IMPACT OF INTRAVENOUS LIGNOCAINE ON INTRAOPERATIVE HAEMODYNAMICS, RECOVERY CHARACTERISTICS, POST-OP PAIN SCORES, POST OP ANALGESIC REQUIREMENT & RETURN OF BOWEL FUNCTION IN PATIENTS UNDERGOING LAPROSCOPIC CHOLECYSTECTOMY

Dissertation Submitted for the Degree of

DOCTOR OF MEDICINE

BRANCH – X (ANAESTHESIOLOGY)

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THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI
TAMILNADU

BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled **"IMPACT OF INTRAVENOUS LIGNOCAINE ON INTRAOPERATIVE HAEMODYNAMICS, RECOVERY CHARACTERISTICS, POST-OP PAIN SCORES, POST-OP ANALGESIC REQUIREMENT & RETURN OF BOWEL FUNCTION IN PATIENTS UNDERGOING LAPROSCOPIC CHOLECYSTECTOMY"** is a Bonafide Record Work done by **Dr. RAVI. P** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of University regulation for M.D, Branch X – Anaesthesiology

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CERTIFICATE FROM THE GUIDE

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DECLARATION

I, **Dr. P. RAVI** solemnly declare that, this Dissertation titled **"IMPACT OF INTRAVENOUS LIGNOCAINE ON INTRAOPERATIVE HAEMODYNAMICS, RECOVERY CHARACTERISTICS, POST-OP PAIN SCORES, POST-OP ANALGESIC REQUIREMENT & RETURN OF BOWEL FUNCTION IN PATIENTS UNDERGOING LAPROSCOPIC CHOLECYSTECTOMY"** was done by me.

I also declare that this Bonafide Work / a part of this work was not submitted by me / anyone else, for any Award, Degree / Diploma to any other University / Board either in India / abroad. This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of **MD Anaesthesiology,** to be held in April 2015.

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INTRODUCTION

Recently there is an increase in concerns about ambulatory surgery among surgeons as it facilitates early hospital discharge, less health care related and improving post-operative quality of life. However this has cause new challenges to the attending anesthesiologists as anesthesia management for ambulatory surgery needs careful titration of anesthetic agents targeting at early post-operative recovery, stable hemodynamics in intra operative and post-operative period and adequate pain relief without any sedation in post-operative period.

In common with all other types of pain, acute postoperative pain is an extraordinarily complex sensation which may be described as an interpretation of these signals by higher centers involving memory experiences of painful situation, and an affective component which generally comprises anxiety and /or depression.

Uncontrolled post-operative pain has an adverse sequel of delayed resumption of normal pulmonary function, restriction of mobility (thus contributing to thromboembolic complications), nausea, vomiting and

cardiac work load was increased and myocardial oxygen consumption also increased due to catecholamine release induced by the stress response.

Post-operative pain control is an essential component of Anesthesia management. Adequate pain control is one of the crucial factors in ambulatory surgical plan. Management Plan should be aimed at providing adequate pain relief and at the same time minimizing side effects like sedation. So that the patient can be safely discharged from surgical facility without any major influence on the patient's ability to resume their normal activities of daily living**.** Control of acute post-operative pain and the timing, and duration (e.g., Pre-emptive Analgesia), is important in facilitating short and long-term patient convalescence.

For many years, Opioid based perioperative Analgesia Management was being used. Although potent analgesics opiods are associated with multiple perioperative side effects including respiratory depression, sedation and drowsiness, urinary retention, post-operative nausea and vomiting, pruritus and decrease in bowel movements leading to Ileus and constipation. These side effects may lead to prolonged post-operative stay, delay hospital discharge and increase in hospitalization costs.

In addition, the use of conventional method of administration of intra-muscular opioids in standard prescribed doses, may be too large (causing side effects), or too small (causing inadequate analgesia). Therefore, Anesthesiologist are increasingly concerned towards non-conventional techniques as adjuvant for managing post-operative pain to reduce the adverse effects of opioid based Analgesic drugs.

From the non-conventional methods, the infiltration of long-acting local Anaesthetics as an adjuvant for regional or local anaesthetic techniques, improve postoperative pain management, furthermore, when administrated before surgery, these simple techniques can also decrease anaesthetic and requirement of analgesia during surgery as well as reduce the need for opioid containing analgesic postoperatively.

Inspite of several advantages of laparoscopic procedures over laparotomy, it does not take away the disadvantage like the post-operative pain which results in an unpleasant experience for the patient and thereby delay the discharge. Pain usually occurs on the first day following surgery and it may be a visceral, parietal or shoulder pain.

By evaluating the patho-physiology of pain it is shown that we can prevent or reduce pain by blocking the nociceptors before their stimulation by use of local anaesthetics.

Post-operative right upper abdominal pain and shoulder is a diffuse abdominal which was experienced on the day of surgery. The cause of this pain is thought to be related to abdominal muscle distension during laparoscopic procedure, irritative effects of residual Carbon-di-oxide in the abdominal cavity and prolonged elevation of diaphragm by pneumoperitoneum.

BACKGROUND

Post-operative pain after laproscopic cholecystectomy procedures are being rated from mild to severe in the literature. Post operative pain Most of the patients suffer from miled to severe pain after laparoscopic cholecystectomy. Different modalities for adequate control of pain management in these scenario are described including opioids, NSAIDs, local anesthetic infiltration of port site and local anesthesia infusion through epidural route, but none of them are proven to be consistently satisfactory.

AIMS AND OBJECTIVES

To study the impact of intravenous Lignocaine on intra-operative Haemodynamics, recovery characteristics, post-op pain scores, post-op analgesic requirement and return of bowel function in patients undergoing Laproscopic Cholecystectomy.

LAPAROSCOPY

Laparoscopy conventionally means visualization of the abdominal cavity through an especially designed scope (Endoscope).

TECHNIQUE OF LAPAROSCOPY

In laparoscopic surgeries, gas will be insufflated (usually carbondioxide) into the abdominal cavity under pressure to separate the organs from the abdominal wall. This produces a pneumoperitoneum, leading to increase in intra-abdominal pressure. Gas is insufflated in the peritoneal cavity at an initial rate of 4-6 liter per minute which gets cut off when the intra-abdominal pressure exceeds the pressure limits set by the clinician (usually 12-14 mm Hg). After the initial insufflations, the pneumoperitoneum and the pressure is maintained by a constant flow of 200-400 ml per minute of insufflating gas.

VENTILATORY AND RESPIRATORY CHANGES DURING LAPAROSCOPY WITH CO_2 PENUMOPERITONEUM

EFFECT OF RAISED INTRA-ABDOMINAL PRESSURE

There is a cephalad displacement of the diaphragam due to the increased intra abdominal pressure which causes reducing FRC and compliance by as much as 30% to 50%, Peak airway pressure and plateau pressures rise by 50% and 80%, respectively, and pulmonary compliance is reduced by 47%. This results in an increase in the airway resistance and work of breathing. Intrapulmonary shunting common in obese which is the cause for Hypoxemia.

INCREASE IN PARTIAL PRESSURE OF CO_2

There is increase in $PaCO_2$ when carbondioxide is used to create pneumoperitoneum. This $PaCO_2$ rises gradually and reaches a Plateau 20 to 30 min. after the beginning of intra-peritoneal insufflations. The increase in $PaCO_2$ values correlates with increase in the intra-abdominal pressure and ranges between 15% and 30%. In patients without respiratory or cardio-vascular co morbid illness, the increase in $PaCO_2$ results mainly due to CO_2 absorption from pneumoperitoneum. This pneumoperitoneum does not significantly modify physiologic dead space or shunt and as a result, the gradient between $PaCO_2$ and end-tidal

PCO_2 does not change. Due to this correlation end-tidal monitoring of Carbondioxide adequately reflects $PaCO_2$ values in blood and can be used to guide adjustment of ventilation so that hyper-capnia can be avoided. In patients with impaired pulmonary ventilation and perfusion matching like chronic obstructive pulmonary diseases, the resulting increased physiologic dead space and reduced alveolar ventilation leads to increased $PaCO_2$ to end-tidal CO_2 gradient. This leads to an increase in $PaCO_2$ values and adequate hyper-ventilation is required, so that hyper-capnia can be avoided. In these patients, capnography may not provide reliable measurement of $PaCO_2$. It is shown in studies that diffusion of Carbondioxide diffusion to the body is more during extra-peritoneal than intra-peritoneal insufflations. Also extra-peritoneal Carbondioxide insufflations lead to higher $PaCO_2$ values in the post-operative period.

CHANGE IN MINUTE VENTILATION

During laparoscopy with local anesthesia, minute ventilation increase by as much as 60% to maintain normo-capnea. If general anesthesia is maintained without muscle relaxation, i.e., with spontaneous breathing, the compensatory hyper-ventilation may not occur due to of anesthetic-induced respiratory depression leading to hyper-capnia. If the patients who were maintained on controlled ventilation, it is shown

that 15 to 20% increase in minute Ventilation is needed to prevent increase in $PaCO_2$.

RESPIRATORY COMPLICATIONS CAN OCCUR DURING LAPAROSCOPY

The major complication which can occur during Laparoscopy are :

1. Carbondioxide subcutaneous emphysema
   - due to extra peritoneal insufflations
2. Pneumothorax, Pneumomediastinum, Pneumopericardium
3. Endobronchial intubation
   - due to cephalad movement of the diaphragm
4. Gas Embolism
5. Aspiration of Gastric contents.

HEMODYNAMIC CHANGES DURING LAPAROSCOPY WITH CO_2 PNEUMOPERITONEUM

EFFECTS OF HYPERCAPNIA

Hypercapnia due to CO_2 pneumoperitoneum leads to activation of the sympathetic nervous system, that in turn leads to increase in arterial blood pressure, increase in heart rate, increase in myocardial contractility, and it may even lead to arrhythmias. Increase in Carbondioxide content in blood sensitizes the myocardium to circulating catecholamines, and this phenomenon is exaggerated particularly when inhalational anesthetic agents are used for maintenance of anesthesia.

EFFECTS OF RAISED INTRA-ABDOMINAL PRESSURE

Intra-abdominal pressure maintained less than 15mm Hg causes increase in venous return to heart, as blood is squeezed out of the splanchnic and mesenteric venous system, and leading to an increase in cardiac output.

On the other hand, when intra-abdominal pressure is maintained more than 15mm Hg, Cardiac output decreases transiently due to :

1. **DECREASED VENOUS RETURN**

   a. Inferior vena cava obstruction.

   b. Pooling of blood in the legs

   c. Increased venous resistance

2. **MYOCARDIAL DEPRESSANT EFFECT OF ANESTHESIA INDUCTION AGENTS**

3. **RAISED SYSTEMIC VASCULAR RESISTANCE**

   a. Release of neuro-humoral factors

   b. Increased vascular resistance of organs

The increase in systemic vascular resistance causes an increase in arterial pressures. Central venous pressure and pulmonary artery wedge pressure are elevated due to increased intra-thoracic pressure and in the scenario of pneumoperitoneum these cannot be considered as reliable indicators of cardiac filling pressures. Cardiac arrhythmias during laparoscopy are due to multiple causes-hypercapnia, peritoneal stimulation, reduced venous return, hypo-volemia and venous gas embolism.

EFFECTS OF PNEUMOPERITONEUM ON OTHER ORGAN SYSTEMS

Renal Effects : There is a decrease in renal blood-flow and Glomerular Filtration rate due to the reduction in cardiac output. Renal vascular resistance may also increase due to the raised intra-abdominal pressure. This can manifest as a reduction in intra-operative urine output. Patients with pre-existing renal dysfunction are at risk of further deterioration. However, normally these changes are reversible and the urine output improves once intra-abdominal pressure comes down at the end of surgery.

Splanchnic and Hepatic Blood-Flow: The effect of penumoperitoneum on splanchnic and hepatic blood-flow is unclear. Anim al studies suggest that while hypercapnia has a vasodilatory effect, this is opposed by the vasoconstricting effect of the raised intra-abdominal pressure.

Gastrointestinal Effects: Raised intra-abdominal pressure can predispose the patient to regurgitation and aspiration. However, it is believed that the changes in the intra-abdominal pressure are transmitted to the lower esophageal sphincter, preventing this complication.

Neurologic Effects: The combination of factors like hyper-capnia, head-low position, and elevated systemic vascular resistance can leads to increase in intra-cranial pressure with a resultant decrease in cerebral perfusion pressure. This may be exaggerated in patients with ventriculoperitoneal shunts which do not have a unidirectional valve.

Ocular Effects: A slight increase in intraocular pressure has been described, though its clinical significance is not clear.

Neuroendocrine Effects: Increased levels of stress hormones. Increase in ADH and aldosterone levels.

EFFECTS OF POSITION

Laparoscopic Cholecystectomy is usually carried out in the reverse Trendelenberg positon, with the patient's arms by the side. This position helps by displacing the abdominal organs far from the surgical site, to facilitate surgical accessibility.

For laparoscopic procedures depending on site of surgery either trendelenburg (for pelvic surgeries) or reverse trendelenburg (for upper abdominal surgeries) position is used. The effects of surgical positioning are:

- **Reverse Trendelenburg (Head-up):** Preload is decreased, resulting in lowered pressure (MAP). Blood pooling in the lower extremities may increase the risk of venous thrombosis and pulmonary function is improved.
- **Trendelenburg Position (Head down):** Cardiac output and central venous pressure increase. Pulmonary effects include impaired diaphragmatic function secondary to the Cephalad displacement of abdominal viscera, resulting in decreased functional residual capacity, decreased total lung capacity, and decreased pulmonary compliance, predisposing the patient to developing atelectasis. Cephalad movement of the trachea may result in endobronchial intubation.

Intravenous Lignocaine has been used to suppress cough during tracheal intubation, Laryngospasm and cough during extubation. It has also been used to suppress airway hyperactivity and mitigate broncho-constriction after tracheal intubation.

PHYSIOLOGY OF PRESSURE RESPONSES

Direct laryngoscopy and tracheal intubation following induction of anaesthesia is almost always with hemodynamic changes due to reflex sympathetic stimulation. Herein, with have explained briefly, the

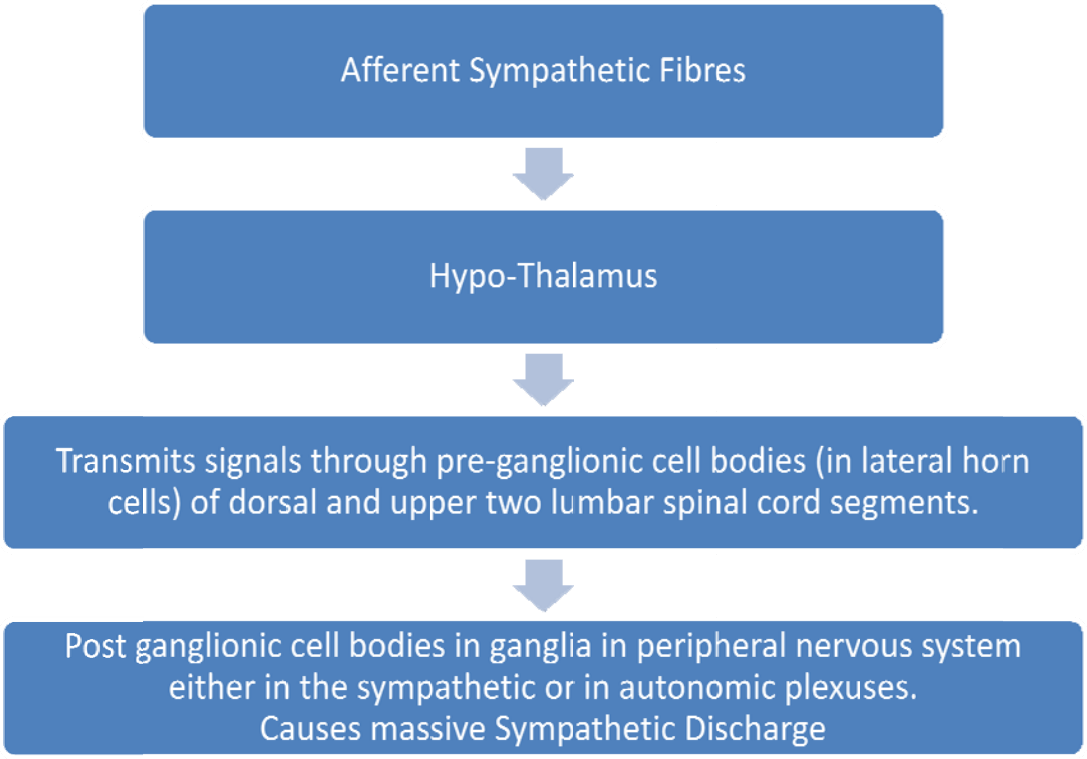
Physiology and effects of Laryngoscopy and Intubation to understand the haemo-dynamic changes.

