A RANDOMIZED CLINICAL STUDY TO COMPARE THE EMERGENCE PHENOMENA AFTER GENERAL ANAESTHESIA WITH 2% LIGNOCAINE JELLY AND 4% LIGNOCAINE INTRACUFF

Submitted by:

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DISSERTATION

Submitted to the

KLE University, Belgaum, Karnataka.

In partial fulfillment of the requirements for the award of the degree of

M. D.

IN

ANAESTHESIOLOGY

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MAY 2010

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DECLARATION BY THE CANDIDATE

I hereby declare that this thesis entitled **"A RANDOMIZED CLINICAL STUDY TO COMPARE THE EMERGENCE PHENOMENA AFTER GENERAL ANAESTHESIA WITH 2% LIGNOCAINE JELLY AND 4% LIGNOCAINE INTRACUFF"** *is a bonafide and genuine research work carried out by me under the guidance of* **Dr. C. S. Sanikop**$_{M.D,D.A}$ Professor and Head of Department, Department of Anaesthesiology, J. N. Medical College, Belgaum.

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Dr. Nitish Desai

ABSTRACT

Background: Anaesthesiologists do experience varying degree of problems with extubation. Coughing during emergence from general anesthesia is a common problem. We sought to determine whether inflating endotracheal tube cuff with lignocaine would create a reservoir of local anaesthetic, which might diffuse across the cuff membrane to anaesthetize the mucosa, thus attenuating stimulation during extubation of the trachea.

Objective: To assess incidence of coughing and hemodynamic changes post extubation with 2% lignocaine jelly and 4% lignocaine intracuff.

Study design: 1 year randomized clinical study.

Methods: After obtaining approval from institutional ethical committee, 140 patients of ASA I and II, age of 18 to 65 years were studied. After intubation with an endotracheal tube, the cuff inflated with 4% lignocaine in one group and air in another group, 2% lignocaine jelly applied over the cuff which was to be inflated with air and cuff pressure set at 20 cm H_2O with Rusch Endotest cuff inflator. Cuff pressure recorded just before extubation. After extubation, a blinded observer recorded incidence of coughing, heart rate and blood pressure. Data was analyzed statistically.

Results: The groups were demographically comparable. Significant changes were observed in cuff pressures. We observed that 4% lignocaine intracuff reduces the incidence of coughing for the time period 0-5 minutes post extubation ($P<0.001$). There was significant decrease in hemodynamic response ($P<0.001$) in intracuff lignocaine group.

Conclusion: 4% lignocaine intracuff significantly reduces post extubation coughing and decreases hemodynamic response to extubation in addition maintains constant cuff pressure.

Key words: Intracuff lignocaine, lignocaine jelly, coughing.

LIST OF ABBREVATIONS

µgm	–	Microgram
ASA	–	American Society of Anaesthesiologists
ECG	–	Electrocardiograph
EtCO2	–	End tidal carbon dioxide
ETT	–	Endotracheal tube
Group I	–	Intracuff lignocaine group
Group J	–	Lignocaine jelly group
HR	–	Heart rate
Hr	–	Hour
i.e.	–	That is
I.V	–	Intra venous
Inj	–	Injection
kg	–	Kilogram
max	–	Maximum
mg	–	Milligram
min	–	Minutes
NIBP	–	Non invasive Blood Pressure
NS	–	Non significant
S	–	Significant
S.D	–	Standard Deviation
SpO2	–	Oxygen saturation
viz..	–	namely, that is

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INTRODUCTION

Anaesthesiologists do experience varying degree of problems with extubation. The frequency of problems encountered during endotracheal extubation probably exceeds the problems encountered during intubation. Endotracheal extubation is an art that one develops over a period of time with experience.

Cuffed endotracheal intubation offers additional safety to the patient by preventing aspiration syndromes. However, amongst the sequelae inherent to the usage of cuffed endotracheal tube, the ischemic injuries to the tracheal mucosa caused by prolonged inflation of cuff and the resulting increase in cuff pressure are the prominent ones which result in post intubation morbidities like coughing, sore throat, difficulty in swallowing and hoarseness.

Tracheal cuff pressure should always be maintained below the mean capillary mucosal perfusion pressure of 30 cmH_2O to prevent ischemic damage. Obstruction to the tracheal mucosal blood flow begins at 30 cmH_2O and becomes absolute at 45-50 cmH_2O. [1]

Endotracheal extubation occurs at the end of general anaesthesia and patients are usually in the lighter planes of anaesthesia. Hence a lot of problems are encountered during endotracheal extubation. These problems could range from mild coughing to severe hemodynamic changes that can even result in a catastrophe.

Laryngoscopy and tracheal intubation are accompanied by increase in the heart rate and blood pressure. Maneuvers in attenuating these cardiovascular responses are well established and practiced. Similar changes do occur during tracheal extubation. This is

attributed to release of catecholamines. Other factors are due to the irritation of tracheal mucosa caused by endotracheal tube cuff and pain from the surgical site.[2]

Coughing during tracheal extubation is also a common clinical problem during general anaesthesia. The presumed mechanism for this is the irritant or stretch stimuli caused by the tube and its cuff. Rapidly acting receptors are found throughout the trachea and are primarily superficial.[2] Coughing and cardiovascular response can lead to variety of complications like Bronchospasm, Surgical site bleeding, Myocardial Ischemia, arrhythmias, Increased intraoccular and Intracranial pressure.[3,4,5]

Many studies have been performed and published exploring the measures to minimize/eliminate post-intubation morbidities, viz, Use of high volume –low pressure cuffed Endotracheal tubes,[6] Use of smaller sized endotracheal tubes,[7] Use of lidocaine by various routes,[8] Topical application of lubricant jelly.[9]

In some of the recent studies done they have shown that lignocaine can be used intracuff [10] and this serves as a reservoir and also allows diffusion of lignocaine molecules through the cuff to the tracheal mucosa at a constant rate.[11]

In this study we have evaluated the efficacy of 4 % lignocaine intracuff in comparison with application of lignocaine jelly 2% which is routinely practiced, on the incidence of coughing and hemodynamic parameters post extubation.

OBJECTIVES

1. To asses incidence of coughing post extubation with 2%lignocaine jelly and 4%lignocaine intracuff.

2. To asses hemodynamic changes post extubation with 2% lignocaine jelly and 4% lignocaine intracuff.

REVIEW OF LITERATURE

Control of the airway was to be one of the defining moments in the field of anaesthesiology. Anaesthesiologists do experience varying degree of problems with extubation. The frequency of problems during tracheal extubation probably exceeds the problems encountered during endotracheal intubation. The appropriate time to remove an endotracheal tube is an art that one develops with experience. Hence the common problems during tracheal extubation can range from mild coughing to severe hemodynamic changes that can even result in a catastrophe.

Intubation from the neck through a tracheostomy wound was performed in 1858 by John Snow in anaesthetized animals. Friederich Trendelenberg in 1871 intubated in human beings by occluding the trachea by an inflatable cuff. Addition of inflatable cuff to the rubber tube by Ralph Waters and Arthur Guedal in 1928 paved way for closed circuit anaesthesia and true isolation of the airway.[12] A pilot balloon was introduced in 1893 by Victor Eisenminger and reintroduced in 1939 by Langton Hewer.[13]

Nitrous oxide has been a cornerstone of anaesthetic practice since its first use in 1840`s, but, it has got its own disadvantages. The important one among them being nitrous oxide induced tracheal injury due to increased intracuff pressure.

J.C.Raeder and others studied the changes in cuff pressure of tracheal tubes in 60 patients undergoing lower abdominal surgery. The cuffs were inflated either with air or anaesthetic gas mixture. The pressure in air filled cuffs increased steadily throughout the procedure. They concluded that filling the cuff with anaesthetic gas mixture is a simple and reliable way to achieve the stable cuff pressure.[1]

M.Cobley and others showed that the endotracheal cuff pressure when inflated with routine syringe was high compared to the pressure produced in the same cuff which was inflated just to prevent air leak using Cardiff cuff controller. They suggested that the method of inflating the cuff by air filled syringe may lead to over inflation and subsequent excessive pressure on the endotracheal wall.[14]

A study using air, saline and a mixture of nitrous oxide and oxygen to inflate the cuffs of tracheal tubes in three groups of patients by Mitchell V showed increase in the intracuff pressure in the air group, decrease in the gas mix group with gas leak in some patients and the cuff pressure remained stable in the saline group although there was some difficulty in initial adjustment of cuff pressure.[15]

With the patient in lighter planes of anaesthesia, extubation can produce a significant increase in heart rate and arterial blood pressure, which can persist in to the recovery period. In a patient who is awake and has fully recovered from neuromuscular blockade, tolerating the endotracheal tube is quite uncomfortable. This along with the added effects of extubation can cause lot of hemodynamic changes. The hemodynamic response to tracheal extubation is well tolerated by majority of patients. However, patients with co-existing cardiovascular disease may be unable to tolerate this response. While others may demonstrate an exaggerated response that might be poorly tolerated.[3]

The exact mechanism of these cardiovascular changes in unknown but is believed to be due to release of catecholamine's causing increase in heart rate, myocardial contractility and systemic vascular resistance. In patients with coronary artery disease the hemodynamic responses to tracheal extubation may affect the myocardial oxygen supply demand ratio leading to myocardial ischemia.

The hemodynamic response to endotracheal extubation was studied in patients after CABG surgery and mechanical ventilation. Patients showed significant increase in heart rate, arterial blood pressure, right atrial pressure and cardiac index within 1 minute of tracheal extubation, returning to pre-extubation levels by 5 minutes.[16] These hemodynamic responses mimic the responses seen during laryngoscopy and tracheal intubation in the same group of patients in most studies. In majority of patients these hemodynamic responses will not affect the outcome or contribute to morbidity of the patient, but there remains a small group of patients in whom a single hyper dynamic episode can result in a catastrophe.

In a study of patients undergoing CABG surgery by Slogoff and Keats, perioperative myocardial ischemia was related to the number of episodes of tachycardia rather than hypertension.[17] In most cases a single episode of hypertension and tachycardia may be well tolerated, but there may be some who are very sensitive to the changes. Hence it seems reasonable to minimize the hemodynamic responses to extubation in all patients with coronary artery disease.

In hypertensive patients, emergence from general anaesthesia and tracheal extubation is commonly associated with increase of heart rate and blood pressure. These changes are similar to those with intubation. Fuji and colleagues studied the cardiovascular response to tracheal extubation or laryngeal mask airway removal in normotensive and hypertensive patients. They concluded that the hemodynamic changes are greater in hypertensive than in normotensive patients. Hypertensive patients thus exhibit an exaggerated response to tracheal extubation.[18]

In patients with severe preeclampsia the cardiovascular response to tracheal extubation is exaggerated.[19] The maternal mortality and morbidity is closely related to the degree of hypertension. In most of the cases studied, the immediate cause of death was either pulmonary or cerebral hemorrhage. As these complications are likely to be related to the severity of hypertension, it is important to be aware of the sequelae. The exaggerated response in these patients should be kept in mind during the event of anaesthesia.

There are many maneuvers that are being done to attenuate the response to extubation. Extubation of the trachea with the patients in a deep plane of anaesthesia achieved by inhalational or intravenous anaesthetic opioids can attenuate all the responses. Lignocaine has been used to suppress the cardiovascular responses of tracheal extubation. Different routes of administration of lignocaine such as intravenous and tracheal have been attempted in this regard with variable responses.

Tsutsai T used a mixture of Nicardipine and Dilitiazem with excellent control of blood pressure in patients undergoing surgery during the time of extubation. Hypertensive responses to emergence and extubation were successfully attenuated compared to the control group with no significant change in the heart rate.[20]

Kovac A.L and colleagues in their study showed that Nicardipine in doses of both 0.015mgkg $^{-1}$ and 0.03mgkg $^{-1}$ attenuated the hypertensive response to extubation with no significant effect on heart rate as compared to the placebo group.[21]

Lignocaine has been used for topical application by spraying 60 mg down the endotracheal tube prior to removal or 40 mg during removal of the tube with beneficial effects in reducing the heart rate and blood pressure during extubation.[22]

Winkel E and Knudsen J compared intubation with dry endotracheal tubes and with endotracheal tubes applied with 1% cinchocaine jelly in 248 patients and they concluded that the use of 1% cinchocaine jelly was effective in reducing the post-operative coughing and sore throat. [9]

Other methods of application of lignocaine have been tried such as 10% lignocaine spray on the distal end of the endotracheal tube prior to intubation and 2% lignocaine jelly application on the distal end of the tube.[5] Modified endotracheal tubes which permits spraying of local anaesthetic through its wall onto the laryngotracheal mucosa, reduces endotracheal tube induced coughing without prolonging the emergence.[5]

Several studies indicate that intravenous lignocaine in a dose of 1-2mgkg $^{-1}$ transiently suppresses coughing and other airway reflexes in humans in different clinical setting but certain features limits its clinical usefulness in suppressing the cough response during emergence from general anaesthesia. First, plasma lignocaine levels needed to effectively suppress cough are fairly high (3mgml $^{-1}$). Secondly systemic lignocaine produces sedation and prolongs emergence from anaesthesia. Hence the optimal timing of administration of lignocaine is difficult to ascertain.

Gonzalez and others in one of their studies concluded that the common complications during tracheal extubation are coughing, oxygen desaturation and airway obstruction. The incidence of cough during extubation may be as high as 96%.[5] Search for a technique that will allow patients to tolerate an endotracheal tube, while allowing them to maintain airway protection with intact supraglottic reflexes will be desirable. Maneuvers like intravenous administration of narcotics or local anaesthetics prior to extubation are associated with postoperative respiratory depression. The benefit of topically applied drugs before tracheal intubation is limited.

Asai and colleagues did a prospective study in 1005 patients undergoing general anaesthesia, to assess the respiratory complications seen during tracheal intubation as well as extubation. Common complications after extubation were coughing (incidence 6.6%), oxygen desaturation (SpO_2 <90%),(incidence 2.4%) and in the recovery room airway obstruction (3.8%) and coughing (3.1%). The incidence of respiratory complications after extubation was higher than during intubation. Coughing in itself is not a complication as it serves as a physiological protective mechanism against aspiration. It can however be associated with increased heart rate, blood pressure and when there is associated airway obstruction and oxygen desaturation it is regarded as a complication. [23]

The mechanism of coughing is presumed to be irritant or stretch stimuli in the trachea caused by the endotracheal tube and its cuff. Rapidly acting receptors are found throughout the trachea and are primarily superficial and thought to be the irritant receptors involved in the cough reflex.[23] Most stimulation may come from the cuff itself rather than the tube as it in closest contact with tracheal mucosa.

Gonzalez and others in one of their study stated that inflating the cuffs of endotracheal tubes with varying concentrations of lignocaine been used to decrease the stimulation caused by the cuff [5]. The cuff of an endotracheal tube is permeable to local anaesthetics, including lignocaine, allowing the cuff to act as a potential reservoir of local anaesthetics and enabling subsequent diffusion and anaesthesia of underlying mucosa.

Endotracheal tubes are made of polyvinyl chloride which is primarily hydrophobic. The thin polyvinyl chloride membrane, which constitutes the tube cuff,

allows simple diffusion of lignocaine across it. Sconzo demonstrated that 4% lignocaine placed in the cuff of the endotracheal tube diffused across the cuff membrane.[11] Assuming the co-efficient of diffusion and the thickness of the material to be standard across a specific range of endotracheal tubes, the limiting factors for diffusion are lignocaine concentration and time.

In vitro studies were done by Waka Hirota using tracheostomy tube cuffs inflated with 5ml lignocaine 4% solution and air at 20 cmH_2O and then placing them in 20 ml distilled water at 37°C . 100µL of this water was subsequently sampled over a period of 24 hours. The concentration of lignocaine in the samples was measured by high performance liquid chromatography. Thirty and sixty minutes after cuff inflation; lignocaine concentrations in the water bath reached approximately 8 and 17 $\mu g\ ml^{-1}$ respectively. Lignocaine concentrations of 140µM are required to produce a 50%reduction in Na^+ channel activity. The above data suggest that a minimum time period of 2 hours would be required to attain these concentrations.[24] Lignocaine has also been used in vivo to inflate endotracheal tube cuffs and its effects were studied.

