும் மேலும் ஆராய்ச்சி குழுவினர் எனக்கு அளிக்கும் அறிவுரைகளை தவறாது பின்பற்றுவேன் என்றும் உறுதியளிக்கிறேன். ☐
6. இந்த ஆராய்ச்சிக்குத் தேவைப்படும் அனைத்து மருத்துவப்பரிசோதனைகளுக்கும் ஒத்துழைப்பு தருவேன் என்று உறுதியளிக்கிறேன். ☐
7. இந்த ஆராய்ச்சிக்கு யாருடைய எற்புறுத்தலுமின்றி எனது சொந்த விருப்பத்தின் பேரிலும் சுயஅறிவுடனும் முழுமனதுடனும் சம்மதிக்கின்றேன் என்று இதன் மூலம் ஒப்புக்கொள்கிறேன். ☐

நோயாளியின் கையொப்பம் / பெருவிரல் கைரேகை
இடம்: தேதி:

ஆராய்ச்சியாளரின் கையொப்பம்:
இடம்: தேதி:

STUDY PROFORMA

GROUP:

S. NO. :

NAME: AGE& SEX : OP/IPNO:

WT: HT: BMI: ASA GRADE:

DIAGNOSIS:

SURGERY PLANNED:

PREMEDICATIONS: INJ.FENTANYL (2 microgram/Kg) - micrograms GIVEN AT
INJ.ONDANSETRON -4mg
INJ.RABEPRAZOLE-20mg

EVENT	TIME(am/pm)
START OF INDUCTION	
OBLITERATION OF EYELASH REFLEX	

TIME TAKEN FOR INSERTION OF LMA	Seconds
JAW RELAXATION	YES/ NO
LMA INSERTION	EASY/ DIFFICULT
NUMBER OF ATTEMPTS	

CONFIRMATION BY 1. CHEST EXPANSION 2. AUSCULTATION 3. CAPNOGRAPHY	
COMPLICATIONS	YES / NO
DETAILS OF COMPLICATIONS, IF PRESENT	

	Time	PR	SBP	DBP	MBP
Baseline value					
Pre induction					
After induction					
Immediately after insertion of LMA					
1 min after insertion					
3 min after insertion					
5 min after insertion					

MASTER CHART

PROPOFOL GROUP

SL.No.	Name	Age	IP No	Sex	Wt	Ht	BMI	ASA	Air Way	Procedure	Size
1	Parvathy	38	11627	Female	58	168	20.5	ASA-1	MPG-1	Right Fibroadenoma excision	Size-3
2	Amutha	40	10344	Female	52	153	22.5	ASA-1	MPG-2	Fractional curettage	Size-3
3	Sekar	42	13113	Male	64	162	24.4	ASA-1	MPG-1	Right URS	Size-4
4	Thangaraj	45	10242	Male	60	155	25	ASA-1	MPG-1	Right DJ stenting	Size-4
5	Anjali	24	10667	Female	32	140	16.4	ASA-1	MPG-1	DHL	Size-3
6	Kuppan	50	11718	Male	57	149	25.7	ASA-2	MPG-1	Left URS	Size-4
7	Yamuna	30	10034	Female	63	160	24.6	ASA-1	MPG-2	TAT	Size-3
8	Sumathy	28	10212	Female	48	151	21.1	ASA-1	MPG-1	DHL	Size-3
9	Senthil	34	11890	Male	61	164	22.7	ASA-2	MPG-1	Lipoma excision-R forearm	Size-4
10	Thilaga	25	12490	Female	52	163	19.6	ASA-1	MPG-2	DHL	Size-3
11	Meena	22	10067	Female	53	149	23.9	ASA-1	MPG-1	DHL	Size-3
12	Prema	31	10923	Female	37	150	16.4	ASA-1	MPG-2	TAT	Size-3
13	Govindhan	44	11159	Male	64	161	24.7	ASA-2	MPG-2	R URS	Size-4
14	Mary	42	10990	Female	35	152	16.2	ASA-2	MPG-1	Fractional curettage	Size-3
15	Kokila	23	12667	Female	60	155	25	ASA-1	MPG-1	DHL	Size-3
16	Muniraj	35	12645	Male	57	157	23.1	ASA-2	MPG-1	OIU	Size-4
17	Selvaraj	46	11342	Male	49	163	18.4	ASA-2	MPG-1	Left URS	Size-4
18	Gandhimathy	45	12118	Female	52	160	20.3	ASA-2	MPG-1	Fractional curettage	Size-3
19	Kalpana	26	11783	Female	70	164	26.8	ASA-1	MPG-1	DHL	Size-3
20	Perumal	32	11541	Male	65	153	24.5	ASA-1	MPG-1	L DJ stenting	Size-4