SYMPATHETIC NERVOUS SYSTEM

The sympathetic efferent nerve fibres originate from nerve cells in the lateral grey column of the spinal cord between the 1stthoracic and 2nd lumbar segments. Pre-ganglionic fibres are myelinated. Ganglia situated either in the para-vertebral sympathetic trunk or in pre-vertebral ganglia, such as the celiac ganglion. The sympathetic part of postganglionic fibres are long, non-myelinated sympathetic part of the system has a wide spread action on the body as the resulting preganglionic fibres synapsing on many postganglionic nerve fibres and the suprarenal medulla releasing the sympathetic transmitters epinephrine and nor epinephrine. The sympathetic nervous system prepares the body for emergency situation and sever muscular activity. There is no sympathetic out flow from cervical part of the cord nor from the lower lumbar and sacral parts. Those pre-ganglionic fibres which are destined to synapse with cell bodies whose fibres are going to run with cervical nerves must ascend in the sympathetic trunk and those of lower lumbar and sacral nerves must descend in the trunk to lumbar and sacral ganglia.

AFFERENT SYMPATHETIC FIBRES

Cell bodies of all the afferent sympathetic fibres are located in the dorsal root ganglia of spinal nerves. The afferent fibres reach the spinal nerve in the white Ramus communicants. Central processes enter the spinal cord by posterior nerve root. From there, they ascend through the cord to brain stem.



THE CARDIOVASCULAR REFLEXES

The cardiovascular and hemodynamic responses to airway manipulation are caused by firing of proprioceptors located in the supraglottic region and tracheal mucosa. These pro-prioceptors are actually mechano receptors consisting of small diameter myelinated fibers; slow adapting stretch receptors with large diameter myelinated fibres and polymodal endings of non-myelinated nerve fibers. These receptors are located in close proximity to airway mucosa. Stimulation of these receptors lead to afferent impulse to brain stem transmitted through glossopharyngeal and vagal nerves and this in turn leads to activation of autonomic nervous system including both sympathetic and parasympathetic nervous system manifesting as hemodynamic and cardiovascular events. The superficial location of the pro-prieoceptors and their nerves has the advantage that topical anaesthesia of the airway with local anesthetics provides an effective means of blunting cardiovascular and hemodynamic responses to airway manipulations.

In adults and adolescents, the most common result of airway manipulation is increase in blood pressure and increase in heart rate. These are mediated by the sympathetic chain ganglia and cardio accelerator fibers arising from them. This response leads to secretion of

noradrenaline from sympathetic nerve terminals and adrenaline from the adrenal medulla.

Following intubation stimulation of cerebral cortical activity is also shown to be increased leading to increase in elctro-encephalographic activity, increased metabolic rate and increase in cerebral blood-flow corresponding to increase in metabolic activity.

PHARMACOLOGY OF LOCAL ANAESTHETICS STRUCTURE ACTIVITY RELATIONSHIP OF LOCAL ANAETHETICS

Local Anaesthetics contains lipophilic group, a Benzene ring, separated from a hydro-philic group, usually a tertiary amine, by an intermediate chain which includes an ester or amide linkage. Local Anaesthetics are weak bases. At physiological pH they usually carry a positive charge at the tertiary amine. On the basis of the nature of the intermediate chain local anaestheticsare classified into Amide or Ester groups. Local anaesthetics act by penetrating lipoprotein cell membrane in the non-ionized state. In order to make them suitable for injection, the non-ionized base has to be converted to the ionized state by injecting them in an acid solution as the hydrochloric salt so, tertiary amine group becomes quaternary and then they become water soluble and suitable for injection.

STRUCTURAL ACTIVITY RELATIONSHIP

A) POTENCY

An increase in the lipid solubility and/or increase in the molecular weight increase the potency of local anaesthetic, e.g., adding a butyl group to Mepivacaine (less potent local Anaesthetic) converts it into Bupivacaine (more potent local Anaesthetic).

POTENCY IS AFFECTED BY

1. **Fiber Size:** The smaller unmyelinated fibers (e.g. sensor C Fibers) are more effectively blocked than the large myelinated fibers (motor A Fibers).
2. **pH:** Acidic environment decreases the potency.
3. **Nerve Firing Frequency:** Access of local Anaesthetic to Na channels is enhanced by repeated opening of those channels.

B) SPEED OF ONSET OF ACTION: AND THIS DEPENDS ON:

1. **pKa of the Drug:** When the pKa of local Anaesthetics are closer to the physiologic pH the onset of action is enhanced. This is due to higher concentration of the non-ionized free base at

physiological pH that enhances the drug crossing the nerve cell membrane.

2. *Molecular weight:* Local anaesthetics with smaller molecular weight have more rapid onset of action.

C) DURATION OF ACTION

It depends on the aromatic group which affects the plasma protein binding. The higher the plasma protein binding the slower the clearance and thus the higher the duration of action. Also, higher protein binding increase the duration of affection of the local anaesthetic to the Na channels and thus prolong the action.

The myelinated nerves are protected by the myelin sheath which acts as an insulator. Outside of nerve membrane a resting potential of –70mV is maintained when there are no stimulation, and called as resting membrane potential. During any stimulation the Resting membrane potential rises to about –55mV , the firing threshold, before it jumps upto +20mV to form an action potential which constitutes a change of about 90 mV. This is caused by influx of sodium ions and efflux of potassium ions through ion channels located in the cell wall. The cells thus become depolarized. During recovery, the ionic movement occurs in the opposite direction and the cell once again regains the negative resting membrane

potential. Local Anaesthetics block the sodium channel and thus prevent the depolarization in the neuronal membrane and prevents conduction of impulses.

PHARMACOKINETICS OF LOCAL ANAESTHETICS

1. ABSORPTION

Factors that affect the absorption of local Anaesthetic are:

a. **Site of Injection:** Highly Vascular tissues show increase in the systemic absorption of local Anaesthetic and thus increase toxicity (I.V > tracheal > epidural > sub-cutaneous).
b. **Presence of Vasoconstrictors:** Vasoconstrictors decrease the systemic absorption and thus decrease the toxicity, this is only effective in short acting local anaesthetic, e.g. Lignocaine.
c. **Type of local Anaesthetic:** Local Anaesthetics with high tissue binding are more slowly absorbed. e.g. Etidocaine.

2. DISTRIBUTION

Distribution of local Anaesthetics are affected by:

a) **Tissue perfusion:** Highly perfused Organs (Brain, Liver) show higher uptake than poorly perfused organs (muscles and fat)

b) **Plasma Protein Binding:** the higher the protein binding the longer the time of retain of local anaesthetic in the blood.

3. METABOLISM

The Pharmacokinetic Property (Metabolism, Distribution and Excretion) of local Anaesthetics differ depending upon their chemical structure. Local anaesthetics with ester group are predominantly metabolized by pseudocholinesterase. Also one of the metabolites of ester local anaesthetics is P-Amino-Benzoic Acid (PABA) which is highly allergenic. In patients, who have genetically abnormal pseudo-cholinesteras enzyme there is increased risk of toxic effects with this group of local anesthetic agents.

Amide local anaesthetics are metabolized by microsomal enzymes in the liver. Metabolism will be impaired in patients with altered hepatic function or there is a decrease in hepatic blood flow thus leading to systemic toxicity. However allergic manifestations are less common with these group of local anesthetics.

4. PROTEIN BINDING

Local Anaesthetics are bound to plasma proteins to varying degrees. It is assumed sometimes that drugs with the greatest degrees of

protein binding are less toxic because only a small fraction of the total amount in plasma is free to diffuse into the tissues and produce toxic effects. Furthermore, even if a drug is bound to protein, it is still available to diffuse into the tissues down a concentration gradient, as the bound portion is in equilibrium with that in solution in plasma.

PHARMACOLOGY OF LINGNOCAINE

Lignocaine was synthesized in 1943 in Swedan by Lofgren, it was introduced into anaesthesia practice by Gordh in the year 1948.

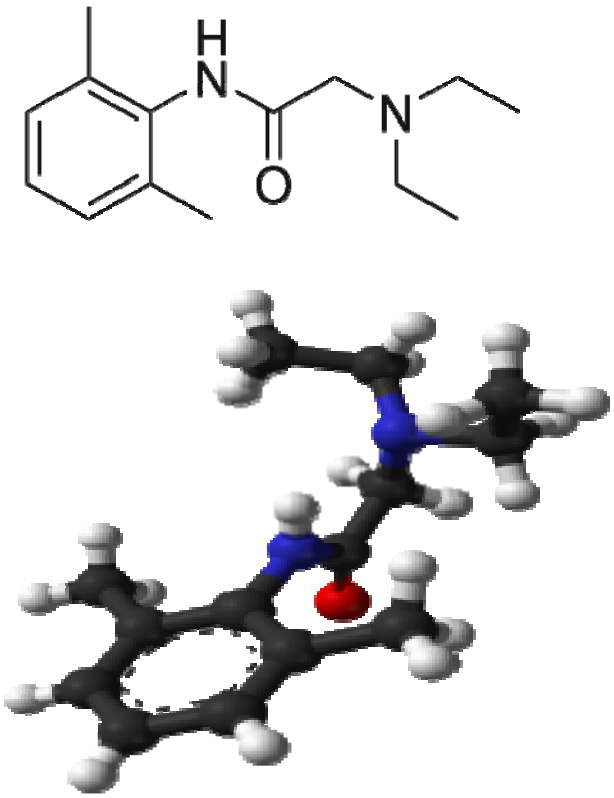


Figure 1

2-(Diethylamino)- *N*- (2,6-Dimethylphenyl) Acetamide

Lignocaine is a synthetic amino amide derivative of local anesthetics with intermediate potency and duration. It is the standard local anesthetics to which all other agents are compared. It is the most commonly used local anesthetic. It can also be used as an anti-arrhythmic. It can be given by all routes. (intravenous, intrathecal, infiltration, local applications like ointment, sprays, jelly, etc.)

MECHANISM OF ACTION

Lignocaine after entering the cell membrane binds to the inner portion of the resting sodium channel receptor. It prevents the propagation of nerve impulse through the ion-selective sodium channel in the nerve membranes. This decreases the depolarization rate, so that the threshold potential is not reached. Thereby, action potential generation is inhibited, without altering the membrane potential.

PHYSIO-CHEMICAL PROPERTIES

Molecular Weight : 234

Weak Base with a pKa : 7.6 – 7.8

Stable, not decomposed by heating Acids or Alkalies compared to Bupivacaine, it is less lipid soluble

PHARMACOKINETICS

ABSORPTION

Lignocaine is absorbed to systemic circulation from the site of application or injection. Blood-flow to that area and use of Epinephrine determines the rate or absorption.

METABOLISM AND EXCRETION

It is metabolized in liver by Oxidative de-Alkylation to mono-ethyl glycin exylidide which on further hydrolysis produces xylidide. Hepatic blood flow determines its metabolism. 80% activity of the parent drug is by mono ethyl glycine xylidide and 10% of its activity by Xylidide. 75% of Xylidide is excreted in the urine as 4–hydroxyl – 2,6 – dimethylaniline.

ONSET OF ACTION

The onset of action varies with the site of application of Lignocaine. For topical Anaesthesia, its onset 5–10 minutes, For conduction anaesthesia, in small nerves, 5-10 minutes. For large nerves, 10–15 minutes. For intravenous administration 1–2 minutes.

70% of the drug binds to α1 Acid Glycoproteins. Volume of distribution 91 liters.

Lignocaine has a triphasic distribution.

Rapid Distribution Phase (A) : During this phase, the drug is distributed to highly vascular regions with t ½ & of 1 minute.

Slow Disappearance Phase (B) : Here the drug is distributed to slowly equilibrating tissues with t½ 1b pf 9.6 min. Finally the drug goes to slow transformation and Excretion phase (S) :

Where the t½ is 1.6 hrs.

Clearance – 0.95 litres per minute.

AVAILABILITY

- 2% lignocaine ointment, 5% lignocaine ointment
- 10% lignocaine spray
- 2% lignocaine Jelly
- 4% lignocaine aqueous solution
- 4% lignocaine viscous
- 4% lignocaine with adrenaline as 1 in 200000 dilution – 30 ml vial.
- 2% lignocaine with adrenaline as 1 in 200000 dilution

- 2% Lignocaine – plain – 30 ml vial – contains methyl and propyl paraben as preservative
- 2% Lignocaine (Xylocard) without preservative – 50 ml vial for intravenoususe
- 5% heavy 2 ml ampoules which contain 50 mg of lignocaine/ml with 75 mg 100 mg of dextrose

PHARMACODYNAMICS

LOCAL ACTIONS

It acts locally causing loss of pain, touch and temperature sensation. Motor power and vasomotor tone is also lost in the region supplied by the nerves blocked.

SYSTEMIC ACTIONS

Its action depends on the site/route (intravenous) of administration.

CARDIOVASCULAR SYSTEM

It stabilizes the myocardial cell membrane. It depresses myocardial automaticity by inhibiting the action potential and reducing the duration of effective refractory period. At higher concentration, cardiac

conductivity and contractility are depressed. Lignocaine causes blockade of sodium channels in cardiac muscle leading to membrane changes and cardiac toxicity. It suppresses ectopic foci by stabilizing the membrane of damaged and excitable cells.

VASCULAR SMOOTH MUSCLE

It acts on vascular smooth muscle producing vasodilatation.

RESPIRATORY SYSTEM

Lignocaine depresses the ventilator response to low PaO_2 (Hypoxic drive). Direct exposure of local anaesthetic to higher centres like medulla or peripheral nerves (intercostals, phrenic nerve) causes respiratory depression. Lignocaine has the property of relaxing bronchialsmooth muscles.Intravenous lignocaine is shown to be effective in reducing the reflex bronchoconstriction that can occur following airway manipulation.

Central nervous System: Lignocaine causes a sequence of stimulation followed by depression. It causes sedation on intravenous administration. Sympathetic stimulation associated with airway manipulation leads to increased cerebral blood flow and increase in intra cranial pressure. Intravenous administration of lignocaine is shown to blunt the sympathetic surge and attenuate these responses. Lignocaine

injection is capable of reducing the Minimal Alveolar Concentration of volatile Anaesthetics by 40%.

Musculo-Skeletal : Lignocaine causes lytic degeneration, edema and necrosis of muscle fibres.

Hematological : It enhances fibrinolysis thereby increasing the clotting time.

INDICATIONS

For infiltration block, peripheral nerve blocks, topical anaesthesia, intravenous regional anaesthesia, intra cuff injection (with sodium bicarbonate), spinal and epidural anaesthesia

ANTI-ARRHYTHMIC

Lignocaine is an antiarrhythmic drug because of cardiac membrane stabilizing properties. It is classified under class IB anti arrhythmic drug. It is used in the treatment of

- Ventricular tachyarrhythmia
- Arrhythmia due to acute myocardial Infarction during cardiac surgery

- Digitalis toxicity – since it does not worsen Atrio Ventricular – block

1. It is used to prevent increases in intracranial pressure during intubation antitussive effect may be the reason for this action
2. It suppresses noxious reflexes (coughing) & sympathetic stimulations associated with endotracheal suctioning and intubation.
3. It is used intravenously as an analgesic for chronic pain states
4. It is used as a supplement to general anaethesia

CONTRA-INDICATIONS

1. It should not be used in patients with (hypersensitivity) allergic disorder.
2. It should not be used along with vasoconstrictor in digits of hand, feet and penis
3. It is contraindicated in patients with stokes Adams syndrome, severe degree of heart block

DOSES :

MAXIMUM RECOMMENDED DOSE :

Plain `` - 3mg / kg

Plain with adrenaline - 7mg / kg

Plain without preservative for reflex suppression – 1.5 mg / kg iv.

DRUG INTERACTIONS

B Blockers : Co-administration of beta blockers, increases the toxicity by increasing the serum levels of Lignocaine by reducing Lignocaine by reducing Lignocaine's Metabolism.

Anti-Convulsant Agents : It increase the metabolism of lignocaine

Non-Depolarizing Muscle Relaxants : Lignocaine potentiates the blockade of non-depolarizing muscle relaxant.

Opioids and a 2 Adrenergic Agonists : These drugs increases the analgesic action of Lignocaine.

Anti-Arrhythmic Agents : Anti Arrhythmic Effect of Lignocaine is increased by these agents.

TOXICITY :

Lignocaine Toxicity is due to systemic absorption of locally administered Lignocaine or due to accidental intravenous administration of large doses of Lignocaine. The central nervous system is most commonly affected.

SYMPTOMS AND BLOOD LEVELS

Light headedness, Tinnitus, Circumoral and Tongue Numbness (Anti-convulsant and Anti-arrhythmic activity) : 4 micrograms / ml

Visual disturbances	:	6 micrograms / ml
Muscular twitching	:	8 micrograms / ml
Convulsions	:	10 micrograms / ml
Unconsciousness	:	12 micrograms / ml
Coma	:	15 micrograms / ml
Respiratory arrest	:	20 micrograms / ml
Cardiovascular collapse	:	26 micrograms / ml

TREATMENT OF TOXICITY

Lignocaine toxicity can be identified earlier by continuous monitoring of Cardiovascular and Respiratory status.