Fagan and colleagues compared 4%lignocaine with saline and air in 63 patients undergoing elective surgery. They found no difference in hemodynamic data between the 3 groups but the incidence of coughing was decreased in lignocaine group for the time period of 4-8min post extubation.[10]

In the year 2004 Soltani HA and Aghadavoudi O. performed a study to compare different methods of lidocaine application and their effectiveness in reducing postoperative cough and sore throat. They concluded that lidocaine filled endotracheal tube cuffs, intravenous lidocaine administered before extubation and topical lidocaine

would reduce the incidence of postoperative coughing and sore throat [8].

Using lignocaine to inflate the endotracheal tube cuff also decreases the severity of postoperative sore throat at one hour and both the incidence and severity at 24 hours [25]. Lignocaine time dependently diffuses across tracheostomy tubes. In patients who received a tracheostomy tube with lignocaine containing cuff, tube discomfort was significantly reduced ($p < 0.01$) as evaluated by visual analogue scale.[26]

Lignocaine 10% has also been compared with saline in endotracheal tube cuffs [26]. There were fewer disturbed hemodynamic response and lesser incidence of bucking during tracheal extubation. Lignocaine was also effective in reducing the incidence and severity of postoperative sore throat. Plasma concentrations did not reach toxic levels.[26] Huang C.J and colleagues demonstrated that prefilling of an endotracheal tube cuff with lignocaine for a fixed duration before the test period can increase diffusion across the cuff.[27]

Dollo G and others conducted a study in which only the hydrophobic neutral base form was able to diffuse while for the charged hydrochloride form only a permeation phenomenon occurred concerning only 1% of total drug. In vivo studies by the same group of people showed alkalinized lignocaine diffused faster across the cuffs as compared to plain lignocaine leading to better tolerance of ET tubes.[28]

The toxicity of local anaesthetics must be considered regardless of the route of administration. During intracuff instillation of lignocaine the concerns are twofold, the risks of systemic absorption and the consequences of cuff damage with subsequent leakage of lignocaine into the bronchial tree. Carl Fagan and others used 4% lignocaine to inflate endotracheal tube cuffs with a mean volume of 6.1 ±0.9 ml per tube (244 mg ±

36 mg) which was much below the toxic levels. The incidence of the cuff rupture is also very low.[10]

Estebe JP and colleagues stated that alkalinization of lignocaine prior to filling the ETT cuff has been associated with smoother emergence during tracheal extubation when compared to lignocaine alone. Plasma lignocaine levels were much higher when lignocaine was alkalinized as compared to when it was used alone.[29]

Wetzel and others studied endotracheal emergence in smokers undergoing procedures less than 1.5 hours and concluded that the study did not show a correlation between the use on intracuff lignocaine and a decrease in emergence coughing in procedures lasting less than 1.5 hours.[30]

P A Sumithi and colleagues compared betamethasone jelly with lignocaine jelly applied over endotracheal tube cuff to reduce postoperative sore throat, cough and hoarseness, and concluded that the incidence of sore throat and coughing was lower in the betamethasone group than the lignocaine group.[31]

BASIC SCIENCE

Lignocaine [32] was synthesized by Lofgren in Sweden in 1943. Anaesthetic properties of lignocaine were discovered in 1948 by Lofgren and Lundquist. It was introduced into clinical practice by Gordh in 1949. It is an amide type of local anaesthetic. It also has anti-arrhythmic action apart from its local anaesthetic action.

CHEMICAL STRUCTURE.

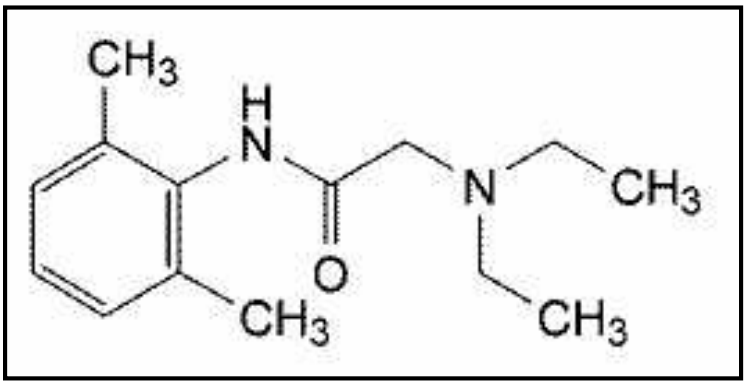


Figure 1 : Lignocaine Chemical Structure

Lignocaine is grouped under amides of amino acids with aromatic amines-aminoacylamides (N-diethyl amino acetyl 2, 6, xylidine hydrochloride monohydrate). It contains a tertiary amine attached to an aromatic system by an intermediate chain. The tertiary amine is the base. The chain contains amide linkage. The aromatic ring is lipophilic, where as the tertiary amine end is relatively hydrophilic, lignocaine is 65% protonated at pH 7.4.

Molecular weight of the base is 234 and that of hydrochloride salt is 270, pKa at 25 degree Celsius being 7.9, partial coefficient 29 and protein binding 64. It is stable and not decomposed by heat and alkali. It is having moderate potency and good penetration as a local anaesthetic.

MECHANISM OF ACTION

The action of the local anaesthetic is on the cell membrane of the axon on which it produces electrical stabilization. The large transient increase in the permeability to sodium ions, necessary for propagation of the impulse is prevented, thus the resting membrane potential is maintained and depolarization in response to stimulation is inhibited. Threshold for electrical stimulation is raised, rate of rise of action potential reduced and conduction closed, eventually propagation fails.

Local anaesthetics block the sodium conductance probably by a dual action on the cell membrane.

1. They act directly on receptors within the sodium channels.
2. They produce non-specific membrane expansion.

ACTION ON SODIUM CHANNELS

This action accounts for about 90% of the nerve blocking effect. Diffusion of the drug is a function of tissue binding and removal is by the circulation. The local anaesthetic permeates the axon membrane and equilibrates with axoplasm. The speed and the extent of these processes depend on particular drug's pka and lipophilicity of its base and cation species. It blocks the voltage gated sodium channels by inhibiting conformational changes that underline channel activation. It causes both phasic (frequency dependent) and tonic (resting) inhibition.

MEMBRANE EXPANSION

This is a non-specific action and analogous to the electrical stabilization produced by a number of nonpolar, lipid soluble substances such as non ionized barbiturates and general anaesthesics. There is 3.5% expansion of the membrane volume but the actual volume of the anaesthetic occupying the membrane however is only about 0.3% or less. So, a number of mechanisms have been suggested for membrane expansion. The most likely one is that there is an unfolding of membrane proteins together with a disordering of the lipid component of the membrane, with consequent obstruction of the sodium channel. Displacement of the membrane bound calcium channel may also be involved.

PHARMACODYNAMICS

LOCAL: Nerve blockade or direct effect on smooth muscle, Vasodilation.

REGIONAL: Loss of pain, Touch, Temperature, Motor power and vasomotor tone.

SYSTEMIC EFFECTS are due to systemic absorption or I.V administration.

1. C.V.S: It stabilizes electrical activity of any excitable tissue. It stabilizes aberrant conduction. It depresses the automacity in abnormal or damaged fibers and suppresses cardiac arrhythmias. It causes vasoconstriction at lower concentration and vasodilatation at higher concentration due to stimulation and inhibition of calcium release.
2. C.N.S: It produces sedation, light headedness, while sometimes anxiety and restlessness occur. With more toxicity numbness of tongue, peri-oral twitching and visual disturbance can occur. Severe toxicity proceeds to convulsion and coma with cardio-respiratory depression as a result of medullary depression.
3. AUTONOMIC NERVOUS SYSTEM: Preganglionic sympathetic blockade leading to vasodilation.

PHARMACOKINETICS:

Lignocaine is poorly absorbed orally (35%), but intramuscular injections result in peak levels within 30 minutes. Protein binding is 33% to 66% and tissue distribution is predominantly to the highly perfused tissues, volume of distribution is 1.7Lkg-^{1}, t ½ α-1 minute, t ½ β-9.6 minute, t ½ χ-1.6 hr, and clearance 0.95Lmin $^{-1}$.

It is metabolized in liver by microsomal enzymes. The initial reaction in this process is dealkylation of lignocaine in the following sequence….

1. Monoethylglycine ⟶ Xylidide (MEGX)
2. The second product of major pathway of metabolism is formation of 2, 6-xylidine.
3. This is further metabolized by hydroxylation of the ring structure which is conjugated forming 4-hydroxy 2, 6-xylidine which is the major urinary metabolite.
4. A minor pathway which gives the metabolite monoethylglycine-xylidide to glycine-xylidide.

Both the end products of metabolism are recognized to have anti arrhythmic properties which are equivalent to lignocaine. The toxic effects may be due to these products. Glycine-xylidide appears to have central effects. It causes headache and altered mental performance. It also potentiates the convulsive activity of monoethylglycine-xylidide and has central nervous system depressant activity.

TOXICITY OF LIGNOCAINE	
Serum level	**Clinical effect/Toxic effect.**
2 $\mu g ml^{-1}$	Antiarrhythmic.
3 $\mu g ml^{-1}$	Perioral and tongue numbness.
4 $\mu g ml^{-1}$	Light headedness and tinnitus.
6 $\mu g ml^{-1}$	Visual disturbances.
8 $\mu g ml^{-1}$	Muscle twitching
10 $\mu g ml^{-1}$	Convulsion
12 $\mu g ml^{-1}$	Unconsciousness
15 $\mu g ml^{-1}$	Coma
20 $\mu g ml^{-1}$	Respiratory arrest
25 $\mu g ml^{-1}$	Cardiovascular depression.

CLINICAL APPLICATIONS OF LIGNOCAINE

1. Lignocaine is used as an effective local anaesthetic for local infiltration, nerve blocks, subarachnoid block, and epidural blocks and as a topical anaesthetic agent.

2. Lignocaine is also used as an antiarrhythmic in premature ventricular complexes, ventricular tachycardia following acute myocardial infraction.

PRECAUTION TO BE TAKEN LIGNOCAINE USAGE

1. ECG monitoring for intravenous administration.

2. During epidural anaesthesia a test dose is recommended.

3. Should be used with caution in advanced heart failure, Hepatic disease, Hypovolemia, Heart block, Stokes Adams syndrome, Shock, Renal disease, Sinus bradycardia.

4. Should be used with caution in elderly patients undergoing intraurethral instillation as they are tonic-clonic seizures.

5. Caution in patients with spinal deformities, preexisting neurological disease, septicemia and severe hypertension undergoing spinal and caudal-epidural anaesthesia.

6. Preparations containing preservatives should not be used intrathecally and intravascular.

Presentation:

Lignocaine 2% jelly.

Store lignocaine 2% jelly at controlled room temperature 20 to 25 degree Celsius or 68 to 77 degree Fahrenheit.

ANATOMY OF LARYNX

Larynx lies at the level of third to sixth cervical vertebrae and is composed of a framework of cartilages interconnected by muscles and ligaments. The cartilages are Thyroid, Cricoid, and Epiglottis is unpaired, while Arytenoid, Corniculate and cuneiform are paired cartilages.

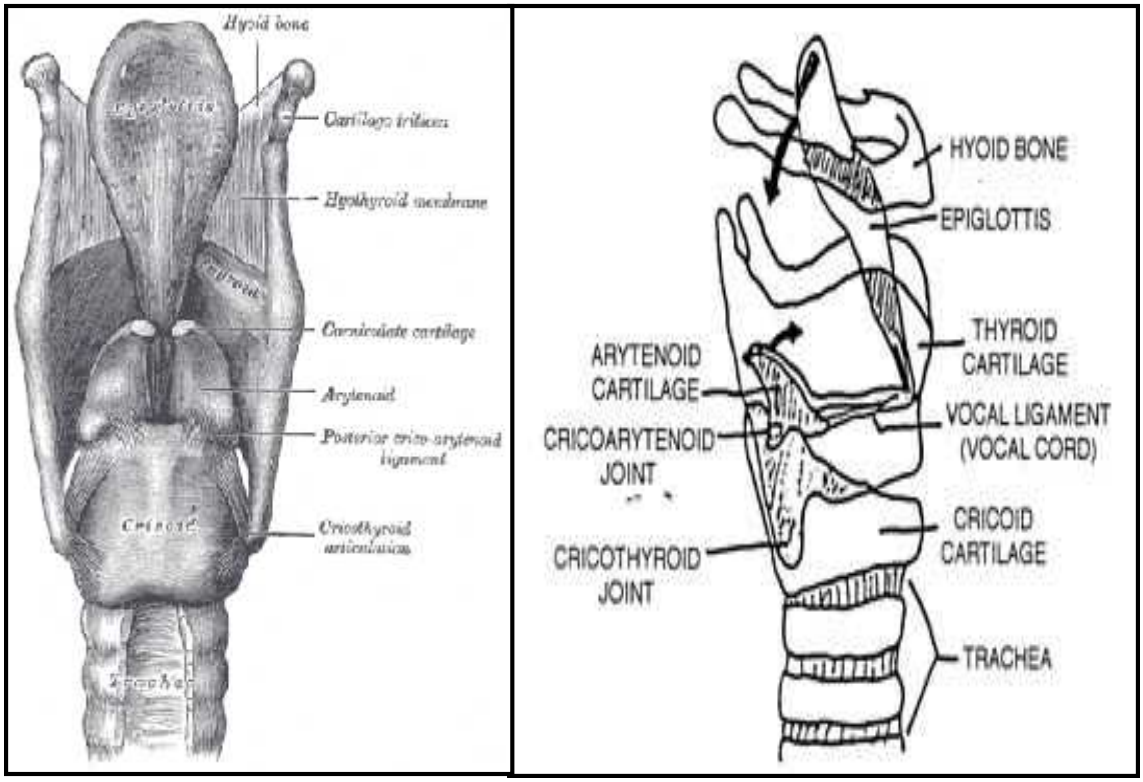


Figure 2 :Anatomy of Larynx

Thyroid cartilages:

It is the largest cartilage of the larynx. It is often described as "shield shaped" and consists of two laminae right and left. The laminae are placed obliquely relative to the midline. Their posterior borders are far apart, but the anterior borders approach each other at an angle of 90° in males and 120° in females. The lower parts of the anterior border of the two laminae fuse and form a median projection called the laryngeal prominence. The upper part of the anterior borders does not meet. They are separated by the thyroid notch. The posterior borders are free. They are prolonged upwards and downwards as superior and inferior cornu. The superior cornu is connected with the greater cornu of hyoid bone. The inferior cornu articulates with the cricoid cartilage to form the cricothyroid joint.

Cricoid cartilage:

This cartilage is shaped like a signet ring. It encircles the larynx below the thyroid cartilage. The ring has narrow anterior part called the arch and the broad posterior part called lamina. The lamina projects upwards behind the thyroid cartilage and articulates superiorly with the Arytenoid cartilages. The inferior cornu of thyroid cartilage articulates with the side of cricoid cartilage at the junction of arch and lamina.

Epiglottis cartilage:

It is a leaf shaped cartilage placed in the anterior wall of the upper part of the larynx. The upper end is broad and free and projects upward behind the hyoid bone and the tongue. The lower end is attached to the upper part of the angle between the two laminae of the thyroid cartilage. It has a mucous membrane covering that reflects as a

glossoepiglottic fold onto the pharyngeal surface of the tongue. On either side of this fold are depressions called valleculae. The epiglottis projects into pharynx and overhangs the laryngeal inlet.

Arytenoid cartilages:

These are two small pyramid shaped cartilages. The apex articulates with the Corniculate cartilage and the base articulates with the upper border of cricoid lamina.