SL.No.	Name	Age	IP No	Sex	Wt	Ht	BMI	ASA	Air Way	Procedure	Size
21	Kavitha	26	11602	Female	59	168	20.6	ASA-1	MPG-1	DHL	Size-3
22	Vinayak	38	10982	Male	53	153	22.2	ASA-2	MPG-2	R URS	Size-4
23	Jeevitha	23	11267	Female	64	162	24.4	ASA-1	MPG-1	DHL	Size-3
24	Murugaraj	43	11875	Male	60	155	25	ASA-2	MPG-1	OIU	Size-4
25	Seetha	21	10983	Female	53	154	22.3	ASA-2	MPG-1	DHL	Size-3
26	Sumitha	25	10995	Female	57	149	25.7	ASA-1	MPG-2	DHL	Size-3
27	Isakkiyammal	27	11530	Female	63	160	24.2	ASA-1	MPG-1	TAT	Size-3
28	Selvam	40	12498	Male	60	155	25	ASA-2	MPG-2	Left DJ stenting	Size-4
29	Kadhiresan	37	11567	Male	61	164	23.8	ASA-1	MPG-1	OIU	Size-4
30	Meera	27	10359	Female	52	153	22.2	ASA-1	MPG-1	TAT	Size-3
31	Latha	24	11356	Female	54	150	23.8	ASA-1	MPG-1	DHL	Size-3
32	Lakshmi	46	11378	Female	53	154	23.9	ASA-2	MPG-1	Fractional curettage	Size-3
33	Panchali	43	10893	Female	53	152	23.2	ASA-2	MPG-2	Fractional curettage	Size-3
34	Usha	28	11209	Female	36	152	16.3	ASA-1	MPG-2	TAT	Size-3
35	Raja	33	11408	Male	60	164	24	ASA-1	MPG-1	Left URS	Size-4
36	Ramani	30	12305	Female	57	150	23.1	ASA-1	MPG-2	DHL	Size-3
37	Radhika	28	10897	Female	49	163	18.4	ASA-1	MPG-1	TAT	Size-3
38	Rajalakshmi	27	10972	Female	52	160	20.3	ASA-1	MPG-1	TAT	Size-3
39	Sundhari	32	11652	Female	71	163	26.8	ASA-2	MPG-2	DHL	Size-3
40	Sakundhala	30	11387	Female	65	154	24.5	ASA-1	MPG-1	TAT	Size-3

SL.No.	Name	Start of induction	Obliteration of Eyelash Reflex(from start of induction)	Time to LMA Insertion	Successful intial mouth opening	LMA Insertion	No of Attempts
1	Parvathy	9.30 am	40secs	12secs	Yes	Easy	1
2	Amutha	9.40 am	45secs	10secs	Yes	Easy	1
3	Sekar	9.35 am	42secs	10secs	Yes	Easy	1
4	Thangaraj	9.45 am	43secs	12secs	Yes	Easy	1
5	Anjali	10.00 am	43secs	11secs	Yes	Easy	1
6	Kuppan	9.30 am	40secs	13secs	Yes	Easy	1
7	Yamuna	9.35 am	45secs	12secs	Yes	Easy	1
8	Sumathy	9.45 am	50secs	25secs	No	Difficult	1
9	Senthil	9.25 am	42secs	13secs	Yes	Easy	1
10	Thilaga	9.35 am	45secs	12secs	Yes	Easy	1
11	Meena	9.20 am	42secs	12secs	Yes	Easy	1
12	Prema	9.40 am	40secs	10secs	Yes	Easy	1
13	Govindhan	9.25 am	42secs	11secs	Yes	Easy	1
14	Mary	9.30 am	40secs	13secs	Yes	Easy	1
15	Kokila	9.15 am	41secs	14secs	Yes	Easy	1
16	Muniraj	9.30 am	45secs	10secs	Yes	Easy	1
17	Selvaraj	9.45 am	42secs	14secs	Yes	Easy	1
18	Gandhimathy	9.35 am	42secs	11secs	Yes	Easy	1
19	Kalpana	9.40 am	48secs	12secs	Yes	Easy	1
20	Perumal	9.20 am	52secs	20secs	No	Difficult	2