- Barbiturates or Benzodiazepines are used to treat convulsion
- Succinylcholine 1 mg/kg used to paralyze and to intubate the patient to have airway control during seizures.
- Ventricular fibrillation can be treated by defibrillation.
- 100% oxygenation, intubation and ventilation.
- Maintain Blood Pressure by rapid infusion of Intra Venous fluids, use of vasopressors and put the patient in Trendelenberg's position.
- Treat electrolyte imbalance.

ADVERSE EFFECTS

1. **Allergic and hypersensitivity reactions:** Methyl paraben used as a preservative causes allergic and hypersensitivity reactions.
2. **Cardio-vascular System :** Hypotension, bradycardia.

MECHANISM OF ACTION OF LOCAL ANAESTHETICS

The local anaesthetics inhibit the conduction of impulses across the nerves by the following mechanism as defined by Carvino. The local anaesthetic drug exists in both charged and uncharged forms.

The Relative concentrations of the two forms are dependent on the pKa of the solutions, pH of the site where injected. The positively charged cation form is the active form. It produces local anaesthetic action.

The uncharged base form is responsible for the diffusion across the liphophilic membranes across the cell. The drug acts from the inside of the cells on sodium ion channel. They occupy specific receptors on the inner side of sodium channel and inhibit the conduction of ions through them. Thus the cell remains in a state of persistent depolarization. This inhibits the propogation of action potential.

Other probable site of actions are channel narrowing and membrane expansion due to nonspecific absorption across the cell membrane. Unchanged base form diffuses across hydrophobic pathways

of lipid membranes to reach specific receptor sites and protonation of drug to bind to inner opening of sodium channel.

THE SURFACE CHARGE THEORY

This theory is based on penetration of the axonal membrane by lipophilic portion of the local anaesthetic drug and neutralization of axolemmal negative charges on surface by the positively charged terminal amino group of drug. The electro-negativity of the external membrane is counteracted by the acquired positive charges. This results in increase in the transmembrane potential without altering much of the intracellular resting potential. This inhibits the conduction of nerve impulses from the normal areas to anaesthetized areas of the nerve membrane. Thus it produces a conduction block across the two portions. According to surface charge theory the active form of local anaesthetic drug is the charged form of drug.

DEFINITION OF PAIN

Pain is defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage.” This definition of pain considers the interplay between the objective, physiologic sensoryaspects of pain and its subjective, emotional and psychological components.

Pain is clinically divided into, acute pain which is primarily due to nociception and chronic pain, which may also be due to nociception, but in which psychological and behavioral factors often play a major role. Postoperative pain is one of the types of acute pain and can be further differentiated based on the origin and feature into somatic and visceral pain. Somatic pain occurs due to abnormal nociceptive input from skin, subcutaneous tissues and mucous membranae.

It is well-localized and usually described in terms of sharp, pricking, throbbing or burning sensation. Visceral pain on the other hand is due to nociceptive input arising from internal organ or one of its covering. It is usually dull diffuse pain. This visceral pain usually accompanied by abnormal parasympathetic and sympathetic manifestations like nausea, vomiting and sweating. It is also associated with changes in blood pressure and heart rate.

MAGNITUDE OF THE PROBLEM

Occurrence, intensity, quality and duration of postoperative pain is influenced by multiple factors including the site, nature and duration of operation, type of incision (thoracic and upper abdominal operations are associated with the most severe pain), the preoperative psychological, physical and pharmacological preparation of the patient, added to this the anaesthetic management and the quality of post operative care (the attitude of the ward staff).

NEURO-PHYSIOLOGY OF PAIN

NOCICEPTORS

Sensation is often described as either Protopathic (Noxious) or Epicritic (non-noxious). Epicritic sensation (light touch, pressure, proprioception, and temperature discrimination) is characterized by low-threshold receptors (specialized endorgans on the afferent neurons) and conducted by large myelinated nerve fibers while; Protopathic Sensation (pain) is sub served by high-threshold receptors (free nerve endings).

Noxious sensations have two components: a fast, sharp, and well-localized sensation “first pain” which is conducted by Aδ fibers; and a duller, slower onset, and poorly localized sensation “second pain”

which is conducted by C fibers. This protopathic pain is transmitted mainly by free nerve endings that sense mechanical or chemical tissue damage.

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SEVERAL TYPES OF THIS PAIN IS RECOGNIZED

1. **Mechano nociceptorsm :** responds to sharp stimulus like pinprick,
2. **Silent nociceptors :** responds only on the presence of inflammatiory mediators
3. **Polygonal mechano heat receptors :** more common type of receptors, they respond to excessive pressure and extreme of temperature, they also respond to pain producing substances.

Nociceptors are either somatic that include those in skin and deep tissues (muscle, tendons, joints), or visceral nociceptors that include those in internal organs.

PAIN PATHWAY

Pain is conducted along three neuron pathways; from the periphery to the cerebral cortex.

FIRST ORDER NEURON

Neuronal cell bodies of the first order fibers are located in the dorsal root ganglia (for the body) and specific cranial nerve ganglia (for the head and neck) e.g. Gasserian ganglion for trigeminal nerve. The Proximal end of their axons reach spinal cord via the dorsal sensory root of cervical, thoracic, lumbar, and sacral level (for the body) and through the cranial nerves (for head and neck).

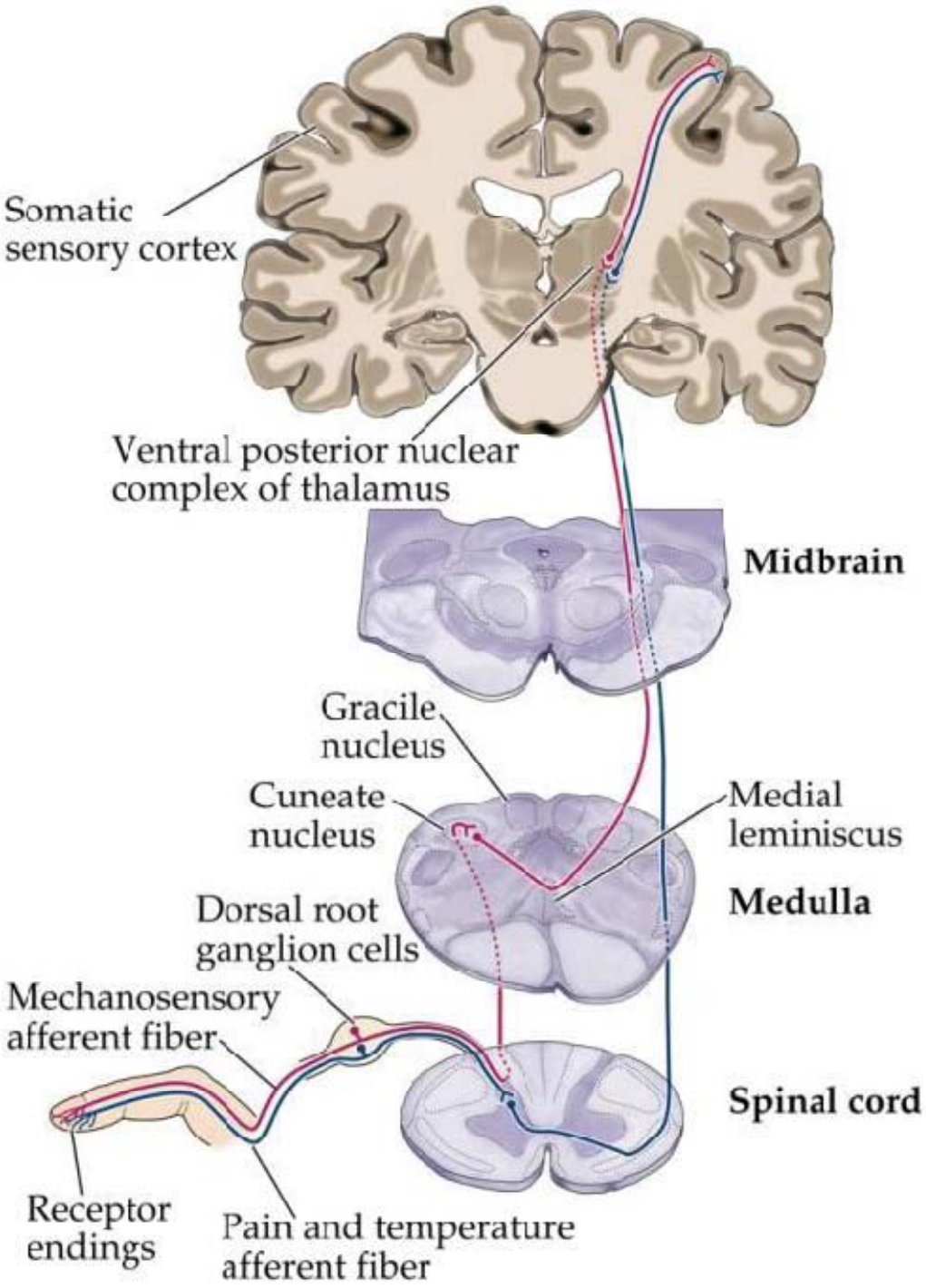


Figure – 2

PAIN PATHWAY

SECOND ORDER NEURONS

Pain fibers may ascend or descend in the spinal cord for approximately three segment levels in the Lissauer‟s tract and then they synapse with the second order neurons in the gray matter of the ipsilateral dorsal horn, this synapsing may be through interneurons. Second order neurons are either; nociceptive specific which serves only noxious stimuli and are normally silent or wide dynamic range (WDR) neurons that can receive also non-noxious afferent input. WDR neurons are more prevalent in the dorsal horn and are responsible for the increased intensity of firing in response to same stimulus “wind-up”.

Lamina II of the gray matter of the dorsal horn of the spinal cord, (also called the substantiagelatenosa) contains many interneurons and is believed to play a role in the processing and modulating nociceptive input.

Most of the Axons of second order neurons cross the midline to the contra-lateral aspect of the white matter of spinal cord forming the lateral spinothalamic tract that relays in the thalamus, synapses with the reticular formation fibers, synapses in raphynucleus. They also relay in periaquidactal gray matter 23,24.

THIRD ORDER NEURONS

Thalamus is the relay center for second order neurons. The nerve cells of third order neurons are situated in the thalamus. These neurons send their axons to relay in the Somato-sensory area I and II of the Cerebral cortex 24,25,26.

PREEMPTIVE ANALGESIA

Preemptive Analgesia is defined as what is administered before surgical incision that prevent the development of central sensitization from incisional injury and inflammatory injuries (i.e., intra-operative and postoperative periods). Review of animal experiments, volunteer studies and clinical trials suggests that preemptive analgesia is a clinically useful and relevant phenomenon. Maximum benefit of preemptive analgesia protocol can occur only when there is near complete blockade of noxious stimuli is achieved.

PAIN AFTER LAPAROSCOPY

Studies have shown that pain after laparoscopy is less severe than same procedures done with conventional open procedures (Laparotomy). The reduction in pain has made the early discharge from the hospital

possible, provided that the control of the residual pain is adequate and that the drugs or techniques used for analgesia are safe.

Various clinical reports have shown that pain after laproscopic cholecystectomy may be localized in the upper abdomen, lower abdomen, in the back, or to the shoulders. Upper abdomen pain more common in laproscopic surgery. It is shown that use of suxamethonium to facilitate tracheal intubation has no correlation with post laparotomy pain severity.

Pain after laparoscopic procedures are usually transient, rarely it may persist for two to four days. After laparoscopic cholecystectomy, visceral pain was found to predominate in the first 24 hours, whereas shoulder pain was less severe in first post operative day and gradually increases and becomes significant on the successive days 28.

MECHANISM OF PAIN IN LAPAROSCOPY

In addition to the trauma caused to the abdominal wall and the visceral organs by the endoscope and the surgical instruments, there are other mechanisms responsible for pain after laparoscopy. Rapid insufflation of Carbondioxide causes distension of the Peritoneum, trauma and tearing of small arteries and veins, traction of the nerve fibers and increase in release of inflammatory mediators and all these factors can contribute to development of post operative pain. Inflammation of the peritoneum is proposed to be the reason for origin of the upper abdominal pain after lower abdominal surgery or after diagnostic laparoscopy. Studies have shown that biopsy of peritoneum performed 2-3 days after laparoscopy showed inflammation and neuronal rupture. This pain may last for two to three days.

Therefore, abdominal distention should better be slow with adequate muscle relaxation to ensure suitable abdominal compliance. Persistant gas in the abdomen may lead to excitation of Phrenic nerve presenting as persisting pain in the shoulder tip. Studies have shown strong correlation between gas bubble dimension and severity of pain score and it is also shown that aspiration of the gas under the diaphragm also decreases the pain score.

A: FACTORS INVOLVING WITH GASEOUS PNEUMOPERITONEUM

1. **Phrenic nerve injury (Neuropraxia) :** It is due to distention of the diaphragm during gas insufflations. The resultant phrenic nerve neuropraxia possibly contributes to the shoulder pain due to the C4 dermatome involvement (referred pain).
2. **The nature of insufflated gas and pH of Abdominal Cavity:** CO_2 in the abdominal cavity may dissolve and create an acidic environment. Studies have shown that intra-peritoneal pH was 6.0 in the immediate post operative period and in the second post operative day it was recorded between 6.8–6.9 and then it gradually over a period of days normalizes to 7.030. The phrenic nerves may get irritated and damaged by the acidic environment and lead to the referred pain in the shoulder. The acidic environment can also trigger inflammation of the peritoneum and lead to persistant abdominal pain.
3. **Intra-abdominal Residual Gas:** Several studies have shown correlation between residual intra-abdominal gas after laparoscopy and the severity of post-operative pain. Residual gas leads to prolonged intra-abdominal acidosis leading to prolonged peritoneal irritation thus increasing the morbidity. Residual gas is

also shown to alter the peritoneal surface tension leading to loss of support to the abdominal viscera. This lack of support leads to traction and visceral pain.

4. **Temperature of the insufflating Gas:** Prospective randomized study conducted in laparoscopic gynaecological procedures comparing standard insufflation gas and gas insufflated in body temperature showed that pain intensity especially diaphragmatic and shoulder pain was significantly low in patients who were insufflated with warm gas and this low pain scores were observed for three days.
5. **Humidity of Gas:** Humidity of gas reduces the post-operative pain laproscopic and abdominal surgery. It significantly reduces the post-operative ileus.

B:OPERATIONAL FACTORS

1. **Wound Pain :** Various type of abdominal incision done in laproscopic surgery. Sub costal incision causes nerve fibres damage. It leads to postoperative neuralgia.
2. **Abdominal Drainage :** Abdominal drains are placed after laparoscopic surgery usually through one of the port site. It is done in the lateral abdominal wall and the incision traverses

multiple muscle layers. The umbilical port is usually avoided as it is related with greater incidence of pain, more risk of infection, and potential risk of incisional herniation if the defect is not closed in proper manner. Persistent drainage tube cause more inflammation leading to increase in post operative pain scores. Rather than routine procedure individualization for insertion for abdominal drain will help in reducing pain scores.

C: PERSONALITY AND SOCIO-CULTURAL FACTORS

Apart from surgical factors the personality of the patient and the socio-cultural factors can also influence the post-operative pain management, recovery of bowel movements and length of hospital stay. Previous pain experience, individual threshold for pain will also influence the individual postoperative pain perception and recovery time. Anesthesiologists have the responsibility of assessing the patients and providing adequate pain relief with appropriate technique on individual basis.

Studies have shown that there is a substantial inter-individual variation in the occurrence, intensity and tolerance of pain after laparoscopic cholecystectomy. The intensity of pain after laparoscopic cholecystectomy peaks within the first 4-8 hours, has been reported to be

unbearable upto the first postoperative morning in one third of the patients. Pain after laparoscopic cholecystectomy involves three different components – incisional pain – involving parietal component deep intra abdominal pain – visceral component shoulder tip pain – presumed referred pain from visceral component. These components differ with intensity of stimulation, duration of stimulus, and pathophysiological mechanisms. Studies have shown that in the immediate postoperative period the intensity of visceral pain dominates the other components.

EFFECTS OF POST-OPERATIVE PAIN

Acute post-operative pain regardless of intensity and regardless of the site can affect all organ system, alter the homeostasis and strongly influences the postoperative morbidity and mortality.

Consequences of acute post-operative pain is classically due to activation of sympathetic nervous system and the neuro endocrine stress response. Studies have shown that reduction in perioperative stress responses (Endocrine, Metabolic and Inflammatory) decreases the incidence of postoperative organ dysfunction, will lead to a reduced morbidity and mortality and thereby to an improved surgical outcome.

(A) CARDIO-VASCULAR EFFECTS

Cardiac disease is a major cause of perioperative death. The realization that, in high risk populations, perioperative myocardial ischemia is most likely to occur after surgery (from day one to day three postoperatively) has led to treatment strategies designed to prevent its development.