Corniculate cartilages:

These are two small cartilages which articulate with apex of the arytenoids cartilages and lie in the posterior part of the aryepiglottic folds.

Cuneiform cartilages:

These are small cartilages placed in the aryepiglottic folds just ventral to the corniculate cartilages.

Muscles of the larynx: These are divided into,

- Extrinsic muscles.
- Intrinsic muscles.

There are a number of extrinsic muscles, including sternothyroid and thyrohyoid muscles. From their position between the larynx and surrounding structures, these are responsible for moving the larynx. The omohyoid elevates the hyoid, there by raising the larynx.

The intrinsic muscles include the cricoartenoid, interartenoid, thyroartenoid and the cricothyroid muscles. Their role is to move the cartilages within the larynx. The

results of these movements are principally to adduct, abduct and adjust the tension in the vocal folds.

Nerve supply:

The nerve supply to the larynx is through the right and left superior laryngeal nerve and recurrent laryngeal nerves, all of which are branches of vagus nerve.

Functions of larynx: It serves two main functions.

- It is a protective valve at the upper end of trachea.
- It is the organ of speech [33].

ENDOTRACHEAL TUBES

These are the tubes through which the anaesthetic gases or vapors along with the breathing gases are conveyed to and from the trachea.

Description:

An endotracheal tube has two ends. The distal end which is beveled is called patient end and the proximal end which is vertically cut is called machine end.

Some endotracheal tubes have a side hole just above and opposite the bevel called Murphy eye. It helps ventilation to occur if the bevels occluded by secretions, blood or the tracheal wall.

In some endotracheal tubes there is a radiopaque marker at the tip or along the length of the tube to detect the position of the tube after intubation.

Various substances like neutral rubber, synthetic rubber, silicon rubber, nylon, teflon, plastic, polyethylene and polyvinyl chloride (PVC) are used for manufacturing endotracheal tubes. Of these synthetic rubber and PVC are most widely used.

To meet the standards of American society for testing and materials (ASTM), materials must pass a United States of Pharmacopeia (USP) implantation.

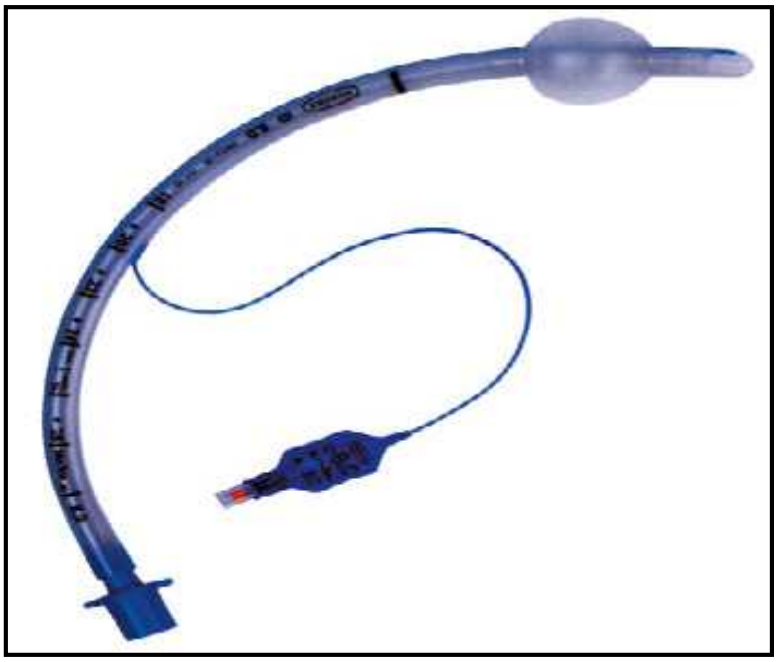


Figure 3 : Endotracheal tube

Types of endotracheal tubes:

I. Depending on the route of intubation.
   - Oral tubes
   - Nasal tubes

II. Depending on presence or absence of cuff.
   - Uncuffed or plain tubes
   - Cuffed tubes

- High volume low pressure cuffed tubes
- Low volume high pressure cuffed tubes

The oral tubes are short and curved with a radius of curvature 14 cm. The bevel angle should not be less than 45° in relation to its long axis.

The nasal tube is usually long and curved with the radius of curvature of 28 cm. Its bevel angle should not be less than 30° in relation to its long axis[34].

Cuff system:

A cuff system consists of the cuff itself and the inflating system which typically includes an inflating lumen in the wall of the tube, an external inflating tube, a pilot balloon and an inflating valve.

Functions:

- To provide the seal between the tube and tracheal wall to prevent the aspiration and gas leak during the positive pressure ventilation.
- To centre the tube in the trachea so that its tip is less likely to traumatize the mucosa.

Cuff:

It is an inflatable sleeve near the patient end of the tube.

Characteristics of cuff:

- The cuff material should be strong, thin, soft and pliable but tear resistant.
- Cuffs are usually made of the same material as that of the tube.

- The edge of the cuff should not encroach on the bevel or Murphy eye (in case of Murphy tubes).
- It should inflate symmetrically.

Types of cuff:

- Low volume high pressure cuff.
- High volume low pressure cuff.

Low volume high pressure cuff:

- It has the small diameter and small residual volume at rest.
- It requires a high intracuff pressure to achieve the seal with trachea.
- The pressure exerted on the wall of the trachea is difficult to ascertain but will be well above the mucosal perfusion pressure.
- Intracuff pressure and the lateral pressure on the tracheal wall increases sharply as volume is incremented to the cuff.

Advantages

Less expensive.

Can be reused.

Offer better protection against aspiration.

Offer better visualization while intubating.

Disadvantages

Ischemic damage to trachea due to the high intracuff pressure.

High volume low pressure cuffs:

- It has large resting volume and large diameter and thin compliant wall.
- It requires low intracuff pressure to achieve the seal with trachea.
- As the cuff pressure increases the cuff adapts to the shape of the tracheal wall.

Advantages

- Intracuff pressure approximates the pressure on tracheal mucosa and can be used to estimate pressure on the trachea.
- Less chances of cuff pressure induced complications.
- It is relatively easy to pass devices like temperature probes around the low pressure cuffs.

Disadvantages

- Costly
- Chances of aspiration and gas leak are relatively more.

ENDOTRACHEAL INTUBATION

History:

- Intubation of animal trachea was first done by A. Vesalius in 1543.
- Human endotracheal intubation was first done by Curry in 1792.
- Magill modernized the endotracheal anaesthesia in 1920.
- Rowbotham was the first to perform blind nasal intubation in 1920.
- Waters and Guedal in 1928 introduced the endotracheal cuff tubes.

Indications:

- Surgery on the head and neck.
- Protection of the respiratory tract.

- During anaesthesia using IPPV and muscle relaxation.
- To facilitate suction of the respiratory tract.
- Thoracic surgery
- Cardiopulmonary arrest.

Preparations:

Availability and function of the following equipments should be checked.

- Laryngoscope
- Tracheal tubes
- Stilette
- Magill forceps
- Securing tape
- Catheter mount
- Lubricant jelly
- Throat packs
- Anaesthetic breathing system and face masks

Anaesthesia for endotracheal intubation:

Adequate depth of anaesthesia is necessary to depress the laryngeal reflexes and provide adequate degree of muscle relaxation. This can be achieved with either of the following,

- **Local anaesthesia**
    - Topical spray
    - Transtracheal spray
    - Superior laryngeal nerve block

- **Inhalational anaesthesia**

  Required depth can be achieved with halothane up to 4% or sevoflurane up to 8%. This may be followed by use of nondepolarising relaxant.

- **Intravenous anaesthesia**

Patient is induced with one of the induction agents like, thiopentone or ketamine or propofol. This is followed by administering depolarizing or nondepolarising muscle relaxant.

Process of intubation:

Head positioning:

The correct position for the head is "sniffing position" with the neck slightly flexed and head extended. One places a pillow under the head and neck but not under the shoulders. This allows a straight line of vision from the mouth to the vocal cords.

Laryngoscopy:

The laryngoscopy is held in the left hand and introduced into the right hand side of the mouth. The tongue is swept to the left and the tip of the blade is advanced until a fold of cartilage is visualized at 12 O` clock. This is the epiglottis, and this sits over the glottis.

The tip of the blade is advanced to the base of the epiglottis, known as the vallecula, and the entire laryngoscope is lifted upwards and outwards. This flips the epiglottis upwards and exposes the glottis below. An opening is seen with two white vocal cords forming a triangle on each side.

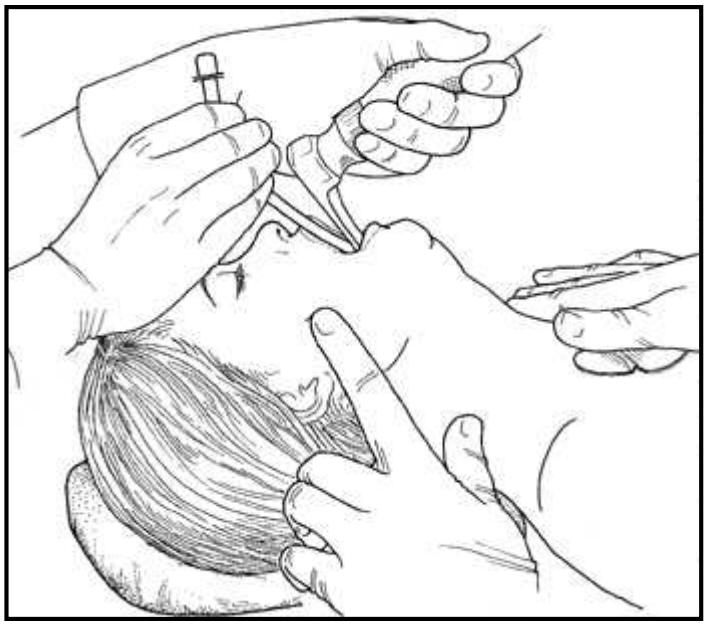


Figure 4 : Endotracheal Intubation

Intubation:

The tip of the endotracheal tube is advanced through the vocal cords to the sufficient length. The correct position of the tube is confirmed by auscultation. The tube is secured at this level and the cuff is inflated [35].

Complications:

The complications are usually due to airway trauma, tube malpositioning, physiological responses to airway instrumentation, or tube malfunction. These complications can occur during laryngoscopy and intubation, while the tube is in place, or following extubation.

During laryngoscopy and intubation:

- Malpositioning
    - Esophageal intubation
    - Endobronchial intubation
- Airway trauma
    - Tooth damage
    - Lip, tongue or mucosal injury
    - Sore throat
- Physiologic responses
    - Hypertension
    - Tachycardia
    - Laryngospasm
- Tube malfunction
    - Cuff perforation

While the tube is in place

- Malpositioning
    - Unintentional extubation
    - Endobronchial intubation
- Airway trauma
    - Mucosal inflammation and ulceration
- Tube malfunction
    - Ignition
    - Obstruction

Following extubation

- Airway trauma
    - Edema and stenosis (glottis, subglottic or tracheal)
    - Hoarseness of voice(vocal cord injury)
    - Cough
    - Difficulty in swallowing
- Physiologic reflexes
    - Laryngospasm

METHODOLOGY

After obtaining approval from institutional ethics committee. Informed consent of patients was taken after explaining the study to patient in there vernacular language. The study was carried out in KLE'S Prabhakar Kore Hospital and MRC, Belgaum and Prabhakar Kore Free Hospital, Belgaum. A total of 140 patients were studied, who were posted for general surgery, urological, gynacecological, neurological and orthopedic procedures. The duration of study was from December 2007 to December 2008.

Inclusion criteria

1. ASA grades I and II, Age between 18-65 years.
2. General anaesthesia for more than 60 minutes.
3. Patients undergoing General surgery, Urological, Gynacecological, Neurosurgical and Orthopedic procedures.

Exclusion criteria

1. Difficult airway: MP III/IV
2. H/S/O Gastro esophageal reflux.
3. Patients with h/o laryngeal or tracheal surgery and h/o asthma.
4. Patients who need nasogastric tube intraoperatively.
5. Patients who needed more than one attempt at intubation.

Sample size

Sample size calculated by considering incidence of coughing as 38% with type I error rate as α =0.05 and type II error rate of β=0.02 with a power of 80%, and using formulae

$$N=\frac{2(2\alpha+2\beta)^2 p(1-p)}{(P_1 - P_2)^2}$$

A total sample of 140 patients. Out of which 70 will be in group (J) and 70 in group (I).

Duration of study : 1 Year

Study design : Randomized double blind study.

Methodology

After having met inclusion and exclusion criteria, all patients were visited the evening before surgery and written informed consent obtained from the patient. Subjects were allocated into two groups , Group(J) were the endotracheal tube cuff was applied with 2% lignocaine jelly and Group (I) were the endotracheal tube cuff was filled with 4% lignocaine intracuff according to computer generated randomization sheet.

Standard monitors attached ECG, NIBP, SPO_2, $ETCO_2$. Study subjects were premedicated with Inj Glycopyrrolate 0.005mg/kg body weight, Inj Midazolam 0.05mg/kg body weight, Inj Fentanyl 1 microgram/kg body weight. Baseline hemodynamic parameters noted. Patients preoxygenated with 100% oxygen for 3 min. Patients induced with Inj Thiopentone 5mg/kg body weight, Inj Vecuronium 0.1mg/kg body weight to facilitate tracheal intubation.

Tube size of 7.5mm was used in females and 8.5mm in males. For group (J) 2% lignocaine jelly 2.5 grams (contains approximately 50mg of lignocaine) was applied , Endotracheal tube inflated with a syringe, with air as inflating agent and the cuff pressure set at 20 cm of water with a Rusch endotest cuff inflator. For group (I) the amount of air required to attain the set cuff pressure that is 20 cm of water measured and the same amount of 4 % lignocaine injected to inflate the cuff.

Anaesthesia maintained with Nitrous: Oxygen (35:65), Inj Vecuronium 1/4th the intubating dose depending upon the etco2 changes, with the use of intermittent Halothane 0.5 % for maintenance of anaesthesia. Lungs mechanically ventilated with tidal volume of 8-10ml/kg body weight. Pulse oximeter, Non invasive blood pressure, ECG and etco2 monitored.

At the end of surgery Nitrous oxide discontinued and 100% oxygen given. Patient taken on mapleson D circuit reversed with Inj Glycopyrrolate 0.005 mg/kg body weight and Inj Neostigmine 0.05 mg/kg body weight. Cuff pressure measured just before deflating the cuff and recorded, Extubation was performed after checking for spontaneous ventilation, ability to follow verbal commands (eye opening, tongue protrusion).

Immediately after extubation patient assessed for coughing, heart rate, blood pressure, and latter at 0 min, 1 min, 3 min, and 5 minutes. Coughing recorded as either occurred or not occurred by another anaesthesiologist who is not present at the time of intubation (Blinding).

Statistical analysis

Demographic data and hemodynamic data was analyzed by using Unpaired Student t test. The incidence of coughing compared using the Test of proportions.