SL.No.	Name	Start of induction	Obliteration of Eyelash Reflex(from start of induction)	Time to LMA Insertion	Successful intial mouth opening	LMA Insertion	No of Attempts
21	Kavitha	9. 30 am	45secs	12secs	Yes	Easy	1
22	Vinayak	9.20 am	40secs	10secs	Yes	Easy	1
23	Jeevitha	9.45 am	45secs	10secs	Yes	Easy	1
24	Murugaraj	9.30 am	42secs	12secs	Yes	Easy	1
25	Seetha	9.20 am	40secs	11secs	Yes	Easy	1
26	Sumitha	9.35 am	42secs	14secs	Yes	Easy	1
27	Isakkiyammal	9.50 am	48secs	12secs	Yes	Easy	1
28	Selvam	9.30 am	43secs	10secs	Yes	Easy	1
29	Kadhiresan	9.35 am	45secs	12secs	Yes	Easy	2
30	Meera	9.20 am	43secs	11secs	Yes	Easy	1
31	Latha	9.30 am	48secs	13secs	Yes	Easy	1
32	Lakshmi	9.35 am	43secs	14secs	Yes	Easy	1
33	Panchali	9.20 am	40secs	10secs	Yes	Easy	1
34	Usha	9.25 am	45secs	10secs	Yes	Easy	1
35	Raja	9.40 am	48secs	11secs	Yes	Easy	1
36	Ramani	9.25 am	42secs	12secs	Yes	Easy	1
37	Radhika	9.30 am	44secs	13secs	Yes	Easy	1
38	Rajalakshmi	9.45 am	60secs	24secs	No	Difficult	2
39	Sundhari	9.25 am	60secs	25secs	No	Difficult	2
40	Sakundhala	9.20 am	44secs	12secs	Yes	Easy	1

SL.No.	Name	Apnoea	Gagging	Coughing	Patient Movement	Laryngospasm	Presence of Blood on LMA
1	Parvathy	Yes	NO	NO	No	No	No
2	Amutha	Yes	NO	NO	NO	No	No
3	Sekar	NO	NO	NO	No	NO	No
4	Thangaraj	YES	NO	NO	NO	NO	No
5	Anjali	YES	NO	NO	NO	NO	NO
6	Kuppan	NO	YES	YES	YES	No	No
7	Yamuna	YES	NO	NO	YES	No	No
8	Sumathy	NO	NO	NO	NO	No	No
9	Senthil	NO	NO	NO	NO	No	No
10	Thilaga	NO	YES	YES	NO	No	No
11	Meena	NO	NO	NO	NO	No	No
12	Prema	YES	NO	NO	NO	No	NO
13	Govindhan	YES	NO	NO	NO	No	NO
14	Mary	NO	NO	NO	No	No	NO
15	Kokila	NO	NO	NO	NO	No	NO
16	Muniraj	NO	YES	YES	YES	No	NO
17	Selvaraj	YES	NO	NO	No	No	NO
18	Gandhimathy	NO	NO	NO	No	No	No
19	Kalpana	NO	NO	NO	Yes	No	No
20	Perumal	NO	NO	NO	Yes	No	Yes

SL.No.	Name	Apnoea	Gagging	Coughing	Patient Movement	Laryngospasm	Presence of Blood on LMA
21	Kavitha	NO	NO	No	No	No	No
22	Vinayak	YES	NO	YES	No	No	NO
23	Jeevitha	YES	NO	NO	NO	No	NO
24	Murugaraj	YES	NO	YES	YES	No	Yes
25	Seetha	YES	NO	NO	NO	No	No
26	Sumitha	YES	NO	NO	NO	NO	No
27	Isakkiyammal	YES	NO	NO	No	No	No
28	Selvam	NO	NO	NO	NO	NO	No
29	Kadhiresan	NO	NO	NO	NO	No	No
30	Meera	NO	NO	NO	NO	No	No
31	Latha	NO	NO	NO	NO	No	No
32	Lakshmi	YES	NO	YES	NO	NO	No
33	Panchali	NO	NO	NO	No	No	No
34	Usha	NO	NO	NO	NO	No	No
35	Raja	YES	NO	NO	No	No	No
36	Ramani	YES	YES	NO	NO	No	No
37	Radhika	NO	NO	NO	No	No	Yes
38	Rajalakshmi	NO	YES	YES	Yes	No	Yes
39	Sundhari	NO	YES	NO	Yes	No	Yes
40	Sakundhala	NO	NO	NO	No	No	NO