Although a variety of factors may contribute to the development of postoperative myocardial ischemia, including hypothermia, anemia, anxiety, and tracheal intubation / suctioning, responses to poorly

controlled pain play a prominent role. In this regard, activation of sympathoadrenal, and neuroendocrine axes may have a major impact on myocardial oxygen supply and demand. Catecholamine-induced tachycardia, enhanced contractility, increased after load and increased preload from hypervolemia caused by enhanced release of arginine vasopressin and aldosterone, are well characterized determinants of increased oxygen demand. Increased oxygen demand, with hypervolemia, may precipitate ischemia and acute cardiac failure, especially in patients with poorly compensated coronary artery or valvular heart disease.

Myocardial oxygen supply may be diminished as a result of pulmonary dysfunction, in particular, atelectasis secondary to pain-induced hypoventilation and pulmonary edema resulting from stress-induced hypervolemia. Other causes of reduced oxygen supply include coronary artery constriction secondary to high circulatory levels of catecholamine and increased coronary sympathetic tone, stress-induced increase in plasma viscosity and platelet-induced occlusion; and serotonin induced coronary vasospasm secondary to platelet aggregation.

(B) PULMONARY EFFECTS

Pulmonary function may be dramatically altered by surgically induced pain. The classical pulmonary response to upper abdominal

surgery, include an increase in respiratory rate with decreased tidal volume, vital capacity, forced expiratory volume and functional residual capacity. Those pathophysiologic alterations are characteristic of acute restrictive pulmonary disease and, as such, may be associated with clinically significant hypoxia and hypercarbia.

Pain, due to activation of sympathetic nervous system causes increase in total body oxygen consumption and carbon-di-oxide production. This effect mandates that work of breathing has to be increased. Patients who undergo upper abdominal and thoracic surgical procedures are in high risk of inadequate ventilation, especially when post operative pain is not adequately controlled. Reduced depth of breathing and inadequate cough leads to further reduction in the tidal volume and functional residual capacity which in turn can cause atelectasis, intrapulmonary shunting and hypoxemia. This leads to further increase in post operative morbidity and mortality.

(C) GASTROINTESINAL EFFECTS

Sympathetic hyperactivity induced by pain increases sphincter tone and decrease motility of intestine, causing ileus, pain also increases stress ulceration due to increase in gastric acid secretion.

(D) ENDOCRINAL EFFECTS

The dominant neuro endocrine responses to pain involve hypothalamic – pituitary - adrenocortical interactions. Those interactions result in increased catecholamine and catabolic hormone release. This effects causes sodium and water retention, and increased levels of blood glucose, free fatty acids and lactate. Sympathetic activation also causes negative nitrogen balance and protein breakdown (catabolism) further impeding post operative outcome.

(E) HEMATOLOGICAL EFFECTS

The neuroendocrine stress response causes decrease in the levels of natural anticoagulants, inhibition of fibrinolysis and increase in platelet reactivity which initiate a postoperative hypercoagulable state. This hypercoagulability causes a series of other events such as deep venous thrombosis and myocardial ischemia.

(F) IMMUNOLOGICAL EFFECTS

The stress response potentiate postoperative immune suppression; the extent of which correlates with the extent of surgery. Stress response has been reported to depress the reticulo-endothelial system which predispose to infection.

G) PSYCHOGENIC EFFECTS

Intense anxiety, fear, and the loss of control that accompany severe tissue injury may have profound impact on the hypothalamic-pituitary axis. Behavioral responses associated with poorly controlled pain include sleep deprivation and reduced morale.

In many patients, uncontrolled postoperative pain can produce a series of long-term emotional disturbances, which could impair the patients' health, and cause undue fear and anxiety if subsequent surgery is required. Studies have shown that up to 20% of major non-cardiac surgical patients experience post-operative cognitive dysfunction. This cognitive dysfunction may persist for 3 months after surgery in approximately 10% of the patients.

(H) DEVELOPMENT OF CHRONIC PAIN

Following major surgery neuropathic pain can develop due to the nerve fibers damage. Neuropathic pain diagnosed by burning sensation stinging or shooting pain and lack of response to doses of Opioid.

Lastly, optimizing treatment of acute postoperative pain can improve health-related quality of life, while poor postoperative pain control may intervene with patients day to day activities.

MEASUREMENT OF PAIN

Pain Measurement can be done by Two methods:

1. Type I Method
2. Type II Method

1. TYPE I METHOD

Those are objective methods, done by the physician as he assigns numbers about the patient condition. It includes the following:

Physiological Indices

- Endocrinal (increase in serum cortisol and catecholamine).
- Cardiovascular (increase in blood pressure and heart rate)
- Respiratory (increase in respiratory rate and decrease in tidal volume)

Neuro-Pharmacological

- Correlation with Beta Endorphin (decreased in acute painful conditions)
- Thermography (Hypo-emission in Chronic pain)

Neuological

- Nerve conduction Velocity
- Evoked Potentials
- Single Positron Emission Tomography (SPET).

Behavioural

Sighing, Crying, Shouting, Trembling.

(2) TYPE II METHODS

It includes either

Single Dimension Methods

- Category Scale (Verbal Rating Scale)
- Numerical Rating Scale
- Graphic Rating Scale

Multi-Dimensional Methods

- McGill Pain Questionnaire, MPQ
- Dartmouth Pain Questionnaire, DPQ
- West Haven-Yale Pain Questionnaire, WHYPQ

Measurement of pain in clinical practice depends largely on verbal dialogue between the patient and the doctor or nurse. A rating scale is mandatory in research projects and ideally when clinical data are being collected.

Although pain is a subjective experience, great attention has been paid to the quantification of this experience. As pain is subjective experience, everyone has different perceptions of that experience. Differences are found in how individuals quantify pain. For example, some individuals would never say that their pain was a (10) on a scale from (0) to (10). On the other hand, other individuals report their pain as a constant (10) despite looking calm and relaxed. Also, all numeric scales used to measure pain have floor and ceiling effects. If the patients describe their pain to be a (10), there is no way to report an increase in pain intensity.

A number of individual differences between patients make comparisons of pain measurements more difficult. For example, the past experiences of the patients influence their present perception of pain. Also, demographic factors such as gender, age, and ethnic background influence the individuals perception of pain. Again, patients who are clinically depressed and anxious tend to report increased pain intensity.

Of most of the methods of pain scoring VAS and VRS are the most commonly used in the Single Dimension Method.

THE VISUAL ANALOGUE SCALE (VAS)

The visual analogue scale uses a straight line with extremities of pain intensity on either end. This line has 10 divisions (1 cm per division) with one depicting “no pain” and the other end being excruciating unbearable pain. The line can be either vertical or horizontal.

The patients are asked to mark on the line to qualify the intensity of pain that they are currently experiencing. From the end labeled as “no pain” the mark placed by the patient is measured and adjusted to the nearest division.

To assist in describing the intensity of pain, words can be placed along the scale (e.g., mild, moderate, or severe). Such descriptors can help to orient the patient for the degree of pain; this particular variation of the VAS has been known as a graphic rating scale. Explanation to the patient is needed by the clinician when using the VAS. Occasionally, the patient may be confused about the line, perceiving it to represent time of degree of relief rather than degree of pain intensity.

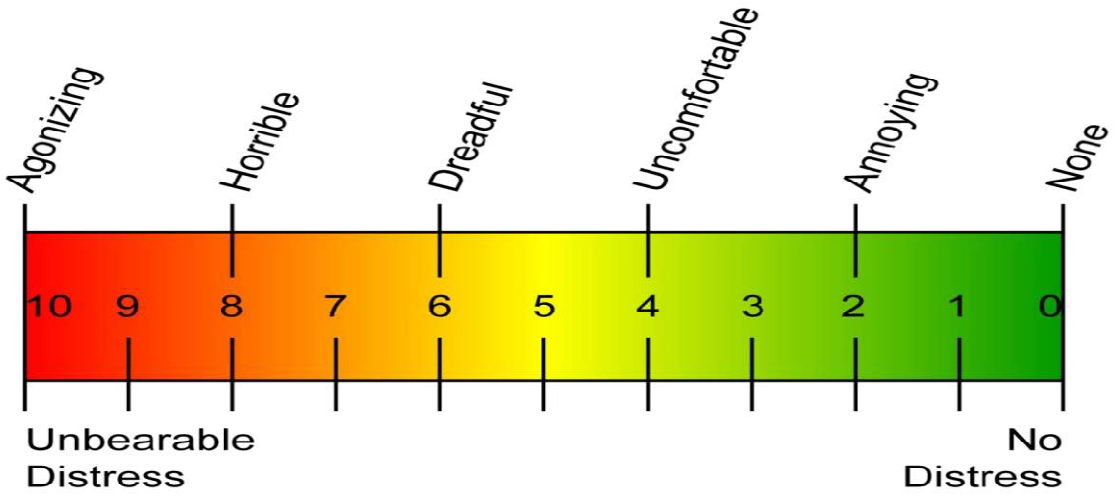


FIGURE: 3:

VISUAL ANALOGUE SCALE

THE VERBAL RATING SCALE

Verbal Rating Scales are another means of assessing the varieties and intensities of pain. A verbal rating scale uses a list of words from which patients choose descriptors of their pain. There are a number of different verbal rating scales including four-item scales, five-item scales, six-item scales, 12-item scales, from the least intense to the most intense. The Prince-Henry pain scale is the most popular scale; it is a 5- point scale, words are often ranked according to severity and numbered sequentially from the scale which quantifies induce pain from 0 to 4 as shown below :

No pain on coughing :

1. Pain on Coughing, but not on deep breathing
2. Pain on Deep breathing, but not at rest
3. Mild Pain at rest
4. Severe Pain at rest

MANAGEMENT OF POST-OPERATIVE PAIN PROPHYLACTIC MEASURES

Postoperative pain can be prophylactically reduced by proper preoperative and postoperative surgical and psychological care. Although the widely accepted definition of pain emphasizes on the cognitive and emotional response to tissue damage, the role of psychological method in the management of acute post operative pain is limited. However studies have shown that Psychological support improves the recovery, reduces the postoperative pain intensity and reduces the psychological distress after any surgical intervention.

Psycho-educational care is described as health-care information provided to patients and patient relatives regarding-

1. Patient preparation for surgery,
2. Timing and conduct of surgical procedures,
3. Function and roles of health-care providers,
4. Self-care actions
5. Pain and discomfort information's
6. Teaching skills like coughing, breathing and bed exercises, relaxation, hypnosis
7. Psychosocial Support (identifying and alleviating concerns, reassurance, problems solving, and encouraging questions).

Apart from pharmacological management good surgical and nursing care also helps to decrease postoperative pain severity. Skillful and smooth surgical handling of tissues in operative site, carry out the operation with dispatch and planning alternative surgical techniques to minimize trauma. Proper post-operative care help to decrease the magnitude of postoperative pain which involves continuing psychological support, proper care of wounds, early ambulation, and of course good nursing care.

ACTIVE MEASURES

Post-operative pain can be partially or completely relieved by one of the following methods :

i. Systemic Analgesics and adjuvant drugs.
ii. Local infiltration and field block.
iii. Regional analgesia using local anaesthetic agents.
iv. Regional analgesia with epidural or intrathecal opioids.
v. Regional analgesia with combined local anaesthetic agents and opioids
vi. Electrical analgesia achieved with transcutaneous electrical stimulation or electroacupuncture.

I. SYSTEMIC ANALGESICS AND ADJUVANT DRUGS

A. NARCOTICS

Exogenously administered opioids mimick the actions of endogenous opioid peptides (Enkephalins, B-Endorphins and Dynorphins) and acts on the specific opioid receptors within the central nervous system and causes pain relief. Pharmacological studies led to the proposal of five classes of opioid receptors. Each receptor mediates a spectrum of pharmacologic effects.

All opiod agents commonly used in clinical practice produce analgesia through similar molecular mechanism. They bind to G-Protein coupled Opioid receptors in the neuronal membrane, subsequently leading to inhibition of adenylatecyclase. This causes activation of inwardly rectifying K-channels, and inhibition of voltage-gated calcium channels thus reducing neuronal excitability and pain impulse transmission.

Whatever the route of administration the cardinal rule is to give the patient sufficient amount of analgesic drug to provide effective sustained pain relief, with minimal side effects. Optimal doses of narcotics given to patients in pain depress the respiratory center slightly; they decrease the ventilation/perfusion abnormality and thus improve oxygenation of

arterial blood, equally important the fact that pain relief permits patients to breath more deeply and to cough somewhat better when they are instructed by nursing and surgical staff.

Although opioids are potent analgesics for control of postoperative pain, concerns regarding their side effects like increase incidence of nausea and vomiting, pruritis, sedation, respiratory depression and potential of inhibiting gastro intestinal motility have limited their use during and after laparoscopic procedures.

ROUTES OF ADMINISTRATION

There is a wide inter-subject and intra-subject variability in the relationships of opioid dose, serum level and pain relief in the management of postoperative pain. Studies have shown that intramuscularly administered narcotics resulted in a wider variability in serum drug concentration than other intravenously administered one.On the other hand , intravenous route provide good and rapid analgesia but produce marked respiratory depression and thus the patient must be observed for 15-20 minutes after first injection to assess pain relief and undesirable side effects.

INTRAVENOUS PATIENT CONTROLLED ANALGESIA

A significant improvement in postoperative analgesia was the development of appropriate delivery system that allows the use of intravenous patient–controlled analgesia (IVPCA). Pumps used allow the patient of inject a small bolus of an intravenous opioid drug whenever he or she feels pain, thus maintaining the analgesic book level in the appropriate range, pumps also has got a “lock-out” system which provides an adequate time delay for the patient to achieve analgesia from each injected dose, and also guards against over dosage that can lead to respiratory depression. Recent machines also provide a continuous infusion of analgesic which give the patient uninterrupted sleep but can lead to an increase in the total quantity of analgesic given. Morphine is the least expensive and perhaps the most popular, but the development of side effects (pruritis, nausea, dysphoria) may require switching to an alternative.

Opioid can be given orally. Sustained release preparations are commercially available. They usually provides quick and effective analgesia and they can also be used to bridge the analgesic gap when there is plan to switch over from patient controlled anesthesia protocol to simple fixed analgesic regimen.

Transdermal opioids (Fentanyl patches) provide excellent alternative, especially when oral route is not allowed. Main advantage of transdermal route is that this technique bypasses the hepatic first-pass metabolism and effective pain relief is achieved for 2-3 days.However the onset of action is slow and unpredictable and option of altering the delivered dose depending upon the analgesic requirement is also not available thus limiting it's use.

PERIPHERAL OPIOID ANALGESIA

Opioid related side effects are mainly related to the receptors present in the central nervous system. Many recent researches have shown that, apart from central nervous system and spinal cord, opioid receptors are also found in peripheral sensory nerves. These receptors are involved in pain modulations which are stimulated by endogenous opioid agonist production by inflammatory leukocytes. Inflammatory cells play a major role in peripheral opioid analgesia by migrating to and delivering opioid peptides to the receptors expressed by the sensory nerve terminal at the very site of tissue damage. Now many research are focused on developing a novel opioid agonist targeting these peripheral opioid receptors.

During surgery the tissue damage leads to inflammation. These inflammations cause leaky capillaries and extravasation of inflammatory cells. Corticotrophin releasing hormone, interleukin-1B and catecholamines play a major role in opioid release from the inflamed tissues. This leads to effective afferent blockade of pain signals and thus reducing the pain perception and sympathetic response to the perceived pain. As this mechanism involves inflammatory cells the action of peripherally applied opioids is apparent only in the presence of ongoing inflammation. Studies have shown that small dose of morphine applied to the injured site i.e incision site, produced significant analgesia with minimal side effects.

PHARMACOLOGICAL PROPERTIES OF NARCOTICS

CNS EFFECTS : Opioids eliminates pain, depresses respiration, suppresses cough, stimulates the third nerve nucleus causing miosis and stimulates the chemoreceptor trigger zone causing nausea and vomiting.

HAEMODYNAMIC EFFECTS : Opioids cause Bradycardia and decrease the sympathetic tone.

SMOOTH MUSCLE EFFECTS : Opioids stimulate circular smooth muscles causing Biliary colic, retention of urine and bronchial constriction which is also partly due to histamine release.

TOLERANCE : Some patients may develop tolerance to particular opiods over a period of time. When this occurs those patients also show tolerance to other opiods also. In this scenario non-opioid analgesics like NSAIDs can be useful for pain control.

B. NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

Non-steroidal anti-inflamatory drugs (NSAID) block the synthesis of prostaglandins by inhibition of the enzyme cyclo-oxygenase. Cyclo-oxygenase enzyme catalyzes the conversion of arachidonic acid to the cyclic endoperoxide, which are the precursors of prostaglandins. As Prostaglandins are mediators of several components of the inflammatory responses like fever, pain and vasodilatation blocking their production will help in control of post operative pain caused by tissue injury and inflammation. Various NSAIDs are availablewith difference in potency with respect to their analgesic, anti-inflammatory and antipyretic properties.