Photo 1: 4% Lignocaine Topical Solution

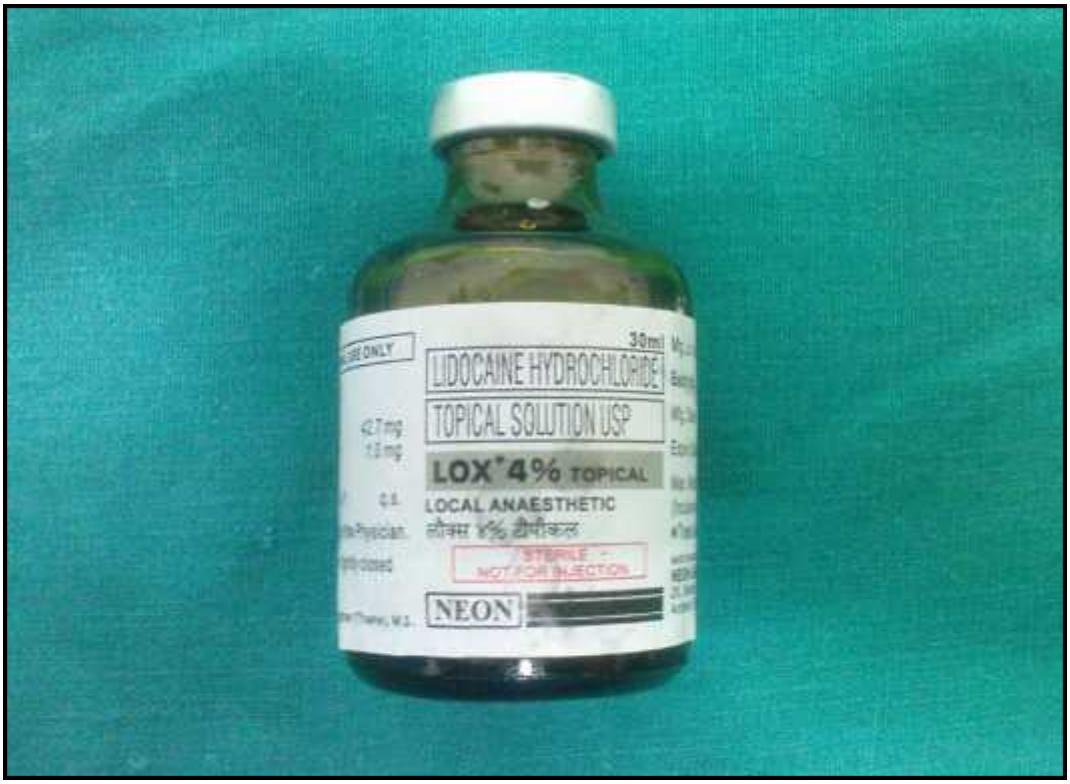
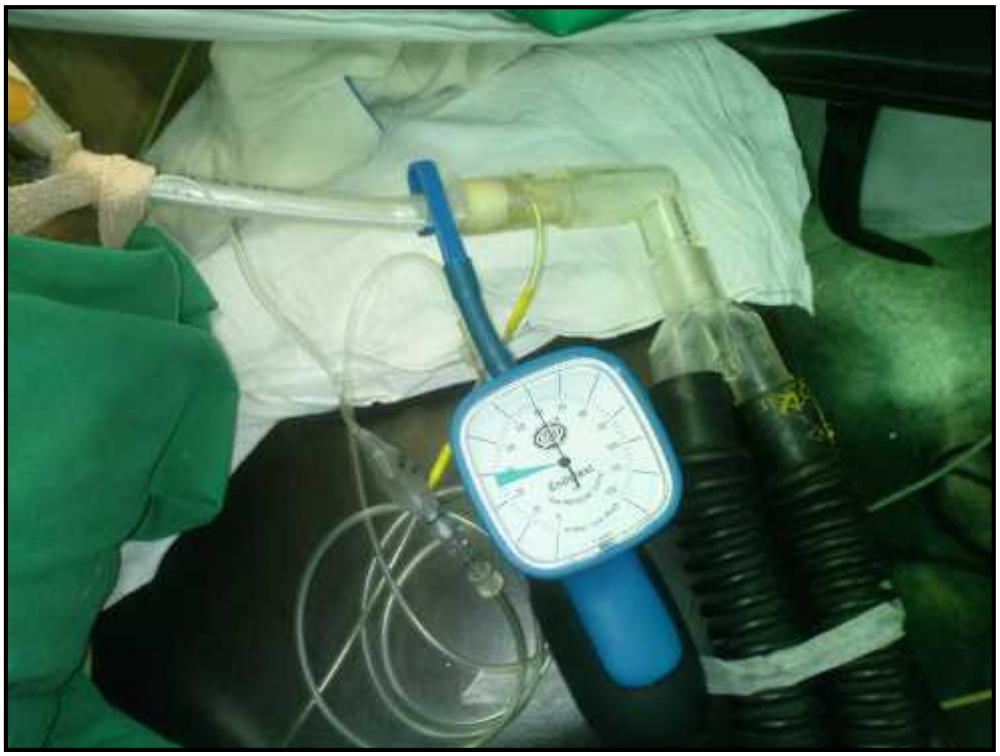


Photo 2: 2% Lignocaine Jelly

Photo 3: Rusch Endotest Cuff Inflator

Photo 4: Monitoring of Cuff Pressure



RESULTS

The objective of the present study was to compare the Emergence phenomena after General Anaesthesia with 2% lignocaine jelly and 4% lignocaine intracuff. The study was carried out in KLES Prabhakar Kore Hospital & MRC, Dept of Anaesthesiology, Belgaum, in between the period December 2007 to December 2008.

The study included 140 ASA grade 1and 2 in the age group of 18 to 65 years. Each group consisted of 70 patients and were divided as group J (Lignocaine jelly n=70) and group I (Intracuff Lignocaine, n =70) by a computer generated randomization table.

Data was collected in both groups and observations of the analyzed data are presented in the tabular form as follows.

Table No. 1 : Distribution of Age and Weight

GROUPS	AGE IN YEARS MEAN±SD	WEIGHT IN KGS MEAN±SD
Group J	41.37±13.76	55.71±8.96
Group I	40.47±13.74	56.36±8.86
'p' value	0.6992	0.6701

*Significant `p` value.

The mean age in group J was 41.37±13.76 and in group I was 40.47±13.74. The mean weight in group J was 55.71±8.96 and in group I was 56.36±8.86 and was comparable, with insignificant `p` value (table no 1)

Table No. 2 : Distribution of Sex.

GROUPS	MALE	FEMALE
GROUP J	36	34
GROUP I	36	34
'p' value	1.000	

*Significant `p` value.

The distribution of sex was similar in both the groups with 36 males and 34 females. Both the groups were comparable and were statistically insignificant.

Table No. 3 : Distribution of ASA Grade

GROUPS	ASA I	ASA II
GROUP J	18	52
GROUP I	12	58
'p' value	0.217	

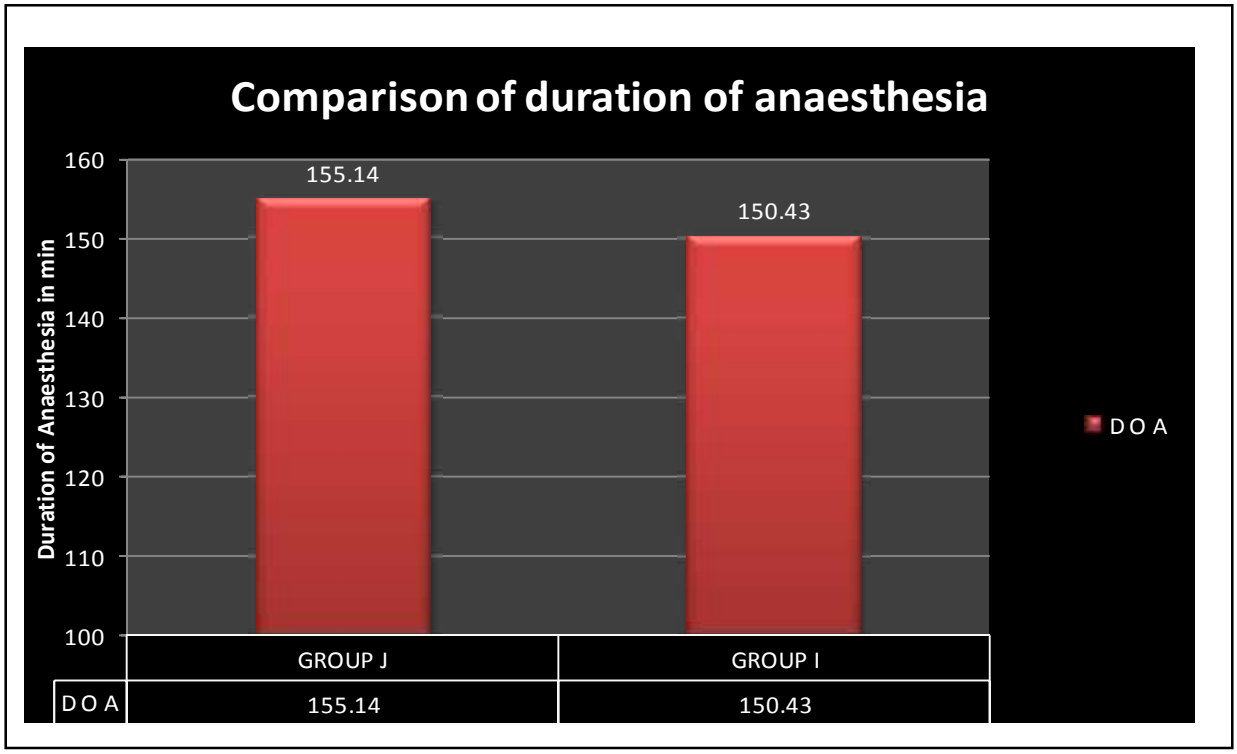
* significant `p` value.

Distribution of patients according to ASA grade was 18 and 52 in group J in ASA I and II respectively.12 and 58 in group I with a non significant P value.

Table No. 4 : Comparison of Duration of Anaesthesia

GROUPS	DURATION IN MINUTES
Group J	155.14±73.46
Group I	150.43±63.67
'p' value	0.6856

*significant `p`value.

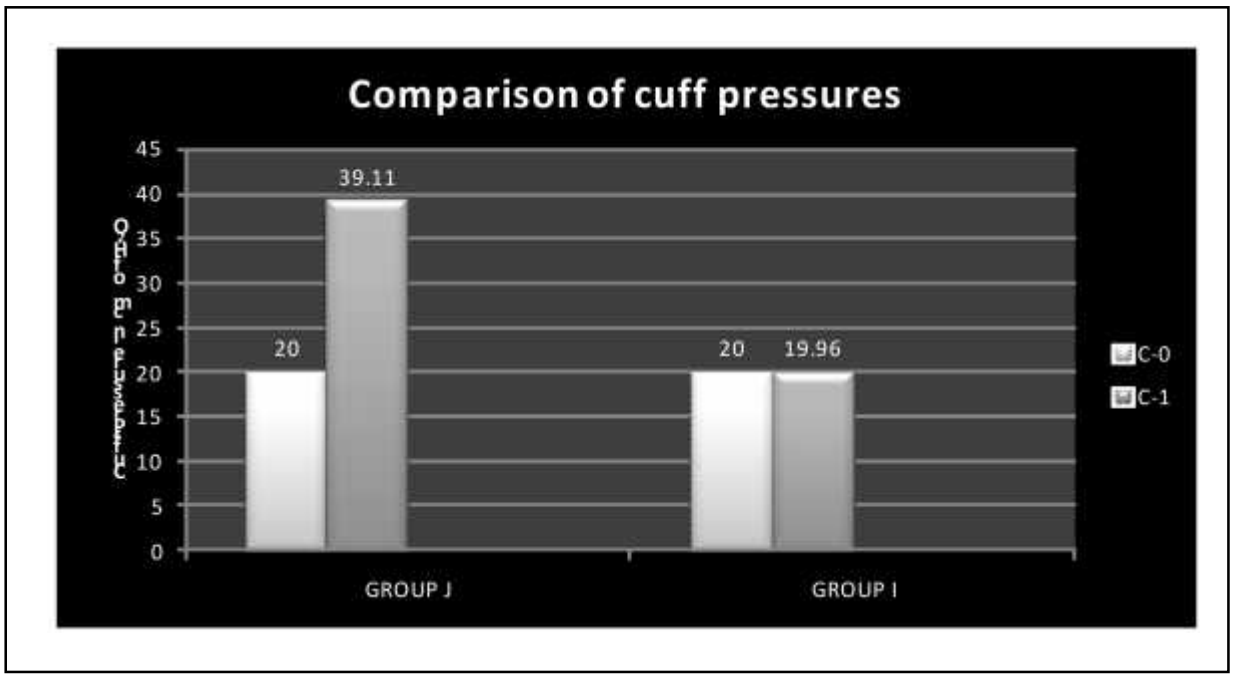


The mean duration of anaesthesia in the group J was 155.14±73.46 and in group I was 150.43±63.67 were comparable and an insignificant `p` value (0.6856) (table no 4).

Table No. 5 : Comparison of Cuff Pressure

GROUPS	CUFF PRESSURE (START)	CUFF PRESSURE (END)
Group J	20.00±0.00	39.11±7.76
Group I	20.00±0.00	19.96±0.77
'p' value	1.000	<0.001*

*significant `p` value.



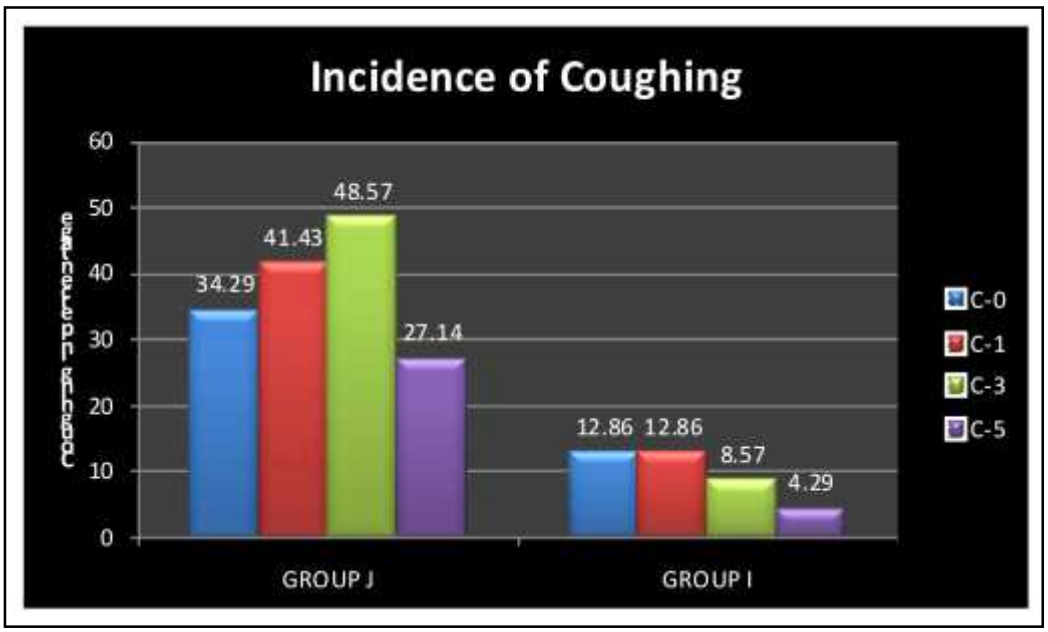
In group J the mean cuff pressure at the end of surgery was 39.11±7.76. In group I the mean cuff pressure at the end of surgery was19.96±0.77. The groups were incomparable and had a significant `p` value (<0.001)

Table No. 6 : Comparison of Incidence of Cough (percentage).

GROUP	C-0	C-1	C-3	C-5
Group J	34.29	41.43	48.57	27.14
Group I	12.86	12.86	8.57	4.29
'p' value	0.003*	<0.001*	<0.001*	<0.001*

*significant `p`value.

C-0 – Coughing immediately after extubation.**C-1, C-3, and C-5** – Coughing at 1minute, 3 minute, and 5 minutes respectively.



Twenty four patients (34.29%) had coughing immediately in lignocaine jelly group as compared to 9 patients (12.86%) in intracuff lignocaine group. The incidence of coughing at 1 minute was 41.43% in lignocaine jelly group and 12.86% in intracuff lignocaine group. At 3 minutes the incidence of coughing in lignocaine was 48.57% compared to 8.57% in lignocaine intracuff group. At 5 minutes the incidence of coughing came down to 27.14% in lignocaine jelly group and the incidence of coughing was 4.29% in lignocaine intracuff group. There was a significant statistical difference between the lignocaine jelly group and intracuff lignocaine group. The `p` value being 0.003, <0.001, <0.001, <0.001 is highly significant (table no 6).