SL.No.	Name	Pre Induction PR	After Induction PR	Immediately after insertion of LMA	1 min after insertion	3 min after insertion	5 min after insertion	Pre induction SBP	Pre Induction DBP	Pre Induction MBP
1	Parvathy	82	72	78	82	87	84	120	80	93
2	Amutha	80	71	74	78	84	82	124	86	97
3	Sekar	86	78	80	85	88	84	130	86	101
4	Thangaraj	84	75	79	85	87	85	130	78	104
5	Anjali	78	68	72	76	81	80	120	80	93
6	Kuppan	80	69	72	75	78	76	124	70	101
7	Yamuna	86	70	74	78	82	84	110	70	87
8	Sumathy	84	76	80	85	90	88	110	78	84
9	Senthil	84	77	80	86	88	83	110	68	87
10	Thilaga	88	78	82	84	87	86	118	70	95
11	Meena	78	72	75	78	80	80	124	70	101
12	Prema	76	67	70	75	78	77	124	80	97
13	Govindhan	78	69	72	76	79	80	118	78	92
14	Mary	81	70	75	80	83	82	120	76	95
15	Kokila	82	68	73	76	79	77	114	68	91
16	Muniraj	85	74	78	84	88	89	120	86	91
17	Selvaraj	78	64	70	76	80	82	114	70	91
18	Gandhimathy	76	68	72	78	81	80	110	72	86
19	Kalpana	90	72	74	78	80	82	108	70	85
20	Perumal	78	70	74	81	84	82	110	64	89

SL.No.	Name	Pre Induction PR	After Induction PR	Immediately after insertion of LMA	1 min after insertion	3 min after insertion	5 min after insertion	Pre induction SBP	Pre Induction DBP	Pre Induction MBP
21	Kavitha	80	76	78	82	84	82	110	70	87
22	Vinayak	84	78	82	87	90	88	110	72	86
23	Jeevitha	88	80	84	90	92	91	120	70	94
24	Murugaraj	86	74	77	81	84	82	114	70	91
25	Seetha	78	70	73	78	80	84	110	68	87
26	Sumitha	92	84	86	90	91	94	124	74	92
27	Isakkiyammal	88	80	83	90	92	93	118	68	97
28	Selvam	86	80	84	90	90	88	120	74	95
29	Kadhiresan	90	78	81	87	88	90	110	68	87
30	Meera	80	72	76	80	84	81	120	72	97
31	Latha	84	76	78	73	75	78	114	68	91
32	Lakshmi	78	70	75	81	83	82	110	66	95
33	Panchali	76	68	73	80	82	80	118	78	92
34	Usha	92	84	88	95	96	97	120	78	95
35	Raja	88	80	83	87	89	91	110	70	87
36	Ramani	82	76	80	85	86	87	108	72	85
37	Radhika	84	72	76	80	83	85	110	64	89
38	Rajalakshmi	70	64	67	72	74	76	128	74	104
39	Sundhari	76	66	70	76	78	80	110	64	89
40	Sakundhala	80	70	74	79	80	83	120	74	95

SL.No.	Name	1 min SBP	1 min DBP	1 min MBP	3 min SBP	3 min DBP	3 min MBP	5 min SBP	5 min DBP	5 min MBP
1	Parvathy	100	68	77	110	72	86	114	76	88
2	Amutha	98	68	75	104	60	84	110	72	86
3	Sekar	104	60	84	110	76	85	118	80	91
4	Thangaraj	104	58	85	106	60	86	114	70	90
5	Anjali	100	62	79	114	72	90	120	80	93
6	Kuppan	102	58	83	118	68	95	124	76	98
7	Yamuna	90	56	71	100	56	81	108	72	84
8	Sumathy	90	62	69	100	62	79	114	70	90
9	Senthil	88	58	69	98	54	80	112	68	89
10	Thilaga	92	64	71	108	60	88	120	70	96
11	Meena	100	62	79	110	70	87	126	80	99
12	Prema	104	68	81	110	58	85	120	68	97
13	Govindhan	92	60	72	114	68	91	124	76	98
14	Mary	100	64	79	118	70	95	128	80	101
15	Kokila	90	58	71	100	58	80	112	68	89
16	Muniraj	102	60	82	114	70	90	120	76	94
17	Selvaraj	90	54	72	102	60	79	110	70	87
18	Gandhimathy	88	52	71	100	60	82	110	74	85
19	Kalpana	88	48	72	102	50	83	114	74	89
20	Perumal	92	62	71	104	60	84	116	70	92