Non-Steroidal Anti-Inflammatory Drugs are commonly used for pain relief in minor surgical procedures. They are also used as bridging drugs in patients who underwent major surgical procedures when they are transferred from opioid based drugs to less potent analgesics over a period of two to three days. NSAIDs have also been used early in the setting of major surgery in combination with opioids. Studies have shown that the quality of pain relief from these combination therapy is better than achieved than opioid alone therapy. Moreover, it has consistently been shown that NSAIDs given soon after major surgery reduce opioid requirements by about onethird.

The three major problems associated with NSAID therapy are

1. Gastropathy,
2. Impaired hemostasis, and
3. Nephrotoxicity.

All are directly related to inhibition of prostaglandin synthesis. NSAIDs can also have idiosyncratic side effects that are not prostaglandin-mediated. Such idiosyncratic reactions are rare but can be serious. These may include exacerbation of bronchospasm, bone marrow toxicity, dermatological reactions, hepatitis and CNS symptoms.

C. PARENTRAL PARACETAMOL

Paracetamol is a one of the non-opioid analgesics commonly used worldwide. It is considered to be the safest and cost-effective non-opioid pain killer when it is administered in analgesic dosages. Studies have shown that both parenteral and rectal paracetomol produces effective pain relief in the postoperative period. Studies have also shown that combined use of paracetamol with a Non-Steroidal Anti-Inflammatory Drugs is superior to paracetomol alone. Although the mechanism of action of paracetamol is not well defined there are increasing evidence of a central antinociceptive effect involving inhibition of COX-2 in central nervous system, inhibition of a putative central cyclooxygenase COX-3and modulation of inhibitory descending serotinergic pathways. Paracetamol inhibits prostaglandin secretion at the cellular transcriptional level, independent of cyclooxygenase activity. Although less potent than standard dose of opioids or the Non-Steroidal Anti-Inflammatory DrugsParacetamolis an effective postoperative analgesic as it has both central and peripheral mechanism of action for control of pain. Currently intravenous compatible preparations are also commercially available and studies have shown that analgesic and anti-inflammatory properties are better than oral and suppository preparations.

D. N-METHYL D–ASPARTATE ANTAGONISTS

Ketamine is an NMDA antagonist and widely used in anesthesia management as anesthetic and analgesic agent. Apart from general anesthesia Ketamine is also used as an adjuvant analgesic agent in regional anesthesia procedures. Studies have shown that use of Ketamine (0.1–0.2 mgkg-1 IV) as an adjunctive to opioids resulted in excellent analgesia, less opioid related side effects, reduced opioid use and greater physician and patient satisfaction. Several studies have described the use of small-dose Ketamine in combined with local anaesthetics or opioid analgesics in management of post operative pain in minor surgical and orthopedic procedures.

Dextromethorphanis another NMDA receptor antagonist commonly used for pain management. One mechanism of pain transmission is wind up phenomenon occurring in dorsal colums of spinal cord mediated by NMDA receptors. As Dextromethorphan blocks NMDA receptors this drug reduces NMDA mediated nociceptive responses in dorsal horn neurons and thus enhance opioid, local anaesthetic and Non-Steroidal Anti-Inflammatory Drugs induced analgesia. Studies have shown that in patients undergoing laparoscopic cholecystectomy and inguinal herniorrhaphy procedures, 90 mg of dextromethorphan given through oral route provided good pain relief,

reduced opioid use, less sedation, less opioid related side effects and caused increased overall well being.

E. ALPHA-2 ADRENERGIC AGONISTS

Clonidine is a centrally acting Alpha Adrenergic Agonist. It is used as oral formulation and also as intravenous formulation for peri operative pain mangement. It is also used as a part of multimodal analgesic regimen along with NSAIDs and opioids. Studies have shown that intravenous Infusion of clonidine in patients undergoing local or regional anesthesia management resulted in stable intraoperative hemodynamics and good post operative pain control.

Dexmedetomidine is a newly introduced selectivealpa 2-agonist. It acts through spinal and supra spinal mechanism and causes reduction in noradrenaline output in pain transmitting fibers. Multiple studies in various type of surgical procedures have shown that use of Dexmedetomidine reduces postoperative pain, maintains sedation and decreases opioid analgesic use and related side effects.

F. MISCELLANEOUS NON-OPIOID COMPOUNDS

Gabapentin initially introduced as an anticonvulsant, now it is being used in acute post operative pain management and also in chronic pain management. Studies have shown that oral administration of Gabapentin (1.2g) as a premedication significantly reduced post-operative pain scores and supplemental analgesic use without increasing adverse effects.

Magnesium, is a divalent cation, and through blocking the calcium mediated pain transmission in spinal cord it provides anti-nociceptive effects. Studies have shown that magnesium 50 mg/kg given intravenously as a bolus dose at induction of anaesthesia resulted in lower pain scores, less opioid requirements and better patient satisfaction in orthopedic procedures. Studies have also shown that magnesium when used intrathecally, It prolongs the fentanyl mediated analgesia.

Neostigmine is a cholinesterase inhibitor that causes increase in acetylcholine levels in the spinal cord. This blocks the ascending pain impulse transmission and also descending pain response thus resulting in pain control. Studies have shown that when used in the dose of 10–200μg in the subarachnoid or epidural spaces better reduction in pain scores are observed. As the mechanism of action is localized to spinal

cord neuraxial administration of neostigmine has been reported to possess better analgesic properties than systemic administration. In patients who underwent knee surgeries epidurally administered neostigmine (1μg/kg) produced more than 5 h of pain relief. Although neuraxial neostigmine acts as good pain killer, 15-30% of patients experience nausea and vomiting. Sedation is also common side effect of neostigmine.

Inositol Triphosphate is a new anti-inflammatory drug, shown to have properties of reducing postoperative pain and reducing the need for opioid analgesics after cholecystectomy.

II. LOCAL INFILTRATION AND FIELD BLOCK

Infiltration of the wounds with dilute solution of Bupivacaine or use of rectus block for abdominal incision has been found effective in partially relieving postoperative pain after laparoscopy. Studies have shown that local anesthesia infiltration before incision offers an advantage of intra-operative pain relief and also supplements post operative analgesia than infiltration after the closure of incision.

REGIONAL ANALGESIA WITH LOCAL ANAESTHETICS

Epidural anaesthesia may be performed at any one of the four segments of the spine (cervical, thoracic, lumbar, and sacral). Sacral epidural anaesthesia is usually referred to as caudal anaesthesia. Thoracic epidural analgesia is technically more difficult and the possibility of injury to the spinal cord is greater.

CONTINUOUS SEGMENTAL EPIDURAL BLOCK

Continuous epidural analgesia is also an attractive option for post-operative pain management. Drug infusion can be intermittent bolus, continuous infusion with commercially available epidural infusion pumps and with patient controlled analgesia regimen. In the technique of patient-

controlled epidural analgesia with a bolus dose adequate pain control is achieved. Then the analgesia is maintained with continuous low dose infusion. And when the patient feels pain bolus drugs will be given by demand injections which are operated by patients but the bolus dose and lock out period controlled by the physician. This technique has the advantage of minimizing drug dosage, flexibility of dosing, better patient satisfaction and reduced demand on physician time. The used pump must be able to give a continuous set infusion rate, to give demand doses with set lockout periods, and to limit a total dose over a set period of time.

REVIEW OF LITERATURE

In a study titled “Perioperative intravenous lidocaine infusion on postoperative pain relief in patients undergoing upper abdominal surgery” The study was done to assess the efficacy of intravenous lidocaine infusion started perioperatively on postoperative pain intensity and analgesic requirement. Sixty patients undergoing major upper abdominal surgery were recruited it was a randomized double blinded study done in patients undergoing upper abdominal surgeries (n=60).

They were divided into two groups, each contains 30 patients. One group received 2% lidocaine (intravenous bolus 1.5 mg/kg followed by an infusion of 1.5 mg/kg/h),and other group received normal saline. Before 30 minutes of skin incision the infusion was started and it was stopped one hour after the end of surgery. The intensity of pain and requirement of analgesia (Diclofenac) were assessed postoperatively for one hour at the interval of 15 minutes then 4th hourly up to 24 hours.

The intensity of pain at rest and movement as well as the total postoperative analgesic requirement were significantly lower (142.50 ± 37.80 mg *vs.*185.00 ± 41.31 mg, $P < 0.001$) in Lidocaine group.

The extubation time was significantly longer in Lidocaine group (14.43 ± 3.50 minutes *vs.* 6.73 ± 1.76 minutes, P < 0.001). The time for the first dose of analgesic requirement was longer in Lidocaine group (60.97 ± 18.05 minutes *vs.* 15.73 ± 7.46 minutes, P < 0.001). It was concluded that perioperative infusion of low dose of Lidocaine decreases postoperative pain intensity, reduces the post-operative requirement of analgesia, without causing much side effects in patients undergoing elective upper abdominal surgeries.

A study titled "The effects of intravenous Lidocaine infusion on hospital stay after major abdominal pediatric surgery". It was a randomized double blinded study which contains eighty pediatric patients aged 1−6 years, ASA II, III posted for abdominal major surgery were randomly divided into two groups, each of forty children. Children in control group received normal saline in a rate of 1.5 ml/kg/h and those in Lignocaine group received Lignocaine 1.5 mg/kg intravenously twenty minutes before induction, then received the infusion of 1.5 mg/kg/hr post-operatively upto 6 hours. They reported the length of hospital-stay and return of bowel function. Plasma Cortisol was recorded at baseline, 10 minutes after continuous infusion, 5 minutes after intubation and 10 minutes after extubation.

Serum lignocaine concentrations were recorded 10 minutes after start of infusion, 10 minutes and 4 hours after extubation. Patients in placebo group showed significant higher plasma cortisol concentrations ($\boldsymbol{P} = 0.001$) in response to induction of anesthesia and extended postoperatively when compared to Lignocaine group.

A Study titled "Perioperative intravenous Lidocaine has preventive effects on postoperative pain and morphine consumption after major abdominal surgery. A randomized prospective study conducted 40 patients undergoing major abdominal surgery participated. Out of 20 patients received intra venous 2% Lidocaine (bolus injection of 1.5 mg/kg in 10 min followed by an IV infusion of 1.5 mg. $kg^{(-1)}$. $h^{(-1)}$), and twenty patients received saline placebo.

The starting time of infusion 30 min before skin incision and was stopped 1 hour at the end of procedure. Ligocaine blood-level measured. Post-operative pain scale (numeric rating scale of 0-10) and morphine consumption (patient-controlled analgesia) were assessed up to 72 hr after surgery. Mean Lidocaine levels during surgery were 1.9 ± 0.7 microg/mL. Patient-controlled analgesia with morphine produced good post-operative analgesia (numeric rating scale at rest, < or = 3; 90% - 95%; no group differences).

Patients who received intra venous Ligocaine group less pain during movement and needed less morphine usage during the first 72 hr after surgery (103.1 ± 72.0 mg *vs.* 159.0 ± 73.3 mg; Student's t-test; $P < 0.05$). Because this opioid - sparing effect is most pronounced on the 3rd post-operative day, IV intra venous Lidocaine may have a true preventive analgesic activity, most likely by preventing the induction of central hyperalgesia in a clinically relevant manner.

A study titled “Effect of Intra-operative Intravenous Lidocaine on Post-operative Pain and Return of Bowel Function After Laparoscopic Abdominal Gynecologic Procedures” A total of fifty patients were included, and five patients were withdrawn from the study, leaving forty five patients (Control group, 21; experimental group, 24) for final analysis. Decreased overall analgesic requirements were noted in the experimental group compared with the control group but failed to achieve statistical significance at any time interval. Pain scores were similar between the groups except on third postoperative measurements, when the overall Visual Analogue Scale score was 3.5 ± 3.1 in the control group compared with 1.6 ± 2.4 in the experimental group ($P = .02$).

A study titled “Intravenous Lidocaine infusion facilitates acute rehabilitation after Laparoscopic Colectomy”. A Randomized prospective

study 40 patients scheduled to undergo laparoscopic colectomy were randomly divided into two groups. Group 1 receive intravenous lidocaine (bolus injection of 1.5 mg/kg Lidocaine at induction of anesthesia, then a continuous infusion of 2 mg.kg.h intra-operatively and 1.33 mg.kg.h for 24 h post-operatively) or an equal volume of saline.

All patients received similar intensive post-operative rehabilitation. Postoperative pain scores, opioid requirement, and fatigue scores were measured. Passing of first flatus, defecation, and hospital discharge were recorded. Postoperative endocrine (cortisol and catecholamines) and metabolic (leukocytes, C-reactive protein, and glucose) responses were measured for 48 hours. Data (presented as median [25-75% interquartile range], lidocaine *vs.* saline groups) were analyzed using Mann-Whitney tests. $P < 0.05$ was considered statistically significant.

A study titled “intravenous Lidocaine speeds the return of bowel function, decreases postoperative pain, and shortens hospital stay in patients undergoing radical Retropubic Prostatectomy”. A randomized study 40 patients undergoing radical retropubic prostatectomy were studied with one half of the patients receiving a Lidocaine bolus (1.5 mg/kg) and infusion (3mg/min, unless weight < 70 kg, then 2 mg/min); the other half received a saline infusion. A blind observer

noted the patient's daily pain score, the time the patient first experienced flatulence and had the first bowel movement, and the total use of analgesics. Lidocaine group patient have experienced first flatulence in a significantly shorter time ($P < 0.01$) than control patients. Lidocaine patients' hospital stay was also significantly shorter ($P < 0.05$); on average, they spent 1.1 fewer days in the hospital. I.V. Lidocaine initiated before anesthesia and continued 1 h postoperatively significantly sped up the return of bowel function. Lidocaine patients were also more comfortable postoperatively. Most of the bowel function benefits attributed to epidural Lidocaine are also present when the drug is administered parenterally. The length of hospital stay also reduced in Lidocaine-group.

A study titled “Intravenous lignocaine infusion forneuropathic pain in cancer patients– a preliminary study” The effectiveness and duration of pain relief with a intravenous Lignocaine infusion was observed in ten cancer patients.

All the ten patients were suffering from pain of neuropathic origin, having two or more of the symptoms: burning, aching, all odynia, reduced sensitivity to touch or pain, hyperaesthesia, nightly exacerbation and sleep disturbance. The patients received intravenous Lignocaine in a

dose of 5 $mgkg^{-1}$ in $1ml.kg^{-1}$ of normal saline over 1 hour. Significant pain relief ('t' value > 't' at 0.01), dysaesthetic sensations, paraesthesias and nightly exacerbations were seen in the majority of patients upto 14 days. Statistical analysis were performed using the unpaired 't' test and analysis of variance (through application of X2 test).

METHODOLOGY

After obtaining necessary approval from Institutional ethical committee, this prospective randomized single blinded case control study was started.

The study was conducted in Institute of Anesthesiology, Govt. Rajaji Hospital, Madurai.

The study subjects were selected from patients posted for Laparoscopic Cholecystectomy.

The inclusion criteria were :

American Society of Anesthesiology (ASA) Grading 1 & 2

Age > 18 years

Both Gender

The exclusion criteria were :

ASA 3, 4

H/o Allergy to Lidocaine

H/o Anti-arrhythmic Drugs

H/o Drug abuse

H/o Chornic Alcoholism

H/o Cardiac Diseases

H/o Bleeding Disorder

H/o Liver Disease

Patient Refusal

After assessing Inclusion & Exclusion criteria 60 patients were selected. Informed consent was obtained from patients who were willing to participate in the study.

On the day of surgery, patients were shifted to operating room. Intravenous access was obtained with 18 gauge Cannula. Electro-cardiogram (ECG), Non-Invasive Blood Pressure (NIBP) and Pulse Oximeter were attached and baseline heart rate, saturation of Hemoglobin (SpO2) and blood pressure were recorded.

The patients were randomized into two groups by closed card method as follows :

i.) **GROUP I** : Patient receive Inj. 2% Lidocaine 1.5mg /Kg IV bolus before induction & intravenous infusion dose of Inj. 2% Lidocaine (1.5mg/Kg) started before skin incision and continued for one hour.

ii.) **GROUP II** : Patient receive 100ml Normal Saline started before skin incision.

Patient were induced with Inj. Fentanyl 2mcs/Kg, Inj. Thiopentone Sodium 5mg/Kg and Inj. Succinyl Choline 2mg/Kg; intubated; controlled ventilation started with $N_2O : O_2$ 3:2

Patients were monitored intra-operatively with ECG, NIBP and SpO_2.

After completion of surgery, the patients were given Inj. Neostigmine and Inj. Glycopyrrolate. After demonstration of recovery, from muscle relaxants patients were extubated and shifted to Post-operative Care Ward.

Post-operative pain-score assessed with VISUAL ANALOGUE SCALE (over 10 points) immediately after extubation and upto 6 hours.

Patients demanding Analgesia or pain score more than 4/10 were given rescue analgesia with Inj. Pentazocine Lactate 15mg through intravenous route. Bowel sounds were assessed every hour and first observed time was noted.