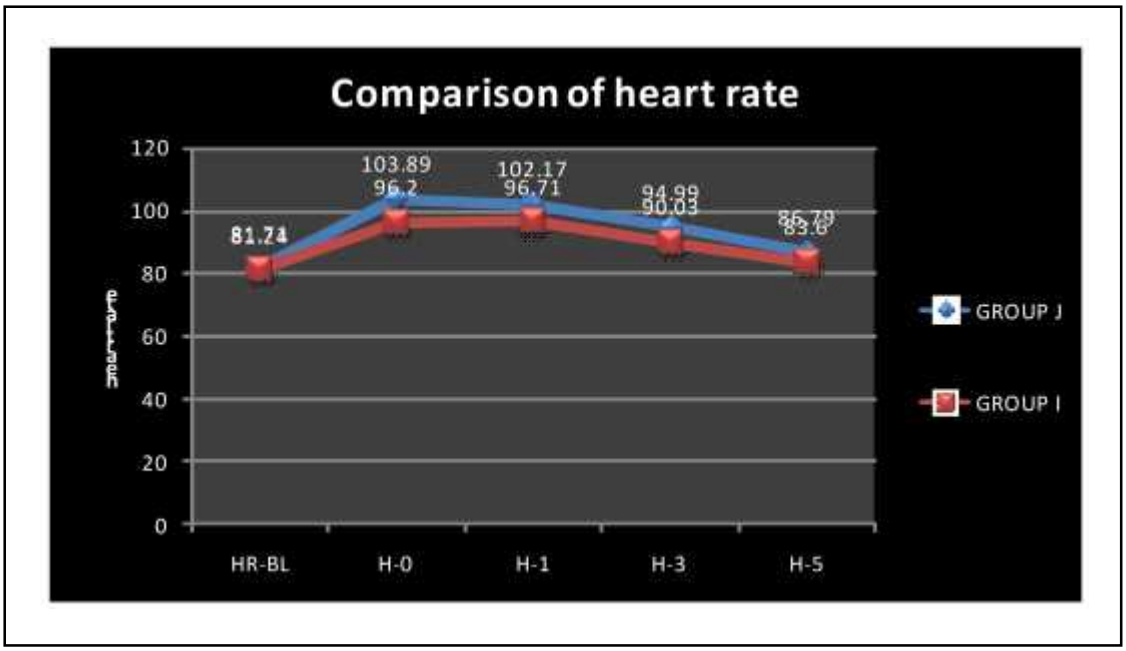
Table No. 7 : Comparison of Heart Rate

GROUPS	HR-BL MEAN±SD	H-0 MEAN±SD	H-1 MEAN±SD	H-3 MEAN±SD	H-5 MEAN±SD
Groupj	81.71±8.4	103.89±13.9	102.17±11.6	94.99±10.70	86.79±9.06
Group I	81.24±9.08	96.20±10.5	96.71±12.42	90.03±9.97	83.60±8.99
'p' value	0.7535	0.0003*	0.0083*	0.0053*	0.0386*

*significant `p`value.

HR-BL - Heart rate at baseline.

H-0, H-1, H-3, H-5 – Heart rate immediately after extubation, at 1minute, 3 minute and 5 minutes respectively.



The mean basal heart rate was 81.71±8.4 and 81.24±9.08 in group J and group I respectively. Basal heart rates were comparable and statistically insignificant. Mean heart rates at 0 minute, 1 minute, 3 minute and 5 minutes were 103.89±13.9, 102.17±11.6, 94.99±10.70 and 86.79±9.06 respectively for group J and 96.20±10.5, 96.71±12.42, 90.03±9.97 and 83.60±8.99 respectively for group I. The above heart rates were statistically significant (table no 7).

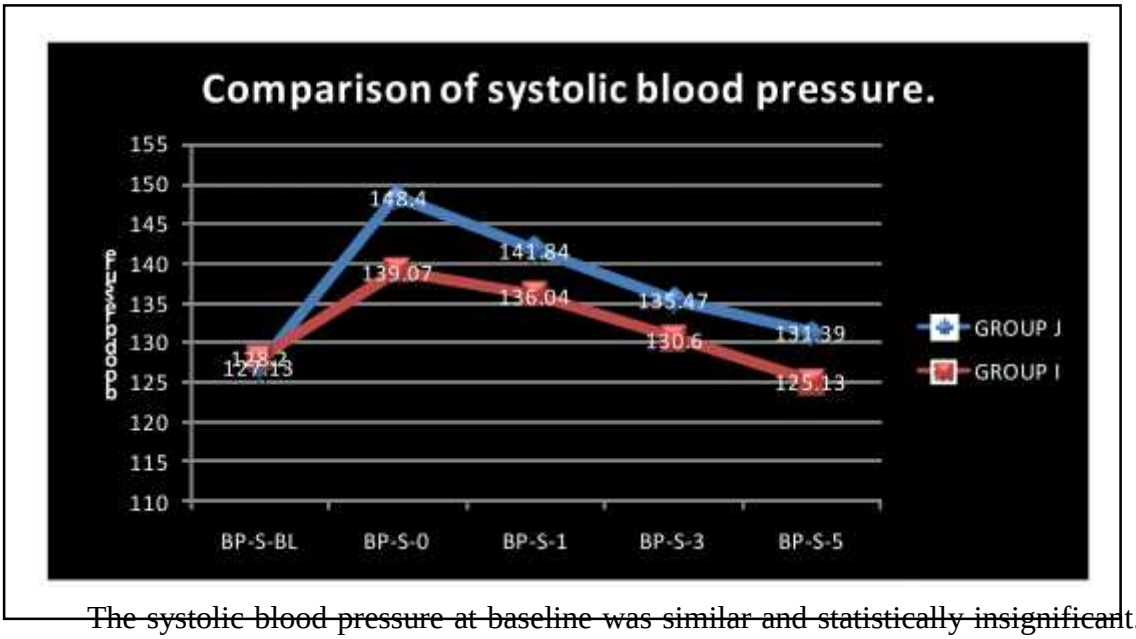
Table No. 8 : Comparison of Blood Pressure (Systolic)

GROUPS	BP-S-BL MEAN±SD	BP-S-0 MEAN±SD	BP-S-1 MEAN±SD	BP-S-3 MEAN±SD	BP-S-5 MEAN±SD
Group J	127.13±10.03	148.40±12.82	141.84±11.87	135.47±10.68	131.39±9.30
Group I	128.20±9.32	139.07±12.23	136.04±10.15	130.60±8.88	125.13±15.69
'p' value	0.5138	<0.001*	0.0023*	0.0039*	0.0047*

*significant `p`value.

BP-S-BL – Blood pressure systolic baseline.

BP-S-0, BP-S-1, BP-S-3, BP-S-5 – Blood pressure systolic immediately after extubation, at 1minute, 3minute and 5minutes respectively**.**



The systolic blood pressure at baseline was similar and statistically insignificant.

Systolic pressure at 0 minutes, 1 minute, 3 minutes and 5 minutes were incomparable and statistically significant when compared (table 8).

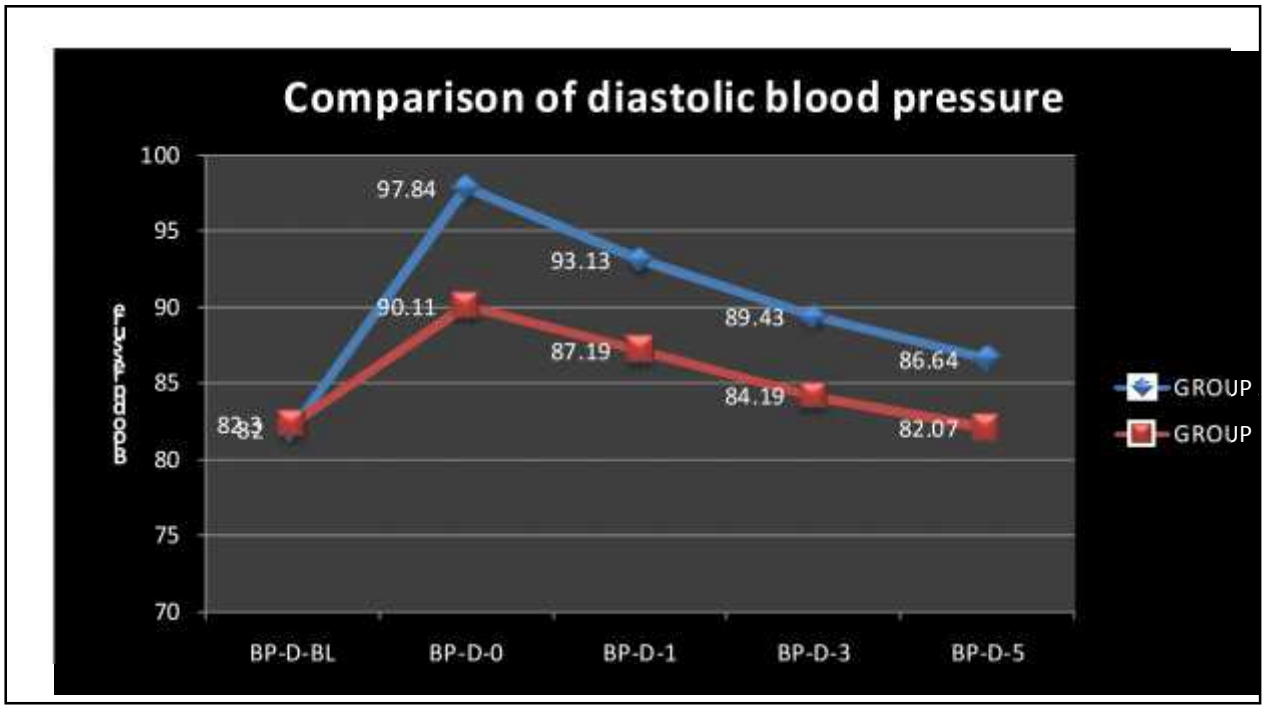
Table No. 9 : Comparison of Blood Pressure (Diastolic).

GROUPS	BP-D-BL MEAN±SD	BP-D-0 MEAN±SD	BP-D-1 MEAN±SD	BP-D-3 MEAN±SD	BP-D-5 MEAN±SD
Group J	82.00±7.99	97.84±9.09	93.13±7.60	89.43±6.42	86.64±5.76
Group I	82.30±7.00	90.11±8.54	87.19±7.29	84.19±7.21	82.07±6.25
'p' value	0.8135	<0.001*	<0.001*	<0.001*	<0.001*

*significant `p`value.

BP-D-BL – Blood pressure diastolic baseline.

BP-D-0, BP-D-1, BP-D-3, BP-D-5 – Blood pressure diastolic immediately after extubation, at 1minute, 3minute and 5minutes respectively.



The diastolic blood pressure at baseline was similar and statistically insignificant. Diastolic pressure at 0 minutes, 1 minute, 3 minutes and 5 minutes were incomparable and statistically significant when compared (table 9).

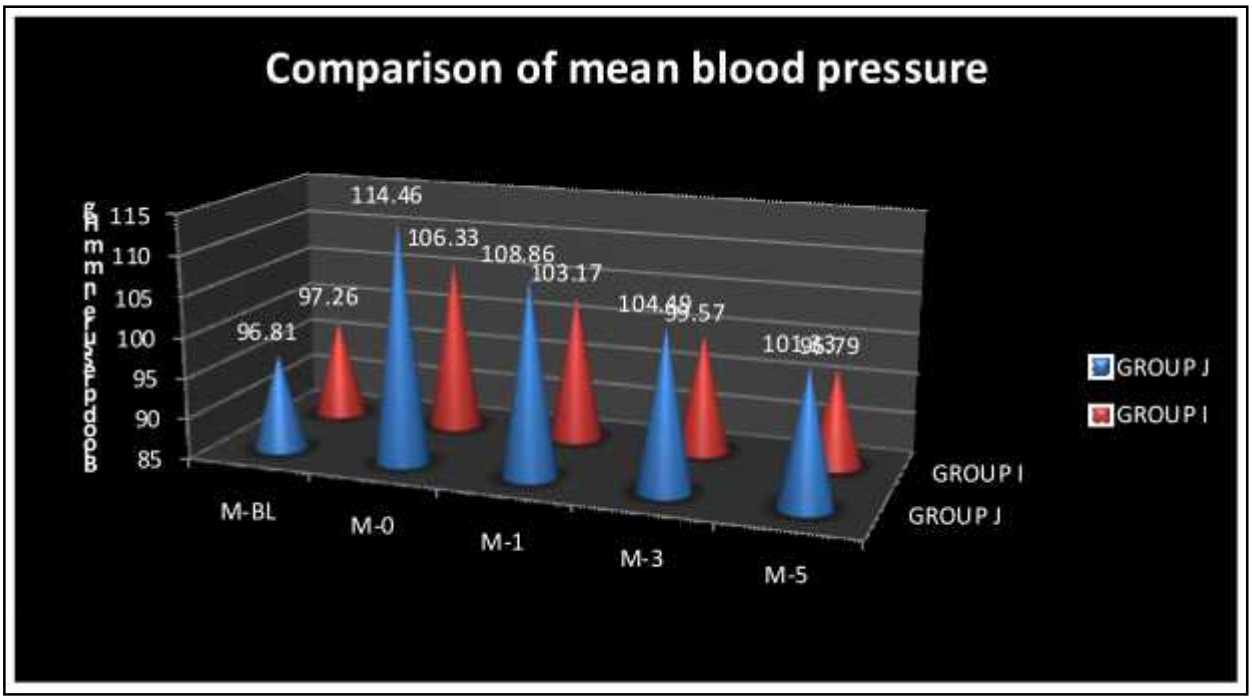
Table No. 10 : Comparison of Mean Blood Pressure

GROUPS	M-BL MEAN±SD	M-0 MEAN±SD	M-1 MEAN±SD	M-3 MEAN±SD	M-5 MEAN±SD
Group J	96.81±8.28	114.46±9.41	108.86±8.45	104.49±6.92	101.33±6.17
Group I	97.26±7.42	106.33±9.40	103.17±8.13	99.57±7.59	96.79±6.36
'p' value	0.7395	<0.001*	0.0001*	0.0001*	<0.001*

*significant `p`value.

M-BL – Mean arterial pressure at baseline.

M-0, M-1, M-3, and M-5 –Mean arterial pressure at immediately after extubation, 1minute, 3minute and 5 minutes respectively.



The baseline mean arterial pressures were comparable. The pressures were 114.46±9.41, 108.86±8.45, 104.49±6.92 and 101.33±6.17 at 0 minute, 1 minute, 3 minute and 5 minutes in group J. Similarly the pressures in group I were 106.33±9.40, 103.17±8.13, 99.57±7.59 and 96.79±6.36 respectively. These values were incomparable and highly significant statistically (table no 10)

DISCUSSION

Extubation of the trachea in a patient undergoing general anaesthesia is associated with its own problems and complications, which can be detrimental to the patient and unpleasant to the attendant anaesthesiologist. Coughing during emergence in a lighter plane of anaesthesia can result in hypertension, tachycardia, and myocardial ischemia, increased intraocular and intracranial pressures. These features are particularly undesirable in patients undergoing neurosurgical or ophthalmic procedures or those who are at an increased risk of adverse cardiovascular events. [16,17,18]

Numerous methods of attenuating cough reflex during tracheal extubation have been advocated such as use of narcotics, extubation in a deeper plane of anaesthesia, and use of I V lignocaine. Lignocaine has been used with varying success as a topical agent [22], for intravenous administration prior to extubation or in endotracheal tube cuffs. In one of the early studies Sconzo and colleagues demonstrated that 4% lignocaine diffuses across the cuff of endotracheal tubes [11]. This finding applied by Waka Hirota showed that when endotracheal tube cuffs were inflated with lignocaine, concentrations of lignocaine in a water bath would reach 8 and 17 $\mu g ml^{-1}$ after 30 and 60 minutes of cuff inflation respectively. Lignocaine concentration of 140µM are required to produce a 50% reduction in Na^+ channel activity .[24] This data suggests that a minimum period of 2 hours would be required for attaining these concentrations.