SL.No.	Name	1 min SBP	1 min DBP	1 min MBP	3 min SBP	3 min DBP	3 min MBP	5 min SBP	5 min DBP	5 min MBP
21	Kavitha	98	58	79	110	60	90	120	78	94
22	Vinayak	98	68	75	110	70	87	124	82	97
23	Jeevitha	104	60	84	118	70	95	126	84	97
24	Murugaraj	100	62	79	114	60	94	120	76	95
25	Seetha	90	60	70	102	60	82	110	72	86
26	Sumitha	92	60	72	102	64	81	114	72	89
27	Isakkiyammal	96	56	77	110	56	91	122	70	98
28	Selvam	92	48	76	100	54	82	110	60	90
29	Kadhiresan	98	54	80	110	60	90	120	76	95
30	Meera	102	62	81	110	70	87	124	82	97
31	Latha	106	56	87	120	76	88	126	80	99
32	Lakshmi	100	52	83	114	70	97	120	74	95
33	Panchali	88	48	72	100	64	88	112	68	89
34	Usha	98	52	81	110	56	81	120	70	97
35	Raja	100	54	82	110	68	87	124	72	100
36	Ramani	96	54	78	110	70	87	126	80	99
37	Radhika	100	60	80	114	72	86	128	76	102
38	Rajalakshmi	102	58	83	110	64	93	122	70	98
39	Sundhari	104	52	87	116	70	87	120	80	93
40	Sakundhala	102	58	83	108	60	88	118	70	95

SEVOFLURANE

GROUP

SL.No.	Name	Age	IP No	Sex	Wt	Ht	BMI	ASA	Air Way	Procedure	Size
1	Ambika	28	11089	Female	58	168	20.5	ASA-1	MPG-1	TAT	Sze-3
2	Savitha	27	10456	Female	52	153	22.5	ASA-1	MPG-1	TAT	Size-3
3	Dharani	32	11076	Female	64	162	24.4	ASA-2	MPG1	DHL	Size-3
4	Muthu	43	11234	Male	60	155	25	ASA-1	MPG-2	Left URS	Size-4
5	Rani	44	11325	Female	32	140	16.4	ASA-1	MPG-2	Fractional curettage	Size-3
6	Kavitha	25	11347	Female	57	149	25.7	ASA-1	MPG-1	DHL	Size-3
7	Saminathan	46	11098	Male	63	160	24.6	ASA-2	MPG-1	URS right	Size-4
8	Sumitha	25	11078	Female	48	151	21.1	ASA-1	MPG-1	TAT	Size-3
9	Geetha	27	11267	Female	61	164	22.7	ASA-1	MPG-1	TAT	Size-3
10	Anjalai	29	11095	Female	52	163	19.6	ASA-1	MPG-1	DHL	Size-3
11	Rukmani	33	10987	Female	53	149	23.9	ASA-1	MPG-2	DHL	Size-3
12	Gangadevi	28	10867	Female	57	150	25.4	ASA-2	MPG-2	TAT	Size-3
13	Gowri	27	10367	Female	64	161	24.7	ASA-1	MPG-1	DHL	Size-3
14	Ranjitha	35	10874	Female	55	152	23.5	ASA-2	MPG-1	Fractional curettage	Size-3
15	Kadhirvel	48	11238	Male	60	155	25	ASA-2	MPG-1	OIU	Size-4
16	Seetha	26	10675	Female	57	157	23.1	ASA-1	MPG-1	DHL	Size-3
17	Ravi	27	11478	Male	49	163	18.4	ASA-1	MPG-1	Left URS	Size-4
18	Singam	22	11634	Male	52	160	20.3	ASA-1	MPG-1	OIU	Size-4
19	Ponmani	37	10583	Female	70	164	26.8	ASA-1	MPG-2	Fibroadenoma right	Size-3
20	Kaja	38	11487	Male	65	153	24.5	ASA-2	MPG-2	OIU	Size-4