IN OUR STUDY, THE FOLLOWING PARAMETERS WERE OBSERVED :

DEMOGRAPHIC FACTORS

Age (Years)

Gender (Male / Female)

Weight (Kilograms)

Surgery Duration (Minutes)

INTRA OP HEMODYNAMICS

(Assessed in Baseline, 10, 20, 30, 40, 50, 60 min. and post extubation)

Heart Rate (Per Minute)

Mean Blood Pressure (mmHg)

SpO_2 (%)

POST OP ASSESSMENT

Visual Analogue Scale (Post extubation, 30 min, 1, 2, 4 and 6th hour)

Episodes of Rescue Analgesic Use

Dose of rescue analgesic used

Return of bowel sound

STATISTICS

NULL HYPOTHESIS

Lidocaine infusion started before induction and continued for one hour intra-operatively will result in **no significant difference** in intra-op haemodynamics, recovery characteristics, post operative pain and early return of bowel function compared to placebo.

STATISTICAL ANALYSIS

SPSS version 16 (SPSS inc, Chicago 2007) was used for statistical analysis.

Student 't' Test used for comparison of Age, Weight, Duration of surgery, post-op pain scores, episodes of rescue analgesia use, dose of analgesics used and return of bowel sounds (Parametric data)

Chi-Square Test used for comparison of Gender, ASA grading and number of patients received rescue analgesics (non-parametric data)

One-way ANOVA used to assess the changes in hamodynamics during intra operative period.

P < 0.05 considered statistically significant

OBSERVATION AND RESULTS

Thirty patients were included in each group.

The mean age, gender distribution and mean weight were comparable between two groups (p>0.05) (Table 1). (Chart 1,2,3)

CHART 1 :

COMPARISON OF AGE

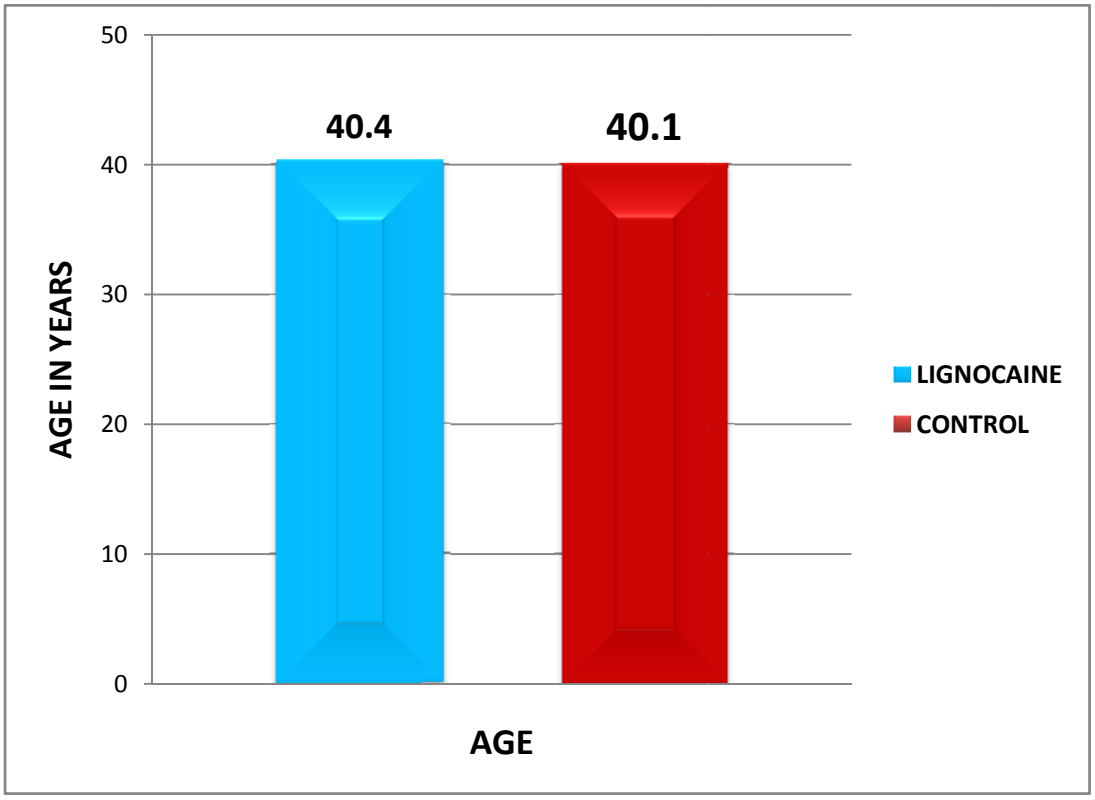


CHART 2 :

COMPARISON OF GENDER DISTRIBUTION

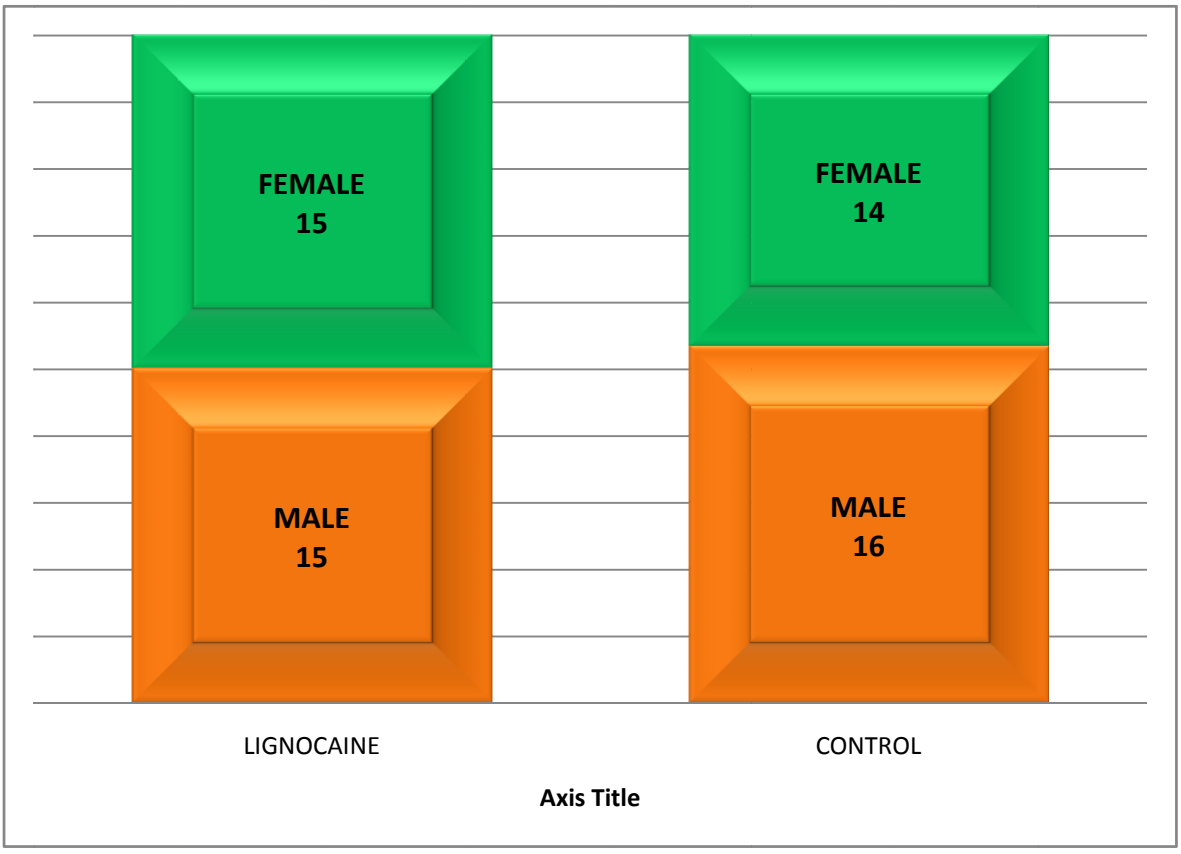


CHART 3:

COMPARISON OF WEIGHT

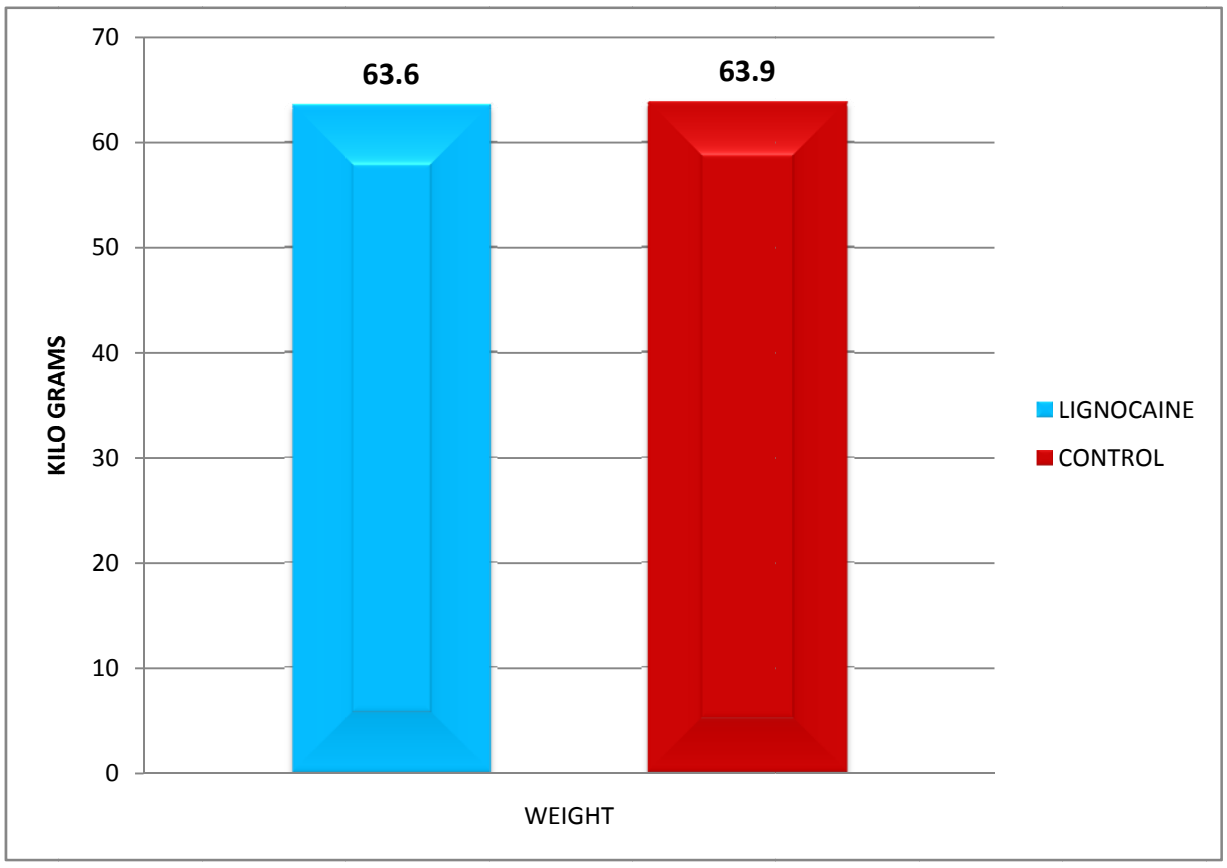


CHART 4 :

COMPARISON OF ASA GRADING

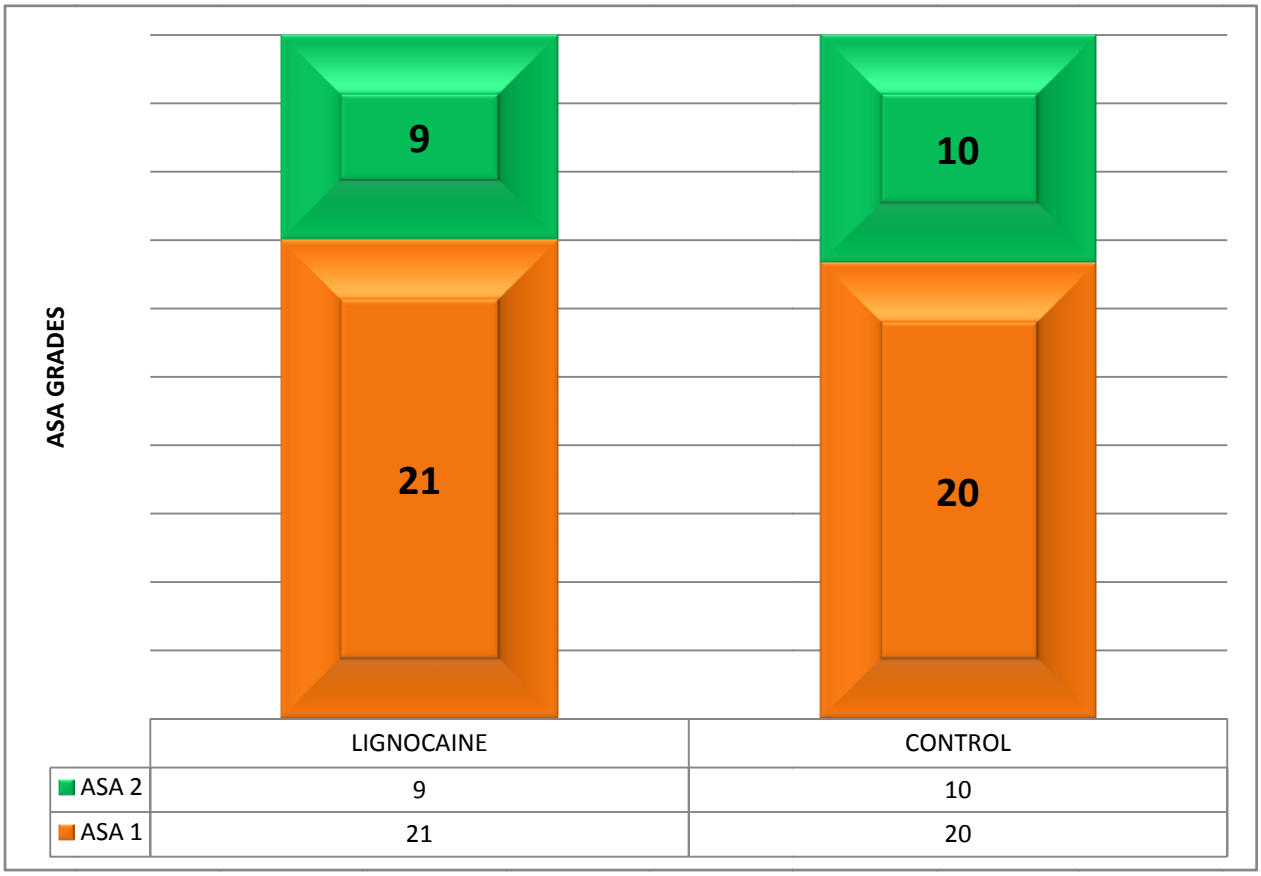


CHART 5 :