The toxicity of local anesthetics must be considered regardless of the route of the administration. In this regard, our concerns were twofold, the risks of systemic absorption and the consequences of cuff damage with subsequent leakage of 4% lidocaine or saline

into the bronchial tree. Although 40 mg/mL lidocaine (4%) was used, the mean volume used per endotracheal tube was 6.1 mL ± 0.9 mL (SD) (244 mg ± 36 mg). This is considerably less than the amount of lidocaine used in a study by Sutherland and colleagues, [36] in which a fixed dose of 370 mg of lidocaine was used in 21 adult patients to topically anaesthetize the airway for fiberoptic bronchoscopy and no incidence of toxic plasma concentrations of lidocaine was recorded.

Another study by Efthimiou with 41 patients undergoing fiberoptic bronchoscopy, using average doses of 9.3 mg/kg of lidocaine, recorded only two patients in which plasma levels exceeded the toxic levels (5.0 µg/mL) and no complications were observed. All tube cuffs were intact post extubation.[37]

The basis of our study was that lidocaine inserted into the endotracheal cuff might cause anaesthesia of the trachea by diffusing across the polyvinyl chloride membrane of which the cuff is composed. Anaesthesia should be confined to the mucosa in contact with the cuff, thus overcoming the difficulties experienced by Gonzalez and others.[5] In addition, the protective cough reflexes above the tube cuff and of the vocal cords should remain intact.

Previously done studies by Carl Fagan and colleagues have compared the incidence of coughing and hemodynamic changes between intracuff lignocaine, intracuff saline and intracuff air. And concluded that incidence of coughing is significantly lower in intracuff lignocaine group.[10] In one study done by Soltani and colleagues compared the incidence of sore throat and coughing after general anaesthesia in six different groups which included spraying of the distal end of ETT cuff with 10% lignocaine, spraying of 10% to laryngopharyngeal structures, application of 2% lignocaine jelly to cuff of the tube, intravenous lignocaine at the end of surgery, Intracuff lignocaine and application of

normal saline to the cuff end of the tube. They concluded that I V lignocaine, intracuff lignocaine considerably decreases the incidence of coughing post extubation.[8]

Since lignocaine jelly is most commonly and widely used as a lubricant and as an agent to decrease the emergence phenomena. We compared intracuff lignocaine which can be easily administered without any special equipment needed. We monitored the cuff pressure changes with air used as an inflating agent when jelly was applied against intracuff lignocaine, which was not done in some of the previous studies. We also recorded the hemodynamic parameters in both the groups and studied the effect of the technique on the hemodynamic parameters.

There was no statistical difference between the 2 groups with regard to age, sex and weight. The mean duration of anaesthesia in the group J was 155.14±73.46 and in group I was 150.43±63.67 were comparable and an insignificant `p` value (0.6856). Some of the previous studies stated that the average duration required for diffusion of lignocaine to attain desirable concentration was 120 min.[24] This criteria was meeting in our study as the average duration of anaesthesia was 150 minutes.

Our data confirmed the increased cuff pressure and cuff volume after air inflation with N_2O and oxygen anaesthesia.[38,39] It has been reported that the over inflation occurring during general anaesthesia was attributable to an increase in temperature and, most importantly, because of more rapid NO_2 diffusion into the cuff than out from the cuff [40,8,39]. This over inflation of the ETT cuff has been associated with damage to the pharyngeal mucosa and recurrent laryngeal nerve palsy.[41] The lack of hyper pressure is probably one advantage of liquid filling of ETT cuffs.[42,43] In our study we monitored the

endotracheal cuff pressure keeping the endotracheal cuff pressure at 20 cmH_20 in both the groups after intubation. In Group J the mean cuff pressure at the end of surgery was 39.11±7.76 and showed a significant rise in cuff pressure. In Group I the mean cuff pressure at the end of surgery was19.96±0.77 showed no significant rise in cuff pressure. The groups were incomparable and had a significant `p` value (<0.001). Thus, confirming the data with previous studies.

The incidence of coughing and sore throat on emergence from general anesthesia in the presence of ETT has been estimated to range from 38% to 96%.[10,5] Selvaraj and Dhanpal found the incidence of postoperative cough and hoarseness to be higher in the lidocaine jelly group than in the **c**ontrol group.[44] Klemola UM studied the effect of laryngeal spray with lignocaine and lignocaine jelly application on 95 patients. The incidence of coughing when both the techniques used was 95%, when lignocaine jelly alone used was 85% and in the control group it was 62%. Thus, stating that the use of lignocaine jelly was associated with a high incidence of post extubation coughing and hoarseness.[45]

In our study the incidence of coughing was recorded in four different intervals. It was recorded as having occurred or not. The severity or grade of coughing was not recorded since categorization is very subjective. Twenty four patients (34.29%) had coughing immediately in lignocaine jelly group as compared to 9 patients (12.86%) in intracuff lignocaine group. The incidence of coughing at 1 minute was 41.43% in lignocaine jelly group and 12.86% in intracuff lignocaine group. At 3 minutes the incidence of coughing in lignocaine was 48.57% much higher than that noted at 1 minute in lignocaine jelly group as compared to 8.57% in lignocaine intracuff group. Thus,

showing a considerable decrease in the incidence of coughing. At 5 minutes the incidence of coughing came down to 27.14% in lignocaine jelly group and the incidence of coughing was 4.29% in lignocaine intracuff group. There was a significant statistical difference between the lignocaine jelly group and intracuff lignocaine group. The `p` value being 0.003, <0.001, <0.001, <0.001 is highly significant. These results were comparable to the previous studies done.

In study done by Estebe JP and others on 60 patients were intracuff alkalinized lignocaine was compared with intracuff saline and intracuff air. The results stated that there was a trend of reduced hypertension and tachycardia in the intracuff air group and alkalinized lignocaine group.[46] In another study done by the same author were alkalinized lignocaine was compared with gel lubricants concluded that there was no significant changes in the arterial blood pressure and heart rate.[47]

Hemodynamic parameters were measured immediately after extubation, at 1minute, 3 minute and at 5minutes in both the groups. The mean basal heart rates were 81.71±8.4, 81.24±9.08 for lignocaine jelly and lignocaine intracuff group. It was statistically insignificant with `p` value 0.7535. Following extubation the maximum increase was noted immediately after extubation and at 1 minute then it gradually decreased (table no 7). The comparisons between the two groups were significant and had significant `p` values.

The mean arterial pressures were also measured at the same intervals as the heart rate. The baseline mean arterial pressures were comparable. The peak values were obtained immediately after extubation and after one minute after extubation. These values

were incomparable and highly significant statistically. The pressures settled after 3minute tending to fall back to baseline values (table no 10). The statistical data was analyzed with unpaired student t test and test of proportions with `p` value less than 0.05 being significant, <0.01 being highly significant and <0.001 being very highly significant.

Our hemodynamic data was not comparable with the earlier studies done by Carl Fagan and colleagues and Estebe JP others which stated that there was no statistical difference in the hemodynamic parameters in there study.[10,46]

These results varied as the sample size was much larger 140 patients in our study as compared to 63 in their study. The extubation criteria were also different in both the studies which could have had an impact on the hemodynamic parameters.

LIMITATIONS OF THIS STUDY:

Measurement of plasma lignocaine levels was not done in our study. There was no practical way of assessing the amount of lignocaine that diffused across the cuff.

FUTURE SCOPE OF THE STUDY:

Intracuff lignocaine should be studied in ICU were patients are on mechanical ventilation were endotracheal tube tolerance is one of the biggest problem. Also with the use of intracuff lignocaine the cuff pressure can be kept constant over a period of time. Thus, avoiding repeated monitoring of the cuff pressure.

CONCLUSION

Lignocaine 4% intracuff helps in reducing post extubation coughing and also has decreased hemodynamic response to extubation when compared to lignocaine jelly which offers no advantage other than its lubricating effect.

In addition lignocaine intracuff maintains a stable cuff pressure during oxygen nitrous anaesthesia thus, preventing the damages that occur due to increased cuff pressure during general anaesthesia.

SUMMARY

The present study **"**A Randomized clinical study to compare the Emergence phenomena after General Anaesthesia with 2% Lignocaine jelly and 4% Lignocaine intracuff`" was carried out in the Department of Anaesthesiology , KLES Prabhakar Kore Hospital and MRC, Belgaum and Prabhakar Kore Free Hospital, Belgaum.

The study included 140 ASA grade 1 and 2 patients between the age group of 18 to 65 years divided into two groups consisting of 70 patients each after randomization by a computer generated randomization table .

Group J – Lignocine jelly group.

Group I – Intracuff lignocaine group.

4% lignocaine was used intracuff to inflate the cuff and keep the cuff pressure at 20 cm of H_2O. Emergence phenomena after general anaesthesia was compared with 2% lignocaine jelly applied over the cuff. Coughing and hemodynamic parameters were recorded post extubation for a period of 5min by an anaesthesiologist who was not present at the time of intubation.

Our study revealed that the incidence of coughing was much lower in group I as compared to group J. The study also revealed that there was a considerable decrease in the hemodynamic response in group I as compared to Group J and there were no complications like endotracheal tube cuff rupture and lignocaine toxicity.

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ANNEXURE - I : INFORMED CONSENT

YOUR PARTICIPATION

You Mr/Mrs/Ms. _____I.P. No. _____ are being asked to be a participant in the research study titled **"A RANDOMISED CLINICAL STUDY TO COMPARE THE EMERGENCE PHENOMENA AFTER GENERAL ANAESTHESIA WITH 2% LIGNOCAINE JELLY AND 4% LIGNOCAINE INTRACUFF"** A one year randomized controlled trial conducted by Dr. Nitish R Desai Postgraduate Student, Department of Anaesthesiology, JNMC, Belgaum. You are eligible after looking into inclusion criteria. You read this form and ask any questions you may have before agreeing to participate.

RESEARCH BEING DONE

TO COMPARE THE EMERGENCE PHENOMENA AFTER GENERAL ANAESTHESIA WITH 2% LIGNOCAINE JELLY AND 4% LIGNOCAINE INTRACUFF.

Purpose of the research

- To asses coughing post extubation with 2% lignocaine jelly and 4% lignocaine intracuff.
- To asses haemodynamic changes post extubation with 2% lignocaine jelly and 4% lignocaine intracuff.

Procedures involved

You will be randomly allocated either into group J or group I, if you are in group J then the endotracheal tube cuff would be inflated with air and application of 2% lignocaine jelly during intubation and if you are in group I then the endotrachael cuff would be inflated with 4% lignocaine intracuff without application of jelly. Post operative coughing and hamodynamic changes will be assessed at 0 min 1min 3 min and 5 min.

Potential risks and discomforts:

- No serious side effects.

Benefits of taking part in this research:

- Prevention of postoperative coughing and haemodynamic changes.
- Lesser incidence of sore throat.

Other option

Decline from participation

You have the option to decline from participation in the study without any discrimination and you will be treated as per the existing protocol for your condition.

New information

All information collected during the study from participant will be told as and when required.

Privacy and confidentiality

Privacy of individual will be respected and any information about you or provided by you during the study will be kept strictly confidential.

Injury as a result of participation

There will neither be any compensation to or for the patient and his or her relatives nor there any monetary benefits for the damage incurred.

Costs of participation in this research

Participation is free of cost.

Reimbursement for any expenses for participation in research

No reimbursement for any of your expenditures

Withdrawal or be removed

To start with as the participation was voluntary so is the decision to withdraw. Such a step will not alter the participants management by any staff in hospital. Researcher can remove you from the study if circumstances arise.

Whom to contact

For any information about the study during the study or after that may be collected from

- DR. V. D. PATIL. Principal, JNMC, Belgaum.
- DR. C.S.Sanikop, M.D DA Professor, Department of Anaesthesiology, JNMC, Belgaum. Ph.No.9448863688
- Dr. Nitish R Desai Postgraduate student in Anaesthesiology, JNMC, Belgaum. Ph. No. 9844765636

Signature of the participant or legally authorized person

Participants name:	Witness name
Signature:	Signature:
Date:	Place:

ANNEXURE – II : PROFORMA

Title **"A RANDOMISED CLINICAL STUDY TO COMPARE THE EMERGENCE PHENOMENA AFTER GENERAL ANAESTHESIA WITH 2% LIGNOCAINE JELLY AND 4% LIGNOCAINE INTRACUFF"**

Patients Name : I.P. No.:

Age : Wt: Sex:

Occupation : Date of operation:

Address :

Anaesthesiologist:

PRE-ANAESTHETIC EVALUATION:

Chief Complaints:

Past History:

a) HTN/D.M/Asthma/Epilepsy/Drug allergy.

b) Drug therapy.

c) Previous exposure to anaesthesia.

Family History:

General Physical Examination

Pallor / Icterus / Clubbing / Lymphadenopathy / Odema

P.R: B.P:

R.R:

Musculoskeletal System Examination:

Jaw movements: Teeth:

Airway assessment: Spine:

Systemic Examination:

a. R.S b. CNS

c.C.V.S d. GIT

Investigations:

Hb%

Urine routine

Any others

Pre operative physical status: ASA grade I II

Inclusion criteria

i. Patients of either sex aged between 18-65 years undergoing general surgery, orthopeadic, gynacecologyical, neurosurgical and urological surgeries under GA.

ii. American Society of Anaesthesiologist (ASA) Grade I and II patients.

iii. General anaesthesia lasting for more than one hour.

Exclusion criteria

i. ASA III/IV

ii Difficult airway :MP III/IV

iii H/S/O gastro intestinal reflux

iv Patients with increased intracranial pressure, active upper respiratory tract infection h/o laryngeal or tracheal surgery and h/o asthma.

v Patients who need nasogastric tube intraoperatively.

vi Patients who needed more than one attempt at intubation

Diagnosis

Proposed surgery

Patients will be allocated by computer generated randomization into group J and group I.

On the day of surgery, I.V line secured with 18g branula for males, 20g branula for females in a peripheral vein.

Preoperative baseline

Heart rate:

Blood pressure:

Monitors attached:

Pulse oximeter: Non invasive blood pressure:

ECG: $Etco_2$:

Study subjects will be premedicated with glycopyrrolate 0.005mg/kg body weight, Midazolam 0.05mg/kg body weight, fentanyl 1 microgram/kg body weight.

Patients preoxygenated with 100% oxygen for 3 min. Patients will be induced with thiopentone 5mg/kg body weight, vecuronium 0.1mg/kg body weight to facilitate tracheal intubation.Laryngoscope performed and trachea intubated with a standard cuffed endotracheal tube (portex).tube size of 7.5mm will be used in females and 8.5mm in males. For group (j) 2% lignocaine jelly 2.5 grams (contains approximately 50mg of lignocaine)will be applied ,Endotracheal tube inflated with a syringe, with air as inflating agent and the cuff pressure set at 20 cm of water with a rusch endotest cuff inflator.for group (I) the amount of air required to attain the set cuff pressure that is 20 cm of water measured and the same amount of 4 % lignocaine injected to inflate the cuff.

Anaesthesia maintained with nitrous: oxygen (35:65), vecuronium $1/4^{th}$ the intubating dose depending upon the etco2 changes, with the use of intermittent halothane 0.5 % for maintenance of anaesthesia. Lungs mechanically ventilated with tidal volume of 8-10ml/kg body weight. Pulse oximeter, Non invasive blood pressure, ECG and etco2 monitored.

At the end of surgery Nitrous oxide discontinued and 100%oxygen was given. Patient taken on mapleson D circuit reversed with glycopyrrolate 0.005mg/kg body weight and neostigmine 0.05mg/kg body weight.

Cuff pressure measured just before deflating the cuff and recorded, Extubation will be performed after checking for spontaneous ventilation, ability to follow verbal commands(eye opening, tongue protrusion)

Immediately after extubation patient will be assessed for coughing, heart rate, blood pressure and latter at 0min, 1min, 3min and 5min.