SL.No.	Name	Age	IP No	Sex	Wt	Ht	BMI	ASA	Air Way	Procedure	Size
21	Kala	42	110753	Female	59	168	20.6	ASA-1	MPG-1	FIbroadenoma left	Size-3
22	Neela	25	10983	Female	53	153	22.2	ASA-2	MPG-2	DHL	Size-3
23	Savithiri	28	10872	Female	64	162	24.4	ASA-1	MPG-1	DHL	Size-3
24	Meera	27	11289	Female	60	155	25	ASA-1	MPG-2	TAT	Size-3
25	Sarala	27	11067	Female	53	154	22.3	ASA-2	MPG-2	TAT	Size-3
26	Janaki	29	11047	Female	57	149	25.7	ASA-2	MPG-1	TAT	Size-3
27	Thenmozhi	29	11285	Female	63	160	24.2	ASA-1	MPG-1	Right URS	Size-3
28	Gomathy	26	11679	Female	60	155	25	ASA-1	MPG-2	TAT	Size-3
29	Balraj	45	10876	Male	61	164	23.8	ASA-1	MPG-2	Left URS	Size-4
30	Samamoorthy	48	11213	Male	52	153	22.2	ASA-1	MPG-1	OIU	Size-4
31	Sivakami	26	10739	Female	54	150	23.8	ASA-2	MPG-1	Fibroadenoma Left	Size-3
32	Banumathy	28	11984	Female	53	154	23.9	ASA-1	MPG-2	TAT	Size-3
33	Pavithra	26	12345	Female	53	152	23.2	ASA-1	MPG-1	DHL	Size-3
34	Pitchai	43	12678	Male	52	153	23.3	ASA-2	MPG-2	Hydrocele right	Size-4
35	Kaliyan	47	12986	Male	60	164	24	ASA-2	MPG-2	Right URS	Size-4
36	Kannamal	28	13214	Female	57	150	23.1	ASA-1	MPG-1	TAT	Size-3
37	Meenatchi	28	12763	Female	49	163	18.4	ASA-1	MPG-1	TAT	Size-3
38	Prema	33	12786	Female	52	160	20.3	ASA-2	MPG-1	Fibroadenoma Right	Size-3
39	Kalaiselvi	24	10437	Female	71	163	26.8	ASA-1	MPG-2	DHL	Size-4
40	Sundaram	28	13467	Male	65	154	24.5	ASA-1	MPG-2	Left URS	Size-4

SL.No.	Name	Start of induction	Obliteration of Eyelash Reflex(From start of induction)	Time to LMA Insertion	Successful intial mouth opening	LMA Insertion	No of Attempts
1	Ambika	9.35 am	60secs	23secs	NO	Difficult	2
2	Savitha	9.45 am	55secs	18secs	YES	Easy	1
3	Dharani	9.20 am	62secs	16secs	YES	Easy	1
4	Muthu	9.35 am	55secs	17secs	YES	Easy	1
5	Rani	9.40 am	60secs	18secs	YES	Easy	1
6	Kavitha	9.25 am	62secs	17secs	YES	Easy	1
7	Saminathan	9.30 am	58secs	16secs	YES	Easy	1
8	Sumitha	9.20 am	62secs	18secs	YES	Easy	1
9	Geetha	9.25 am	58secs	18secs	YES	Easy	1
10	Anjalai	9.30 am	58secs	25secs	NO	Difficult	2
11	Rukmani	9.45 am	60secs	18secs	YES	Easy	1
12	Gangadevi	9.20 am	64secs	16secs	YES	Easy	1
13	Gowri	9.25 am	60secs	18secs	YES	Easy	1
14	Ranjitha	9.40 am	57secs	15secs	YES	Easy	1
15	Kadhirvel	9.45 am	58secs	18secs	YES	Easy	1
16	Seetha	9.20 am	60secs	16secs	YES	Easy	1
17	Ravi	9.25 am	60secs	18secs	YES	Easy	1
18	Singam	10.00 am	62secs	27Secs	NO	Difficult	2
19	Ponmani	10.15 am	75secs	22secs	NO	Difficult	2
20	Kaja	10.10 am	85secs	25secs	NO	Difficult	2

SL.No.	Name	Start of induction	Obliteration of Eyelash Reflex(Fr0m start of induction)	Time to LMA Insertion	Successful intial mouth opening	LMA Insertion	No of Attempts
21	Kala	9.30 am	58secs	18secs	YES	Easy	1
22	Neela	9.45 am	60secs	18secs	YES	Easy	1
23	Savithiri	9.30 am	62secs	15secs	YES	Easy	1
24	Meera	9.15 am	55secs	17secs	YES	Easy	1
25	Sarala	9.40 am	58secs	16secs	YES	Easy	1
26	Janaki	9.45 am	56secs	16secs	YES	Easy	1
27	Thenmozhi	10.00 am	60secs	19secs	YES	Easy	1
28	Gomathy	9.25 am	75secs	26secs	NO	Difficult	2
29	Balraj	9.30 am	60secs	19secs	YES	Easy	1
30	Samamoorthy	9.20 am	57secs	18secs	YES	Easy	1
31	Sivakami	9.30 am	62secs	18secs	YES	Easy	1
32	Banumathy	9.35 am	60secs	18secs	YES	Easy	1
33	Pavithra	9.40 am	58secs	17secs	YES	Easy	1
34	Pitchai	9.35 am	62secs	16secs	YES	Easy	1
35	Kaliyan	9.25 am	74secs	30secs	NO	Difficult	2
36	Kannamal	9.15 am	57secs	16secs	YES	Easy	1
37	Meenatchi	9.30 am	60secs	19secs	YES	Easy	1
38	Prema	9.40 am	61secs	18secs	YES	Easy	1
39	Kalaiselvi	9.45 am	62secs	17secs	YES	Easy	1
40	Sundaram	9.40am	70secs	32secs	NO	Difficult	2

SL.No.	Name	Apnoea	Gagging	Coughing	Patient Movement	Laryngospasm	Presence of Blood on LMA
1	Ambika	No	NO	NO	NO	NO	NO
2	Savitha	No	NO	NO	NO	NO	NO
3	Dharani	NO	NO	NO	NO	NO	NO
4	Muthu	NO	NO	NO	YES	NO	NO
5	Rani	NO	NO	NO	NO	NO	NO
6	Kavitha	NO	NO	NO	NO	NO	NO
7	Saminathan	NO	NO	NO	NO	NO	NO
8	Sumitha	NO	NO	NO	NO	NO	NO
9	Geetha	NO	NO	YES	NO	NO	NO
10	Anjalai	NO	NO	NO	NO	NO	NO
11	Rukmani	NO	NO	NO	NO	NO	NO
12	Gangadevi	NO	NO	NO	NO	NO	NO
13	Gowri	NO	NO	NO	YES	NO	NO
14	Ranjitha	YES	YES	NO	YES	NO	YES
15	Kadhirvel	YES	NO	NO	NO	NO	NO
16	Seetha	NO	NO	YES	NO	NO	NO
17	Ravi	NO	NO	NO	NO	NO	NO
18	Singam	NO	NO	NO	NO	NO	NO
19	Ponmani	NO	NO	NO	NO	NO	NO
20	Kaja	NO	YES	NO	NO	NO	NO

SL.No.	Name	Apnoea	Gagging	Coughing	Patient Movement	Laryngospasm	Presence of Blood on LMA
21	Kala	NO	NO	NO	NO	NO	NO
22	Neela	NO	NO	NO	NO	NO	NO
23	Savithri	NO	NO	YES	YES	NO	YES
24	Meera	NO	NO	NO	NO	NO	NO
25	Sarala	NO	NO	NO	NO	NO	NO
26	Janaki	NO	NO	NO	NO	NO	NO
27	Thenmozhi	NO	YES	NO	NO	NO	NO
28	Gomathy	NO	NO	NO	NO	NO	NO
29	Balraj	NO	NO	NO	NO	NO	NO
30	Samamoorthy	NO	NO	NO	YES	YES	YES
31	Sivakami	YES	YES	NO	NO	NO	NO
32	Banumathy	NO	YES	NO	NO	NO	NO
33	Pavithra	NO	NO	NO	YES	NO	NO
34	Pitchai	NO	NO	NO	NO	NO	NO
35	Kaliyan	NO	NO	NO	NO	NO	NO
36	Kannamal	NO	NO	NO	NO	NO	NO
37	Meenatchi	YES	NO	NO	NO	NO	NO
38	Prema	NO	NO	NO	NO	NO	NO
39	Kalaiselvi	NO	NO	NO	NO	NO	NO
40	Sundaram	NO	NO	NO	NO	NO	NO

SL.No.	Name	Pre Induction PR	After Induction PR	Immediately after insertion of LMA	1 min after insertion	3 min after insertion	5 min after insertion	Pre induction SBP	Pre Induction DBP	Pre Induction MBP
1	Ambika	88	90	91	86	88	84	124	80	97
2	Savitha	94	87	92	88	89	85	120	82	93
3	Dharani	94	90	91	90	87	83	120	86	91
4	Muthu	92	90	92	87	85	80	126	86	97
5	Rani	94	90	92	88	84	81	118	80	91
6	Kavitha	88	87	90	87	84	78	110	78	84
7	Saminathan	92	89	92	89	86	80	114	78	88
8	Sumitha	96	93	90	92	88	81	118	80	91
9	Geetha	92	90	91	88	87	80	116	76	90
10	Anjalai	92	93	94	87	86	78	122	80	95
11	Rukmani	84	93	95	90	85	80	120	80	93
12	Gangadevi	88	91	92	87	83	76	126	82	98
13	Gowri	90	93	90	88	84	78	124	84	96
14	Ranjitha	88	90	92	89	82	77	120	86	91
15	Kadhirvel	89	91	93	88	83	78	116	70	92
16	Seetha	87	92	90	86	82	75	118	80	91
17	Ravi	92	92	90	89	86	76	120	80	93
18	Singam	90	91	93	87	84	75	122	82	94
19	Ponmani	91	94	92	89	88	80	126	84	98
20	Kaja	96	92	93	94	90	84	128	84	100