Coughing will be recorded as either occurred or not occurred by another anaesthesiologist who is not present at the time of intubation(Blinding).

Duration of surgery:

Cuff pressure at the beginning:

Cuff pressure just before extubation:

Time interval	**0min**	**1min**	**3min**	**5min**
Coughing yes/no				

Time interval in minutes	**Heart rate**	**Blood pressure**		
		systolic	**diastolic**	**mean**
Base line				
0min				
1min				
3min				
5min				

Side effects : Signature of the Staff Incharge :

MASTER CHART : GROUP J

S.NO	GROUP	DATE	I.P.NO	AGE	SEX	WEIGHT	ASA	D O A	Cf-Pr 0	Cf-Pr 1	C- 0	C-1	C-3	C-5	HR- BL	H- 0	H -1	H- 3	H- 5	BP-S-BL	BP-S-0	BP-S-1	BP-S-3	BP-S-5	BP-D-BL	BP-D-0	BP-D-1	BP-D-3	BP-D-5	M-BL	M-0	M-1	M-3	M-5	SIDE EFF.
1	J	3/1/2008	271291	65	F	60	II	185	20	44	Y	N	N	N	74	103	96	86	79	132	156	144	138	132	88	104	90	96	92	102	121	108	110	105	NILL
2	J	7/1/2008	257401	40	F	54	I	90	20	40	N	N	N	N	79	111	103	99	87	110	130	124	120	120	70	92	90	90	84	83	104	101	100	96	NILL
3	J	7/1/2008	257548	45	M	55	II	75	20	35	N	N	Y	Y	92	113	111	104	90	118	138	130	132	132	76	110	105	100	96	90	119	113	110	108	NILL
4	J	9/1/2008	257392	62	F	70	II	210	20	30	Y	Y	N	N	80	109	111	106	88	110	180	175	170	160	70	100	94	90	84	83	126	119	116	109	NILL
5	J	19/1/2008	258011	26	F	55	II	150	20	45	N	N	N	N	98	119	126	114	106	128	144	130	130	134	84	106	100	96	94	98	118	110	107	107	NILL
6	J	19/1/2008	257977	48	F	60	II	135	20	50	N	N	N	N	120	98	95	90	86	122	160	158	154	149	78	98	96	92	88	92	118	116	112	108	NILL
7	J	21/1/2008	258256	22	M	56	I	165	20	40	N	N	N	N	64	86	94	92	85	130	142	136	130	132	96	105	94	90	90	107	117	108	103	104	NILL
8	J	21/1/2008	271376	42	F	56	II	95	20	34	N	N	N	N	82	85	81	77	72	128	140	132	132	124	78	86	86	80	80	94	104	101	97	94	NILL
9	J	5/3/2008	264412	64	M	82	II	205	20	45	Y	N	Y	N	80	113	107	102	87	140	160	152	146	146	90	110	96	90	90	106	126	114	108	102	NILL
10	J	11/3/2008	264058	60	M	73	II	450	20	62	Y	Y	Y	Y	68	99	104	96	93	124	156	150	146	144	78	100	98	96	96	93	118	102	112	112	NILL
11	J	11/3/2008	264067	30	F	51	I	75	20	35	N	N	N	N	85	73	86	91	88	125	137	132	124	129	91	100	95	86	87	105	115	110	101	103	NILL
12	J	14/3/2008	264575	25	M	60	I	70	20	28	N	N	N	N	72	93	94	86	81	110	133	133	128	120	72	88	86	85	82	84	103	101	99	94	NILL
13	J	14/3/2008	264304	42	F	61	I	310	20	60	N	N	Y	N	64	73	71	68	76	122	118	105	106	118	62	57	61	58	71	83	78	70	75	86	NILL
14	J	14/3/2008	276466	19	F	43	II	120	20	36	Y	Y	Y	Y	92	102	96	90	88	110	154	145	140	126	70	106	100	92	89	83	122	115	108	101	NILL
15	J	18/3/2008	275139	25	M	56	II	190	20	47	Y	Y	Y	Y	69	99	106	96	93	124	156	150	146	144	78	100	98	96	96	93	118	115	112	112	NILL
16	J	18/3/2008	277926	40	M	70	II	165	20	32	Y	N	Y	Y	80	113	107	102	87	140	160	152	146	143	90	110	96	90	90	106	126	114	108	107	NILL
17	J	18/3/2008	277982	36	F	57	II	135	20	35	Y	Y	Y	Y	84	93	94	81	76	132	154	150	138	132	88	103	100	90	92	102	120	116	106	105	NILL
18	J	22/3/2008	276260	22	M	51	II	130	20	33	N	N	N	N	83	99	97	88	81	126	144	130	130	122	82	86	80	80	82	96	105	96	96	95	NILL
19	J	25/3/2008	281106	50	F	61	II	150	20	35	Y	Y	Y	N	90	103	102	88	75	130	155	150	142	124	86	95	90	90	86	100	115	110	107	98	NILL
20	J	27/3/2008	269387	55	M	60	II	335	20	50	N	N	Y	N	84	98	103	96	92	130	156	150	146	144	84	100	96	96	90	99	118	114	112	108	NILL
21	J	3/5/2008	265530	35	M	57	II	345	20	38	N	N	N	N	86	105	106	101	96	146	158	150	140	140	96	104	100	96	90	112	122	116	110	106	NILL
22	J	7/5/2008	270207	42	M	58	II	180	20	36	N	N	N	N	74	98	96	86	78	132	156	144	138	132	86	104	98	92	92	101	121	113	107	105	NILL
23	J	7/5/2008	271810	50	F	56	II	145	20	42	Y	Y	Y	N	74	103	94	87	78	132	154	150	138	132	88	104	100	90	92	102	120	116	106	108	NILL
24	J	7/5/2008	271997	20	F	46	I	255	20	46	N	N	N	N	84	100	96	88	80	106	114	114	110	110	72	84	80	78	80	83	94	91	88	90	NILL
25	J	11/5/2008	271886	38	F	46	II	165	20	42	N	N	Y	N	78	106	106	89	83	138	154	145	140	140	90	104	100	92	88	106	120	115	108	105	NILL
26	J	15/5/2008	281122	34	M	60	II	105	20	33	Y	Y	N	N	84	114	108	96	84	134	157	145	143	135	80	99	95	90	87	98	118	111	107	103	NILL
27	J	9/6/2008	272884	55	F	38	II	100	20	45	Y	Y	Y	N	79	113	102	98	91	110	130	124	120	120	70	92	90	90	84	83	104	101	100	96	NILL
28	J	10/6/2008	279825	65	F	51	II	240	20	42	Y	Y	Y	Y	74	98	96	93	87	134	159	150	138	135	80	105	97	90	92	98	123	114	106	106	NILL
29	J	11/6/2008	275215	55	M	65	II	190	20	46	Y	Y	Y	N	89	118	124	116	102	128	144	136	130	134	84	106	100	96	94	98	118	112	107	107	NILL
30	J	15/6/2008	276175	65	M	61	II	125	20	33	N	N	N	N	85	76	90	97	92	125	137	132	124	129	91	100	95	86	87	105	115	110	101	103	NILL
31	J	20/6/2008	277339	35	M	58	II	105	20	32	N	N	N	N	81	103	105	103	90	132	156	144	138	132	85	105	98	92	92	100	122	113	107	105	NILL
32	J	20/6/2008	276871	35	M	61	II	75	20	28	Y	Y	Y	N	84	101	106	97	91	130	156	150	146	144	84	100	96	96	90	99	118	114	112	108	NILL
33	J	21/6/2008	277543	22	F	42	II	200	20	54	N	N	N	N	87	108	96	94	87	140	156	150	143	136	96	107	100	96	96	110	123	116	111	109	NILL
34	J	28/6/2008	275897	60	F	43	II	220	20	44	N	N	N	N	72	98	99	90	86	110	133	133	128	125	72	88	86	85	80	84	103	101	99	95	NILL
35	J	4/7/2008	278866	52	M	58	II	75	20	28	Y	N	Y	N	72	88	85	78	76	124	136	135	128	124	70	82	80	84	76	88	100	98	98	92	NILL
36	J	4/7/2008	278848	52	M	54	I	120	20	30	N	N	Y	N	72	103	99	92	80	120	145	141	135	127	74	89	85	82	80	89	107	103	99	95	NILL
37	J	10/7/2008	280229	25	M	60	II	150	20	42	Y	Y	Y	Y	90	119	116	111	95	117	146	143	135	119	77	97	90	85	80	90	113	107	101	93	NILL

MASTER CHART : GROUP J

2	J	7/1/2008	257401	40	F	54	I	90	20	40	N	N	N	N	79	111	103	99	87	110	130	124	120	120	70	92	90	90	84	83	104	101	100	96	NILL
38	J	11/7/2008	278832	45	F	53	II	120	20	29	Y	Y	Y	Y	82	110	108	103	98	136	154	150	141	135	84	95	93	90	83	101	114	112	107	100	NILL
39	J	11/7/2008	280060	22	M	61	I	210	20	35	N	N	N	N	90	128	121	106	96	140	167	161	155	143	80	103	99	96	90	100	124	119	115	107	NILL
40	J	17/7/2008	283017	45	M	57	II	180	20	47	N	N	N	N	90	100	99	93	86	130	150	144	135	130	82	96	87	85	80	98	114	106	101	96	NILL
41	J	21/7/2008	279183	40	F	51	II	255	20	32	N	N	N	N	86	112	108	104	96	140	169	165	149	136	86	99	90	90	84	104	122	115	109	101	NILL
42	J	23/7/2008	280392	60	M	61	II	150	20	34	N	Y	Y	Y	90	140	138	131	117	145	169	165	147	137	96	107	105	101	93	112	127	125	116	107	NILL
43	J	24/7/2008	274130	27	M	60	I	60	20	26	N	N	N	N	88	107	97	88	72	135	147	140	135	128	86	94	90	87	82	102	111	106	103	97	NILL
44	J	25/7/2008	279307	58	M	70	II	60	20	32	N	N	Y	Y	77	89	94	86	78	129	144	140	132	132	84	95	93	88	85	99	111	108	102	100	NILL
45	J	25/7/2008	280609	29	M	60	II	165	20	42	N	Y	Y	Y	88	103	104	96	91	132	149	143	137	130	84	97	95	92	87	100	114	111	107	101	NILL
46	J	2/9/2008	286843	38	F	49	I	180	20	36	N	N	N	N	86	103	110	106	100	146	158	150	140	140	96	104	100	96	90	112	122	116	110	106	NILL
47	J	2/9/2008	286871	52	M	60	II	160	20	30	N	Y	Y	N	84	103	108	100	96	130	156	150	146	144	84	100	96	96	90	99	118	114	112	108	NILL
48	J	2/9/2008	285394	55	M	64	II	120	20	40	N	N	N	N	79	109	96	88	80	132	156	144	138	132	86	104	98	92	92	101	121	113	107	105	NILL
49	J	4/9/2008	286958	35	F	44	II	120	20	35	N	Y	Y	N	74	107	100	90	82	132	154	150	138	132	88	104	100	90	92	102	120	116	106	105	NILL
50	J	5/9/2008	286991	65	M	60	II	120	20	34	N	N	Y	Y	84	105	102	92	84	106	114	114	110	110	72	84	80	78	80	83	94	91	88	90	NILL
51	J	5/9/2008	280402	38	F	49	II	120	20	38	N	N	N	N	78	111	108	92	86	138	154	145	140	140	90	104	100	92	88	106	120	115	108	105	NILL
52	J	9/9/2008	287285	54	F	60	I	150	20	36	N	N	N	N	72	98	99	90	86	110	133	133	128	125	72	88	86	85	80	84	103	101	99	95	NILL
53	J	9/9/2008	287606	20	F	46	I	70	20	25	N	N	Y	Y	87	118	98	96	90	140	156	150	143	136	96	107	100	96	96	110	123	116	111	109	NILL
54	J	11/9/2008	257548	50	F	52	II	150	20	39	N	Y	N	Y	84	103	108	100	96	130	156	150	146	144	84	100	96	96	90	99	118	114	112	108	NILL
55	J	12/9/2008	288243	45	F	55	II	90	20	38	N	Y	N	Y	81	113	118	105	92	132	156	144	138	132	85	105	98	92	92	100	122	113	107	105	NILL
56	J	13/9/2008	288414	52	M	63	II	135	20	34	N	Y	N	Y	81	113	118	105	92	132	156	144	138	132	85	105	98	92	92	100	122	113	107	105	NILL
57	J	13/9/2008	288112	36	M	70	I	195	20	42	N	Y	N	Y	85	76	90	97	92	125	137	132	124	129	91	100	95	86	87	105	115	110	101	103	NILL
58	J	13/9/2008	288417	45	F	53	II	150	20	42	N	Y	Y	N	89	124	130	120	106	128	144	136	130	134	84	106	100	96	94	98	118	112	107	107	NILL
59	J	13/9/2008	289999	47	M	22	II	130	20	48	N	Y	Y	N	74	123	91	81	72	132	160	144	138	129	86	104	98	92	84	101	122	113	107	99	NILL
60	J	13/9/2008	288444	45	F	55	II	150	20	43	Y	N	Y	N	90	122	121	115	98	117	146	143	135	119	77	97	90	85	80	90	113	107	101	93	NILL
61	J	16/9/2008	288798	39	M	63	II	90	20	35	N	N	N	N	72	88	85	78	66	124	136	135	128	124	70	82	80	84	76	88	100	98	98	92	NILL
62	J	18/9/2008	289132	27	F	49	I	60	20	34	Y	N	Y	N	77	89	94	86	78	129	144	140	132	132	84	95	93	88	85	99	111	108	102	100	NILL
63	J	18/9/2008	289530	30	M	61	I	100	20	43	N	N	N	N	86	96	93	89	80	122	140	132	127	125	84	92	90	87	83	96	108	104	100	97	NILL
64	J	19/9/2008	288896	25	F	49	II	240	20	52	Y	Y	Y	N	90	89	94	90	84	110	128	128	120	114	76	84	80	82	80	87	98	96	94	91	NILL
65	J	20/9/2008	289412	50	F	52	II	105	20	38	N	Y	N	N	79	117	107	105	94	110	130	124	120	120	70	92	90	90	84	83	104	101	100	96	NILL
66	J	22/9/2008	288993	20	F	45	I	270	20	52	Y	Y	N	N	90	105	99	93	81	132	164	149	132	124	78	105	99	85	85	96	124	115	100	98	NILL
67	J	24/9/2008	289999	22	M	47	I	130	20	48	Y	Y	Y	N	74	152	120	90	82	132	160	144	138	129	86	104	98	92	84	101	122	113	107	99	NILL
68	J	25/9/2008	288928	60	F	45	II	210	20	46	N	N	N	Y	84	95	102	96	82	130	156	150	146	140	84	100	96	90	82	99	118	114	108	101	NILL
69	J	26/9/2008	290062	32	F	59	II	120	20	34	N	N	N	N	72	103	99	92	80	120	145	141	135	127	74	89	85	82	80	89	107	103	99	95	NILL
70	J	27/9/2008	290230	30	F	50	I	60	20	42	Y	Y	Y	N	72	88	85	78	76	124	136	135	124	124	70	82	80	84	76	88	100	98	97	92	NILL