SL.No.	Name	Pre Induction PR	After Induction PR	Immediately after insertion of LMA	1 min after insertion	3 min after insertion	5 min after insertion	Pre induction SBP	Pre Induction DBP	Pre Induction MBP
21	Kala	93	94	92	89	87	81	120	80	93
22	Neela	92	93	93	87	88	84	118	82	93
23	Savithri	90	91	94	86	85	82	116	74	91
24	Meera	88	89	92	85	84	80	124	80	97
25	Sarala	90	90	90	86	86	84	120	84	92
26	Janaki	88	92	93	85	79	80	130	84	102
27	Thenmozhi	87	92	95	86	80	80	128	78	102
28	Gomathy	89	91	90	88	82	76	118	70	94
29	Balraj	91	90	93	89	87	78	120	76	94
30	Samamoorthy	93	90	93	89	86	78	124	80	97
31	Sivakami	87	88	92	85	82	76	120	78	94
32	Banumathy	88	89	90	86	84	75	118	70	94
33	Pavithra	86	90	88	85	80	76	120	76	94
34	Pitchai	88	92	87	86	83	79	122	70	98
35	Kaliyan	94	91	91	88	87	80	120	80	93
36	Kannamal	90	90	92	87	88	82	124	84	96
37	Meenatchi	86	88	94	86	82	79	118	80	91
38	Prema	88	91	95	87	83	74	116	76	90
39	Kalaselvi	87	89	92	85	82	77	110	70	87
40	Sundaram	89	90	89	86	83	78	114	72	90

SL.No.	Name	1 min SBP	1 min DBP	1 min MBP	3 min SBP	3 min DBP	3 min MBP	5 min SBP	5 min DBP	5 min MBP
1	Ambika	110	70	87	108	60	88	114	70	90
2	Savitha	112	74	87	110	68	87	120	76	94
3	Dharani	110	80	83	110	72	86	120	72	96
4	Muthu	106	76	80	112	70	87	120	76	94
5	Rani	110	70	87	114	74	89	120	78	94
6	Kavitha	110	72	86	114	72	90	122	70	98
7	Saminathan	108	70	86	110	70	87	120	76	94
8	Sumitha	104	70	80	110	68	88	118	68	95
9	Geetha	106	72	82	114	64	92	120	72	96
10	Anjalai	104	70	80	110	68	88	122	70	96
11	Rukmani	110	70	87	108	64	86	120	74	95
12	Gangadevi	100	68	77	106	70	82	110	68	88
13	Gowri	106	72	82	112	70	86	118	64	96
14	Ranjitha	108	76	82	110	66	88	120	72	96
15	Kadhirvel	110	70	87	114	72	90	120	72	96
16	Seetha	106	70	83	114	70	91	122	74	97
17	Ravi	106	72	82	118	70	95	124	68	101
18	Singam	104	78	78	112	68	89	120	78	94
19	Ponmani	110	76	84	118	70	95	120	76	94
20	Kaja	106	76	80	120	72	96	114	70	90

SL.No.	Name	1 min SBP	1 min DBP	1 min MBP	3 min SBP	3 min DBP	3 min MBP	5 min SBP	5 min DBP	5 min MBP
21	Kala	110	74	85	116	64	94	120	72	96
22	Neela	110	76	84	118	68	95	120	74	95
23	Savithri	108	68	87	116	68	93	124	70	100
24	Meera	110	74	85	120	72	96	124	74	99
25	Sarala	110	74	85	118	74	93	126	70	102
26	Janaki	106	72	82	120	76	94	124	78	98
27	Thenmozhi	110	72	86	122	80	95	120	76	94
28	Gomathy	108	64	86	120	78	94	118	74	93
29	Balraj	110	72	86	118	78	92	122	70	98
30	Samamoorthy	110	68	88	120	76	94	124	76	98
31	Sivakami	108	68	85	110	72	86	118	80	91
32	Banumathy	110	60	90	118	72	94	114	76	88
33	Pavithra	110	64	88	116	70	92	110	68	87
34	Pitchai	108	64	86	120	78	94	116	68	93
35	Kaliyan	104	68	81	122	70	98	114	70	90
36	Kannamal	110	68	87	124	76	98	118	76	92
37	Meenatchi	108	70	84	124	74	98	120	74	95
38	Prema	104	60	84	120	76	94	116	72	92
39	Kalaiselvi	100	62	79	110	60	90	114	70	90
40	Sundaram	100	64	78	112	64	90	110	68	87