MASTER CHART : GROUP I

S.NO	GROUP	DATE	I.P.NO	AGE	SEX	WEIGHT	ASA	DOA	Cf-Pr 0	Cf-Pr 1	C- 0	C-1	C-3	C-5	HR- BL	H-0	H-1	H-3	H-5	BP-S-BL	BP-S-0	BP-S-1	BP-S-3	BP-S-5	BP-D-BL	BP-D-0	BP-D-1	BP-D-3	BP-D-5	M-BL	M-0	M-1	M-3	M-5	SIDE EFF.
1	I	8/1/2008	269468	52	M	62	II	130	20	20	N	N	Y	N	90	96	98	94	88	110	128	128	120	114	76	84	80	80	80	87	98	96	93	91	NILL
2	I	9/1/2008	258000	40	M	80	II	165	20	20	N	N	N	N	82	95	92	90	86	144	164	156	150	142	94	114	105	103	96	109	130	122	118	116	NILL
3	I	9/1/2008	277356	25	M	53	I	155	20	20	N	N	N	N	86	90	92	92	82	132	144	140	126	125	83	90	92	86	83	99	108	108	99	97	NILL
4	I	9/1/2008	277926	40	M	60	II	110	20	20	N	N	N	N	91	84	83	81	83	130	136	136	130	126	80	84	80	78	78	96	101	98	95	94	NILL
5	I	10/1/2008	258030	18	M	45	I	150	20	20	N	N	N	N	62	96	92	90	90	130	150	140	140	136	70	90	90	90	84	90	110	106	106	101	NILL
6	I	10/1/2008	258167	30	F	48	II	90	20	16	N	N	N	N	82	96	94	86	84	110	130	124	124	122	70	84	76	72	70	83	99	92	89	87	NILL
7	I	12/1/2008	257022	37	M	52	I	120	20	19	N	N	N	N	90	94	88	86	86	130	136	136	132	130	76	82	82	80	80	94	100	100	97	96	NILL
8	I	26/1/2008	269933	65	M	68	II	160	20	20	N	N	N	N	78	100	96	88	82	130	154	150	145	134	82	96	96	92	90	98	115	114	109	104	NILL
9	I	1/3/2008	264056	28	M	52	I	210	20	20	N	N	N	N	100	104	102	100	96	120	124	124	122	120	66	75	72	70	70	84	91	89	87	86	NILL
10	I	1/3/2008	263984	57	M	64	II	135	20	20	N	N	N	N	68	80	140	110	90	123	110	108	106	110	77	70	70	68	72	92	83	82	80	84	NILL
11	I	4/3/2008	264399	45	F	54	II	135	20	20	Y	Y	N	N	96	110	112	106	106	140	156	150	146	144	94	100	98	96	96	109	118	115	112	112	NILL
12	I	14/3/2008	267832	65	F	52	II	150	20	20	N	N	N	N	73	80	89	86	88	133	110	128	124	128	86	70	78	72	74	101	83	94	89	92	NILL
13	I	17/3/2008	257147	62	M	74	II	135	20	20	N	N	N	N	111	96	97	98	109	105	127	144	143	145	70	83	85	91	84	85	103	106	110	105	NILL
14	I	17/3/2008	266570	56	M	63	II	195	20	20	N	N	N	N	82	96	90	88	88	132	140	140	136	130	86	94	90	88	84	101	109	106	104	99	NILL
15	I	19/3/2008	265937	58	M	56	II	210	20	20	Y	N	N	N	82	90	86	80	78	154	160	154	151	140	96	100	98	90	88	115	120	116	110	105	NILL
16	I	21/3/2008	266366	39	F	60	II	105	20	20	N	N	N	N	70	86	94	90	86	132	145	140	138	126	90	98	96	96	84	104	113	111	110	98	NILL
17	I	23/3/2008	266664	45	M	52	II	90	20	20	N	N	N	N	70	74	78	74	72	116	122	122	130	124	80	86	84	80	76	92	98	96	96	92	NILL
18	I	15/4/2008	265608	23	F	54	I	90	20	20	N	N	N	N	102	115	112	107	109	136	145	134	136	134	90	96	93	92	106	110	119	113	111	116	NILL
19	I	20/4/2008	277720	60	F	68	II	125	20	20	N	N	N	N	74	93	90	85	79	130	142	144	132	130	80	96	90	84	84	96	111	108	100	99	NILL
20	I	23/4/2008	277980	25	F	64	II	60	20	20	N	N	N	Y	86	125	117	120	108	128	132	130	126	130	76	89	84	78	80	93	103	99	94	96	NILL
21	I	3/5/2008	282030	40	M	53	II	75	20	20	N	N	N	N	82	95	97	90	76	130	143	140	126	123	82	87	85	85	80	98	105	103	98	94	NILL
22	I	14/5/2008	271787	32	F	62	II	70	20	20	N	N	N	N	78	86	84	76	76	124	132	132	130	128	86	90	86	84	84	98	104	101	99	98	NILL
23	I	17/5/2008	265339	55	M	70	II	195	20	20	Y	Y	N	N	84	96	94	90	82	128	132	130	126	130	76	80	80	78	80	93	97	96	94	96	NILL
24	I	17/5/2008	271580	48	F	44	II	240	20	20	N	N	N	N	78	100	96	88	80	130	142	144	132	130	80	96	90	84	84	96	111	108	100	99	NILL
25	I	18/5/2008	269948	39	F	54	II	165	20	20	N	N	N	N	86	96	100	90	82	130	144	140	126	126	80	92	90	90	88	96	109	106	102	100	NILL
26	I	19/5/2008	265942	65	F	46	II	195	20	20	N	N	N	N	84	96	90	82	76	130	136	136	130	126	80	84	80	78	78	96	101	98	95	94	NILL
27	I	20/5/2008	280778	50	F	44	II	180	20	20	N	N	N	N	84	99	97	89	80	140	156	149	136	126	90	95	89	85	80	106	114	109	102	95	NILL
28	I	24/5/2008	281362	49	M	64	II	120	20	20	N	N	N	Y	80	95	89	84	70	134	145	140	132	129	80	87	85	80	84	98	106	103	97	99	NILL
29	I	10/6/2008	275139	25	M	53	II	170	20	20	Y	Y	Y	N	68	80	140	110	90	123	110	108	106	110	77	70	70	68	72	92	83	82	79	88	NILL
30	I	12/6/2008	289131	33	F	40	II	120	20	20	N	N	N	N	96	110	101	95	89	114	134	130	129	127	78	90	86	83	80	90	104	100	98	95	NILL
31	I	19/6/2008	276846	45	F	45	II	70	20	22	N	N	N	N	78	115	112	107	100	136	145	134	136	134	90	96	93	92	90	105	112	106	106	104	NILL
32	I	20/6/2008	281527	54	F	59	II	150	20	20	N	N	N	N	70	90	85	80	74	130	145	140	135	128	86	93	90	87	84	100	110	106	105	98	NILL
33	I	20/6/2008	276783	56	M	70	II	90	20	19	Y	Y	Y	N	78	86	84	76	76	124	132	132	130	128	86	90	86	84	84	98	104	101	99	98	NILL
34	I	20/6/2008	275536	19	F	42	I	85	20	20	N	N	N	N	78	86	84	76	76	124	132	132	130	120	86	90	85	84	78	98	104	100	99	92	NILL
35	I	20/6/2008	282553	65	F	44	II	150	20	20	N	N	N	N	80	96	93	86	74	130	144	140	137	130	85	95	89	85	80	100	111	106	102	96	NILL

MASTER CHART : GROUP I

S.NO	GROUP	DATE	I.P.NO	AGE	SEX	WEIGHT	ASA	DOA	Cf-Pr 0	Cf-Pr 1	C- 0	C-1	C-3	C-5	HR- BL	H-0	H-1	H-3	H-5	BP-S-BL	BP-S-0	BP-S-1	BP-S-3	BP-S-5	BP-D-BL	BP-D-0	BP-D-1	BP-D-3	BP-D-5	M-BL	M-0	M-1	M-3	M-5	SIDE EFF.
36	I	10/7/2008	280217	50	F	57	II	145	20	20	N	N	N	N	90	96	96	92	86	146	154	136	136	132	90	95	87	90	80	108	114	103	105	97	NILL
37	I	12/7/2008	279615	30	M	62	II	170	20	20	N	N	N	N	86	98	95	88	78	132	144	135	130	124	84	90	87	82	82	100	108	103	98	96	NILL
38	I	12/7/2008	279151	21	M	61	I	165	20	20	N	N	N	N	84	96	95	88	76	130	144	135	130	129	80	90	87	80	80	96	108	103	96	96	NILL
39	I	16/7/2008	280042	25	M	56	I	190	20	20	N	N	N	N	86	96	95	90	80	132	147	135	129	127	84	90	87	83	80	100	109	103	98	95	NILL
40	I	16/7/2008	280097	31	M	63	I	125	20	20	N	Y	Y	N	90	109	105	100	94	135	145	145	141	133	96	105	103	96	90	109	118	117	111	104	NILL
41	I	18/7/2008	279100	28	F	46	II	165	20	20	N	N	N	N	96	109	105	98	90	134	145	143	135	130	86	97	95	92	87	102	113	111	106	101	NILL
42	I	18/7/2008	282713	25	F	53	II	70	20	20	N	N	N	N	76	88	86	79	72	108	109	113	108	99	70	77	82	79	73	76	89	91	88	81	NILL
43	I	26/7/2008	279836	38	F	49	II	150	20	20	N	N	N	N	86	103	97	93	84	122	140	132	127	125	84	92	90	81	83	96	108	104	100	97	NILL
44	I	26/7/2008	281523	35	F	50	II	90	20	20	N	N	Y	N	88	112	105	94	79	130	152	146	132	122	84	96	90	80	77	99	114	108	97	92	NILL
45	I	27/7/2008	281126	65	M	72	II	90	20	20	N	N	N	N	94	117	100	94	86	136	150	149	135	128	84	95	92	87	87	101	113	111	103	100	NILL
46	I	1/9/2008	286787	42	M	58	II	450	20	20	N	N	N	N	78	86	84	76	76	124	132	132	130	128	86	90	86	84	84	98	104	101	99	98	NILL
47	I	1/9/2008	286614	33	F	60	II	180	20	20	N	N	N	N	78	86	84	76	76	121	132	132	130	120	86	90	85	84	78	98	104	100	99	92	NILL
48	I	8/9/2008	278280	44	F	49	II	120	20	20	Y	Y	Y	N	78	86	84	76	76	124	132	132	130	128	86	90	86	84	84	98	104	97	99	98	NILL
49	I	9/9/2008	287544	31	F	42	II	60	20	20	Y	Y	N	Y	78	115	112	107	100	136	145	134	136	130	90	96	93	92	90	105	112	106	106	103	NILL
50	I	10/9/2008	287826	22	F	52	I	90	20	22	N	N	N	N	68	80	140	110	90	123	110	108	106	110	77	70	70	68	72	92	83	82	80	84	NILL
51	I	10/9/2008	287775	36	F	49	II	140	20	20	N	N	N	N	84	96	90	82	76	130	136	136	130	126	80	84	80	78	78	96	101	98	95	94	NILL
52	I	10/9/2008	286748	25	M	64	II	80	20	20	Y	Y	N	N	84	96	94	90	82	128	132	130	126	130	76	80	80	78	80	93	97	96	94	96	NILL
53	I	11/9/2008	257651	43	F	64	I	165	20	20	N	N	N	N	72	105	103	94	90	110	133	133	128	125	72	88	86	85	80	84	103	101	99	95	NILL
54	I	12/9/2008	288100	65	M	74	II	240	20	20	N	N	N	N	84	96	94	90	82	128	132	130	126	130	76	80	80	78	80	93	97	96	92	96	NILL
55	I	13/9/2008	288120	38	F	42	II	180	20	20	N	N	N	N	78	100	96	88	80	130	142	144	132	130	80	96	90	84	84	96	111	108	100	99	NILL
56	I	13/9/2008	281082	49	F	56	II	120	20	20	N	N	N	N	71	110	107	103	90	133	150	145	133	126	92	97	90	85	80	105	114	108	101	95	NILL
57	I	16/9/2008	288450	25	M	60	II	210	20	20	N	N	N	N	86	98	95	88	78	132	144	135	130	124	84	90	87	82	82	100	108	103	98	96	NILL
58	I	16/9/2008	288853	40	F	52	II	240	20	20	N	N	N	N	72	110	103	96	84	120	145	141	135	121	74	89	85	82	80	89	107	103	99	95	NILL
59	I	17/9/2008	288453	33	M	65	II	225	20	19	N	N	N	N	80	90	96	92	86	146	154	136	136	132	90	95	87	90	80	108	114	103	105	97	NILL
60	I	18/9/2008	289431	40	M	59	II	210	20	21	N	N	N	N	76	86	84	76	76	124	132	132	130	128	86	90	86	84	84	98	104	101	99	98	NILL
61	I	19/9/2008	288894	40	M	65	II	150	20	20	N	N	N	N	78	86	84	76	76	124	132	132	130	12	86	90	86	84	84	98	104	101	99	98	NILL
62	I	20/9/2008	289422	36	F	45	II	190	20	22	N	N	N	N	82	95	92	90	86	144	165	156	150	142	94	114	105	103	96	110	131	122	118	111	NILL
63	I	20/9/2008	288879	65	F	60	II	150	20	20	N	N	N	N	86	90	92	92	82	132	144	140	126	125	83	90	92	86	83	99	108	108	99	97	NILL
64	I	22/9/2008	289924	40	F	52	II	120	20	18	N	N	N	N	70	86	90	80	76	129	140	152	138	127	84	96	99	95	89	99	110	116	109	101	NILL
65	I	24/9/2008	289999	22	M	60	II	270	20	21	N	N	N	N	68	92	90	85	76	110	134	123	120	117	75	90	85	85	82	86	104	97	103	93	NILL
66	I	24/9/2008	299904	30	M	65	II	120	20	20	N	N	N	N	70	84	86	80	68	112	132	130	124	122	70	89	87	90	82	84	103	101	101	95	NILL
67	I	25/9/2008	289456	43	F	50	II	70	20	20	N	N	N	N	80	94	102	90	81	130	142	140	126	120	82	96	95	80	76	98	111	110	95	90	NILL
68	I	26/9/2008	289706	22	F	50	I	295	20	20	N	N	N	N	88	112	105	94	79	130	152	146	132	122	84	96	90	80	77	99	114	108	97	92	NILL
69	I	27/9/2008	290231	24	M	52	II	120	20	20	Y	Y	N	N	72	95	89	82	80	124	136	135	124	124	70	82	80	84	76	88	100	98	97	92	NILL
70	I	27/9/2008	289182	52	M	60	II	180	20	18	N	N	N	N	71	110	107	103	90	133	150	145	133	126	92	97	90	85	80	105	114	108	101	95	NILL

KEY TO MASTER CHART.

ASA = American Society of Anesthesiologist.

BP-D-0 = Blood pressure diastolic immediately after extubation.

BP-D-1 = Blood pressure diastolic at 1 minute after extubation.

BP-D-3 = Blood pressure diastolic at 3 minute after extubation.

BP-D-5 = Blood pressure diastolic at 5 minute after extubation.

BP-D-BL = Baseline blood pressure diastolic.

BP-S-0 = Blood pressure systolic immediately after extubation.

BP-S-1 = Blood pressure systolic at 1 minute after extubation.

BP-S-3 = Blood pressure systolic at 3 minute after extubation.

BP-S-5 = Blood pressure systolic at 5 minute after extubation.

BP-S-BL = Baseline blood pressure systolic.

C-0 = Coughing immediately after extubation.

C-1 = Coughing at 1 minute after extubation.

C-3 = Coughing at 3 minute after extubation.

C-5 = Coughing at 5 minute after extubation.

Cf-Pr-0 = Cuff pressure at start of surgery.

Cf-Pr-1 = Cuff pressure at the end of surgery.

D O A = Duration of anaesthesia.

F = Female.

Group I = Lignocaine intracuff.

Group J = Lignocaine jelly.

H-0 = Heart rate immediately after extubation.

H-1 = Heart rate at 1 minute after extubation.

H-3 = Heart rate at 3 minute after extubation.

H-5 = Heart rate at 5 minute after extubation.

HR-BL = Heart rate baseline.

M = Male.

M-0 = Mean blood pressure immediately after extubation.

M-1 = Mean blood pressure at 1 minute after extubation.

M-3 = Mean blood pressure at 3 minute after extubation.

M-5 = Mean blood pressure at 5 minute after extubation.

M-BL = Baseline Mean blood pressure.