COMPARISON OF DURATION OF SURGERY

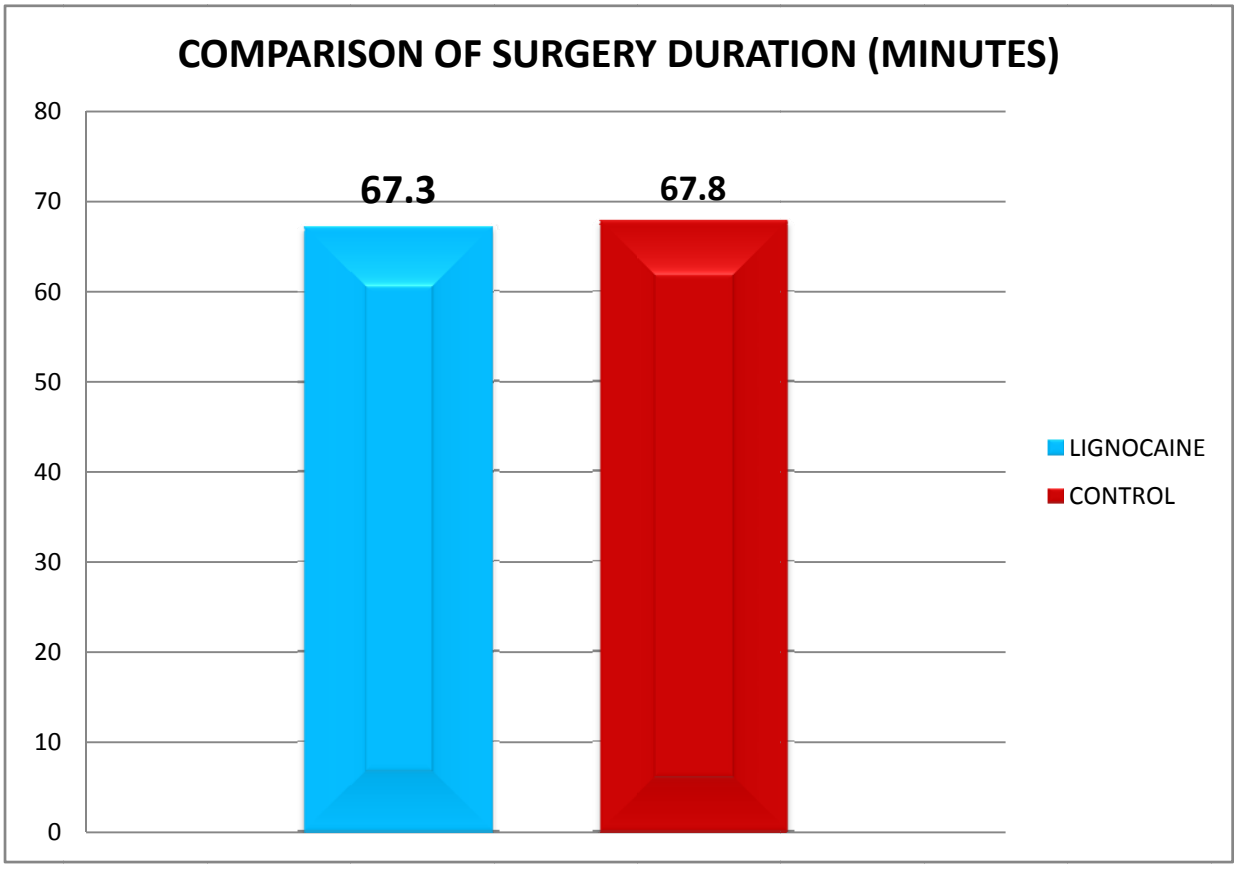
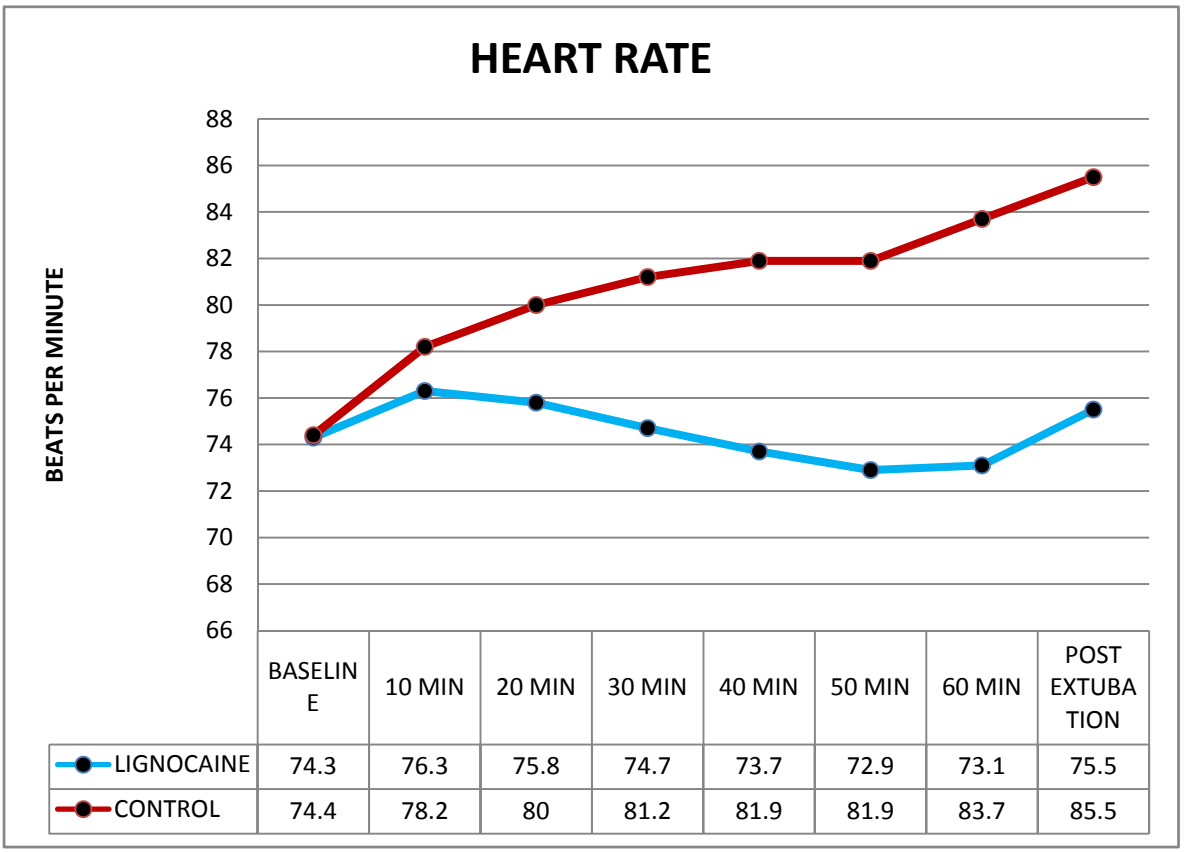


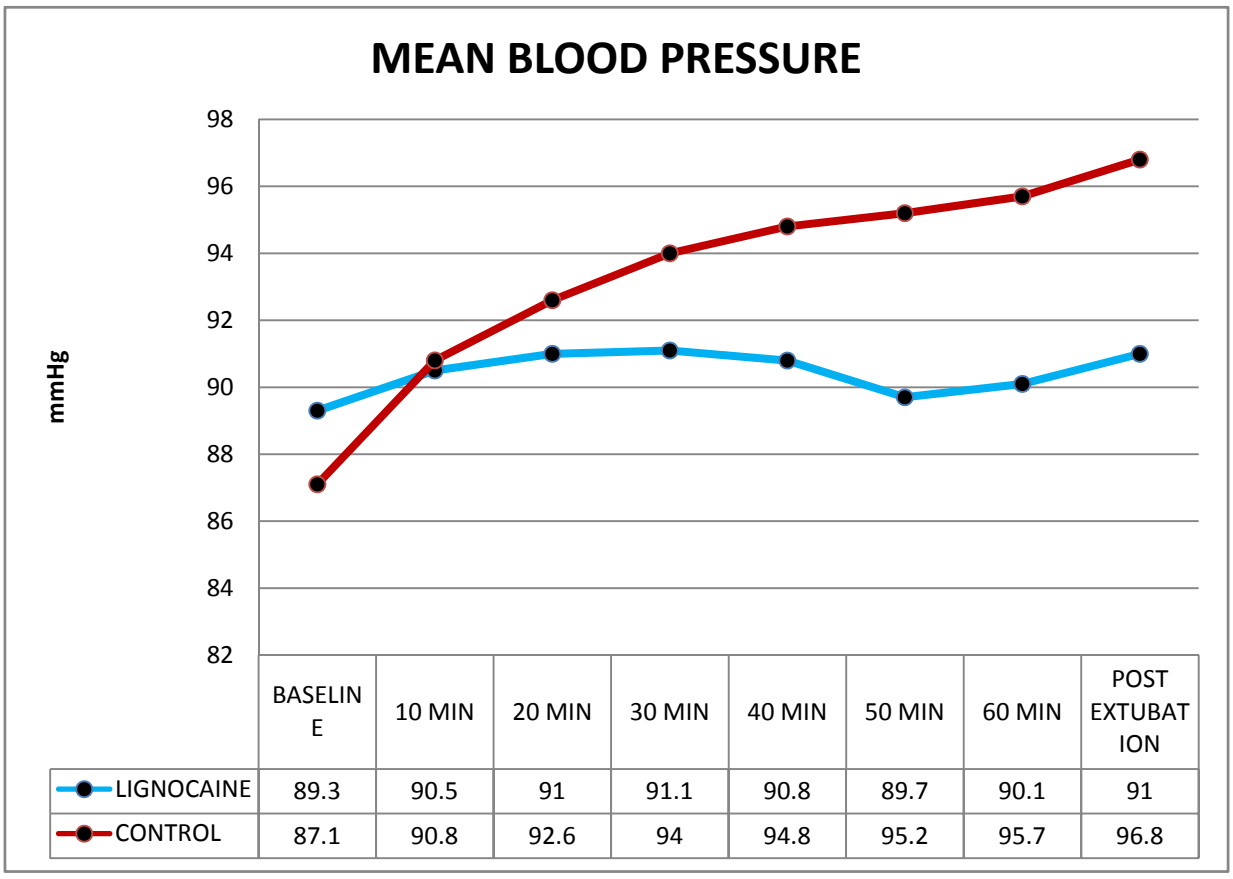
CHART 6:
COMPARISON OF INTRAOPERATIVE HEART RATE



	BASELINE	10 MIN	20 MIN	30 MIN	40 MIN	50 MIN	60 MIN	POST EXTUBATION
LIGNOCAINE	74.3	76.3	75.8	74.7	73.7	72.9	73.1	75.5
CONTROL	74.4	78.2	80	81.2	81.9	81.9	83.7	85.5

CHART 7:

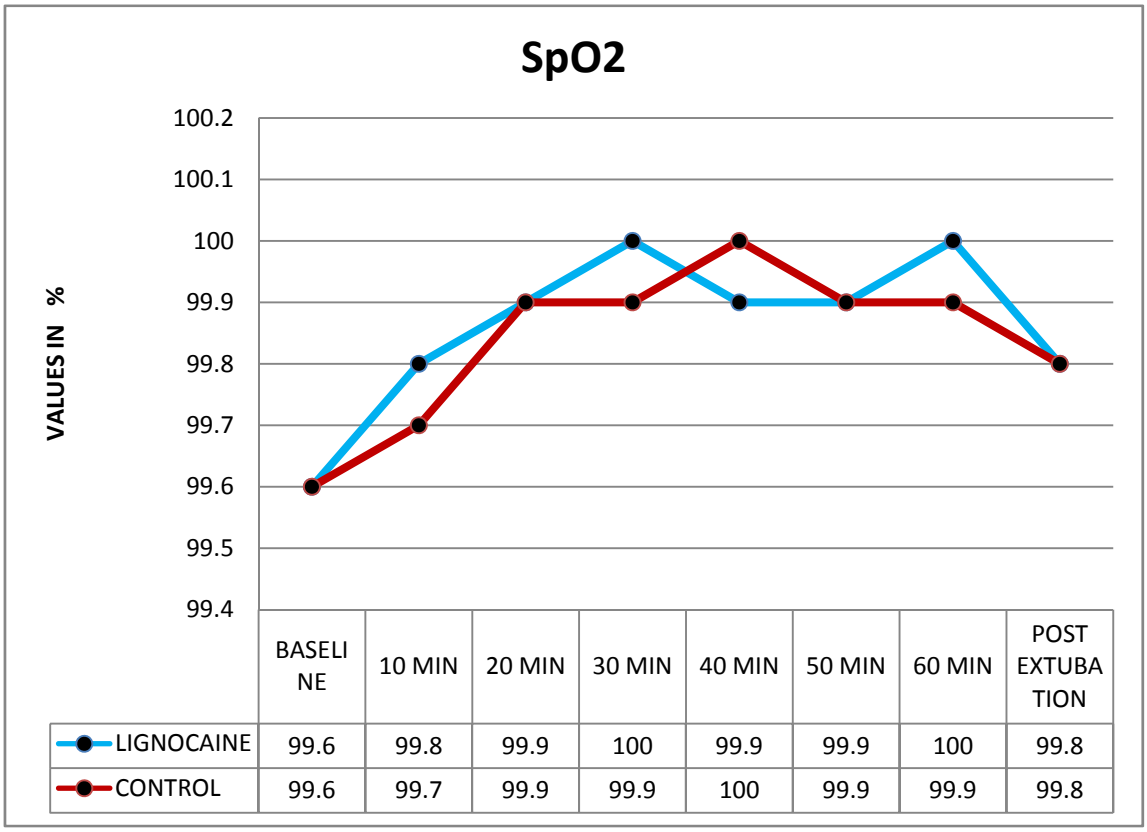
COMPARISON OF INTRAOPERATIVE MEAN BLOOD PRESSURE



	BASELINE	10 MIN	20 MIN	30 MIN	40 MIN	50 MIN	60 MIN	POST EXTUBATION
LIGNOCAINE	89.3	90.5	91	91.1	90.8	89.7	90.1	91
CONTROL	87.1	90.8	92.6	94	94.8	95.2	95.7	96.8

CHART 8:

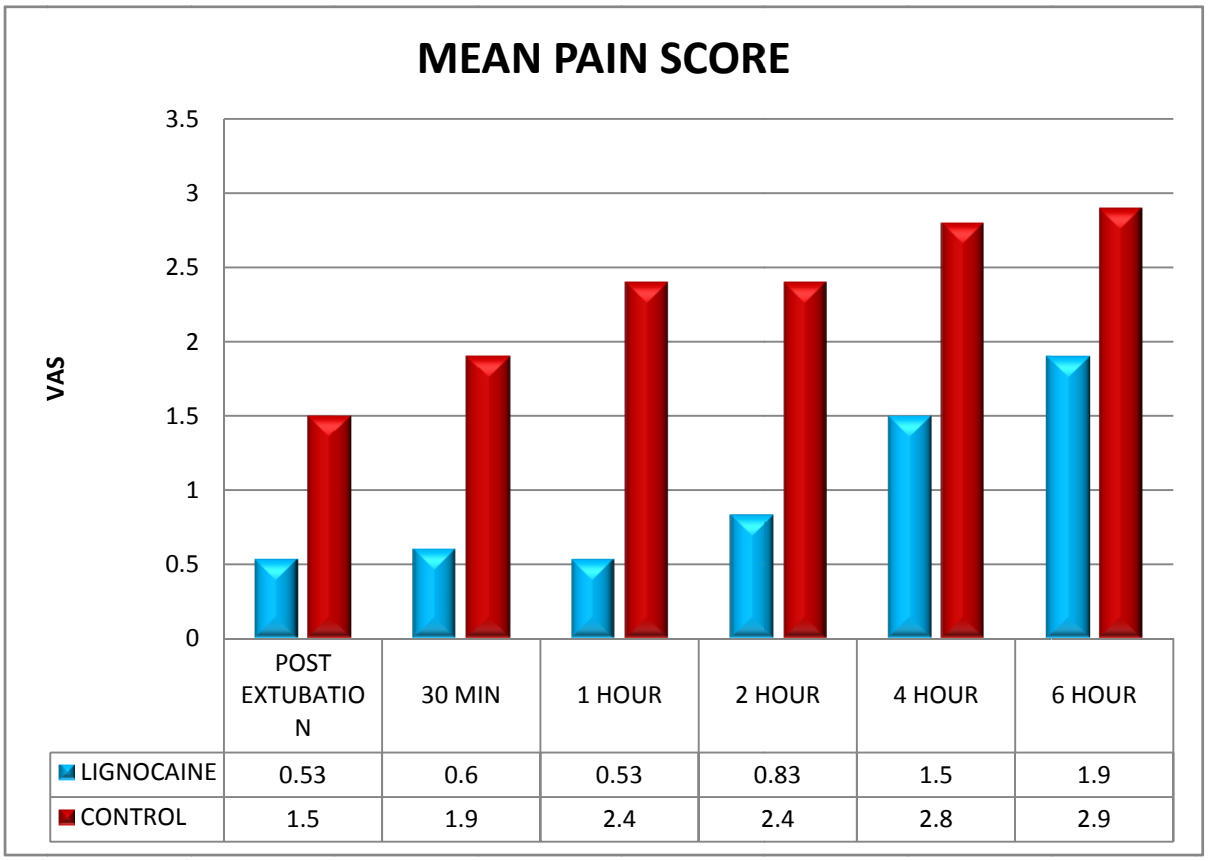
COMPARISON OF INTRAOPERATIVE SPO_2



	BASELINE	10 MIN	20 MIN	30 MIN	40 MIN	50 MIN	60 MIN	POST EXTUBATION
LIGNOCAINE	99.6	99.8	99.9	100	99.9	99.9	100	99.8
CONTROL	99.6	99.7	99.9	99.9	100	99.9	99.9	99.8

CHART 9 :

COMPARISON OF MEAN PAIN SCORE IN POST OPERATIVE PERIOD



	POST EXTUBATION	30 MIN	1 HOUR	2 HOUR	4 HOUR	6 HOUR
LIGNOCAINE	0.53	0.6	0.53	0.83	1.5	1.9
CONTROL	1.5	1.9	2.4	2.4	2.8	2.9

CHART 10:

COMPARISON OF EPISODES OF RESCUE ANALGESIC USE

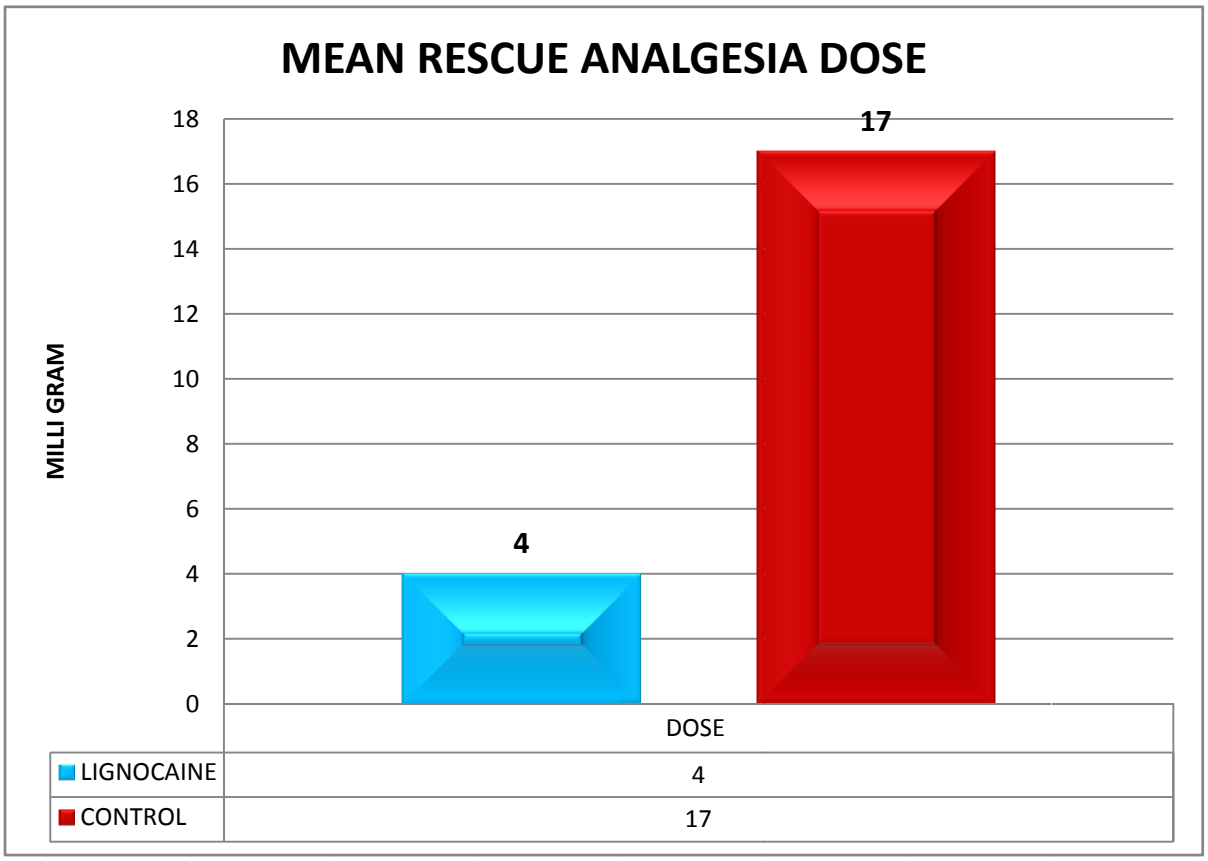


CHART 11:

COMPARISON OF PATIENTS WHO RECEIVED RESCUE ANALGESIA

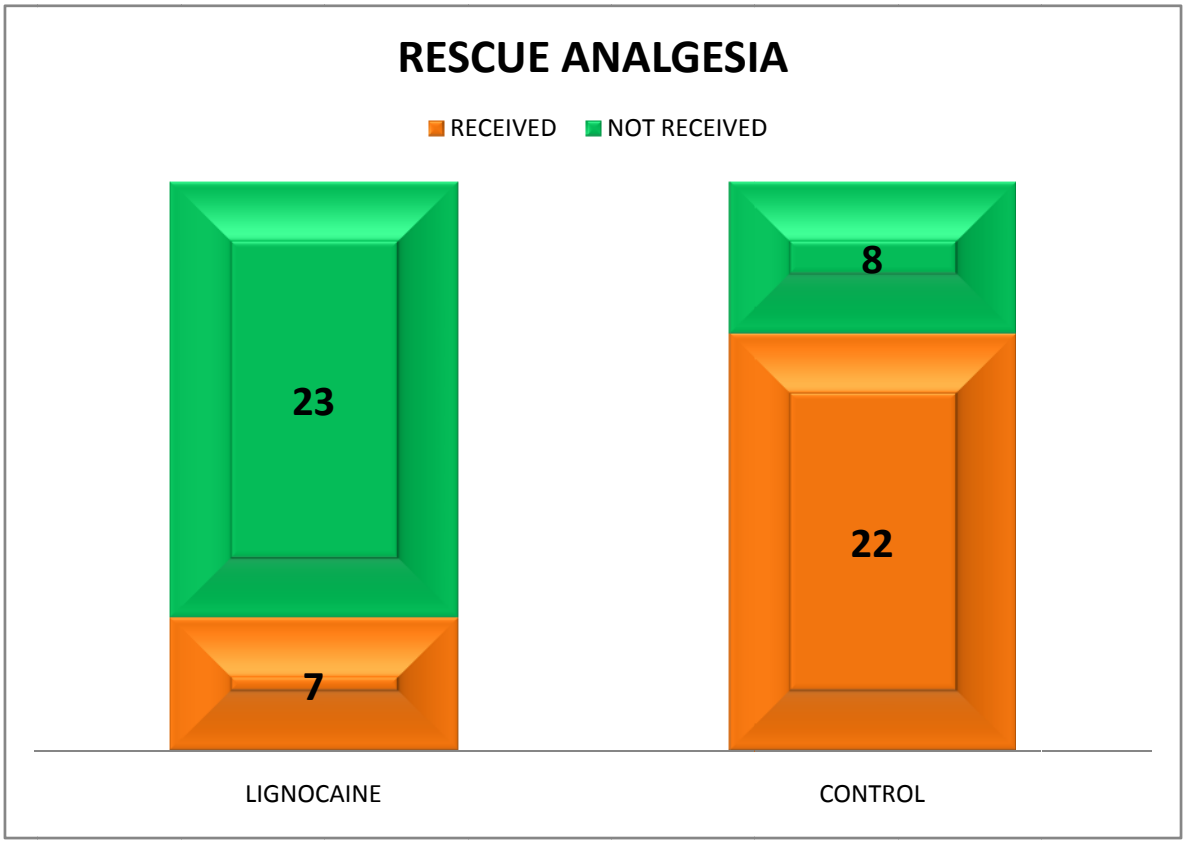
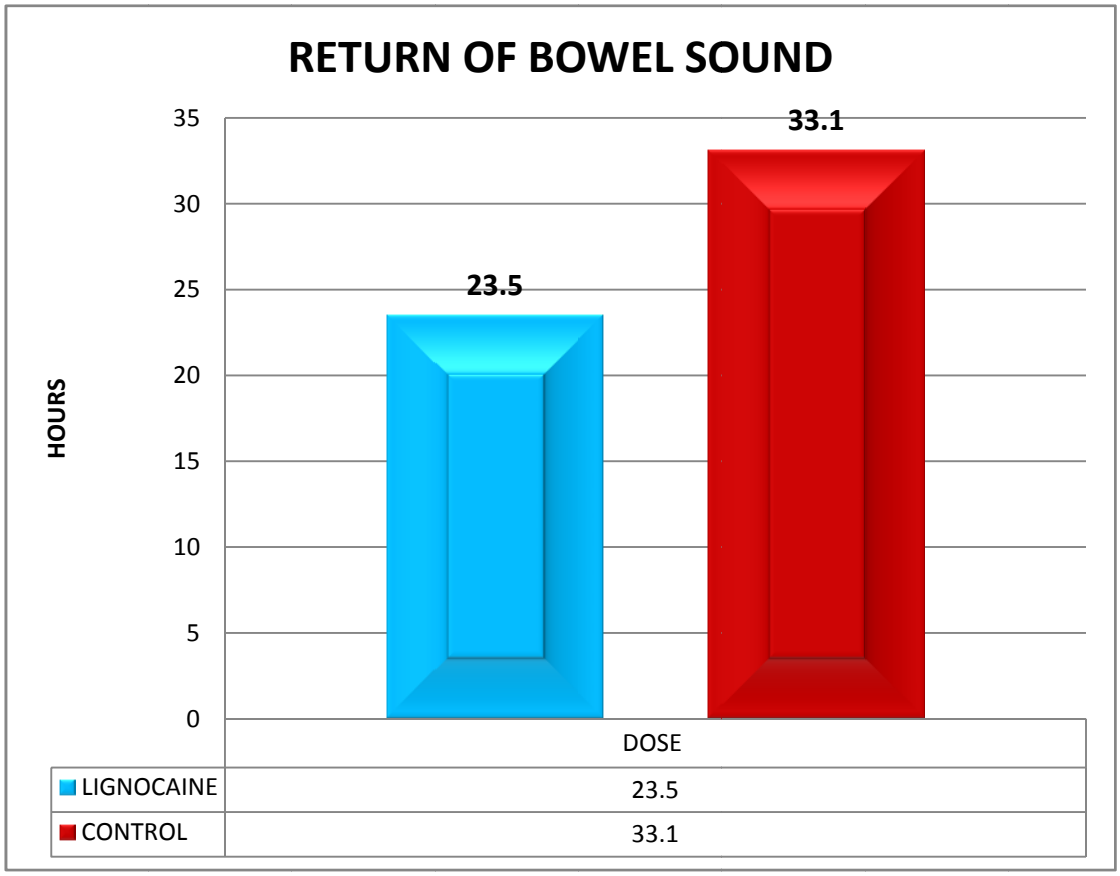


CHART 12:
COMPARISON OF RETURN OF BOWEL MOVEMENTS



The distribution of ASA Grading were comparable between two groups (p-0.781) (Table 1) (Chart 4)

The duration of Surgery were comparable between two groups (p- 0.681) (Table 1) (Chart 5)

On analyzing intra operative hemodynamics, heart rate in Lignocaine group showed no significant difference from baseline (p - 0.402). Similar to heart rate mean blood pressure was also showed no significant difference between baseline (p0.832). This analysis shows that Heart rate and blood pressure were maintained in Lignocaine group (Group I). (Table 2) (Chart 6,7)

On analyzing heart rate and blood pressure changes in control group (Group II) there were statistically significant difference from baseline throughout the surgery ($p < 0.001$) indicating significant alteration in hamodynamics in control group. (Table 2) (Chart 6,7)

Although SpO_2 values in both groups showed statistically significant difference in both group all values were above 99%. (Table 2) (Chart 8)

TABLE 1 :

Intra-operative Hemodynamics

	Base line	**10 min**	**20 min**	**30 min**	**40 min**	**50 min**	**60 min**	**Post extu-batio n**	**p**
Lignocaine Heart Rate	74.3 ± 8.1	76.3 ± 7.0	75.8 ± 6.1	74.7 ± 6.7	73.7 ± 6.8	72.9 ± 6.6	73.1 ± 6.6	75.5 ± 6.2	**0.402**
Control Heart Rate	74.4 ± 7.2	78.2 ± 7.2	80.0 ± 6.4	81.2 ± 6.2	81.9 ± 6.2	81.9 ± 6.4	83.7 ± 6.6	85.5 ± 5.8	**0.000***
Lignocaine mean BP	89.3 ± 5.7	90.5 ± 5.0	91.0 ± 4.3	91.1 ± 4.7	90.8 ± 5.0	89.7 ± 5.5	90.1 ± 5.7	91.0 ± 5.2	**0.832**
Control mean BP	87.1 ± 4.5	90.8 ± 4.2	92.6 ± 4.0	94.0 ± 5.0	94.8 ± 4.1	95.2 ± 4.0	95.7 ± 3.2	96.8 ± 4.2	**0.000***
Lignocaine SpO_2	99.6 ± 0.6	99.8 ± 0.4	99.9 ± 0.2	100	99.9 ± 0.3	99.9 ± 0.1	100	99.8 ± 0.3	**0.000***
Control SpO_2	99.6 ± 0.6	99.7 ± 0.3	99.9 ± 0.2	99.9 ± 0.1	100	99.9 ± 0.2	99.9 ± 0.1	99.8 ± 0.4	**0.000***

Data shown as Mean ± SD

**Denotes Significant 'p' value*

On analyzing the mean pain scores in both groups we found that in patients who received Lignocaine the mean pain scores were low compared to control group in all time points. ($p< 0.001$, $p< 0.001$, $p< 0.001$, $p< 0.001$, p- 0.001, p-0.008). (Table 3) (Chart 9)

TABLE 2 :

Mean Pain Scores in Post-Operative period

	Group I **(i.v Lignocaine)**	**Group II** **(Control)**	**p**
Post extubation	0.53 ± 0.78	1.5 ± 1.0	**0.000***
30 min	0.6 ± 1.03	1.87 ± 1.4	**0.000***
1 hour	0.53 ± 0.68	2.43 ± 1.5	**0.000***
2 hours	0.83 ± 0.87	2.37 ± 1.4	**0.000***
4 hours	1.5 ± 1.5	2.8 ± 1.3	**0.001***
6 hours	1.87 ± 1.6	2.9 ± 1.3	**0.008***

Data shown as Mean ± SD

**Denotes significant 'p' value*

Episodes of rescue analgesia use were also statistically lesser in lignocaine group ($p < 0.001$). Mean analgesic dose was also lesser in patients who received lignocaine ($p < 0.001$) (Table 4)(Chart 10).

Seven patients in group I received rescue analgesia compared to 22 patients in group II and the difference was also statistically significant ($p < 0.001$) (Table 4) (Chart 11)

Mean time of onset of bowel movements was 23.5 hours in Group I and 33.1 hours in Group II and the difference was statistically significant (p-0.009) (chart 12)

TABLE3 :

Rescue Analgesia and Return of Bowel Movements

	Group I (i.v Lignocaine)	**Group II (Control)**	**p**
Episodes of Rescue Analgesia Use	0.27 (0.521)	1.17 (0.913)	0.003*
Dose of Pentazocine (mg)	4 (7.8)	17.5 (13.6)	0.003*
Patins receiving Aescue Rnalgesia (Yes : No)	8 : 22	23 : 7	0.000*
Return of Bowel movements (hours)	23.5 (2.2)	33.1 (3.5)	0.009*

Data shown as Mean (SD)

** Denotes significant 'p' value*

DISCUSSION

In this study, 60 patients were enrolled, divided in to two groups, 30 in each group. The result of this demonstrated that there was no statistically significant difference in age, sex, ASA grading, duration of the surgery and weight of the patients On analyzing intra operative hamodynamics, heart rate in Lignocaine group showed no significant difference from baseline (p-0.402).

Similar to heart rate mean blood pressure was also showed no significant difference between baseline (p0.832). This analysis shows that Heart rate and blood pressure were maintained in Lignocaine group (Group I). On analyzing heart rate and blood pressure changes in control group (GroupII) there were statistically significant difference from baseline throughout the surgery ($p<0.001$) indicating significant alteration in hamodynamics in control group. Although SpO_2 values in both groups showed statistically significant difference in both group all values were above 99%. Post operatively pain score assessed by Visual analogue scale.

On analyzing the mean pain scores in both groups, we found that in patients who received Lignocaine the mean pain scores were low compared to control group in all time points. ($p< 0.001$, $p< 0.001$, $p< 0.001$, $p< 0.001$, $p< 0.001$, p- 0.001, p-0.008). If the patient have VAS more than 4 Pentazocine lactate 15mg. given.

Episodes of rescue analgesia use were also statistically lesser in lignocaine group ($p<0.001$). Mean analgesic dose was also lesser in patients who received lignocaine ($p < 0.001$). Seven patients in Group I received rescue analgesia compared to 22 patients in group II and the difference was also statistically significant ($p < 0.001$). Bowel movements assessed. Mean time of onset of bowel movements was 23.5 hours in Group I and 33.1 hours in Group II and the difference was statistically significant (p-0.009).

SUMMARY

In this study, we evaluate the impact of intravenous Lignocaine on Intraoperative haemodynamics, recovery characteristics, post of pain scores,post op analgesic requirement & Return of bowel function in patients undergoing laproscopic cholecystectomy. Sixty patients of ASA 1&2 of either sex divided into two groups, thirty patients in each group.

Group L – Lidocaine Group

Group S – Saline Group

The study was done at Govt. Rajaji Hospital and Madurai Medical College between the period of 3 years.

In our study, group the Age of the patients, Sex distribution and weight are not statistically significant. There is stable introperative hamodynamics, heart rate, mean blood pressure in lidocaine group. There is lower post operative pain score in lidocaine group. Early bowel movements in lidocaine group.

CONCLUSION

Lidocaine Infusion started (1.5mg/Kg) before induction and continued (1.5mg/Kg/Hr) for one hour intra-operatively results in intra-op hamodynamic stability, lesser post operative pain, lesser use of rescue analgesics and early return of bowel function compared to placebo.

PROFORMA

Name :

Age & Sex :

Weight :

Date & Time of Admission: Date & Time of Discharge:

Diagnosis :

Procedure :

History :

Clinical Examination :

Basic Investigation :

Anaesthetic Technique :

Dosage of Drug :

Post-op. Pain score by VAS

Complication, if any:

Duration of surgery:

Monitoring of Vitals every 15 min

VITALS	**2 min.**	**15 min.**	**30 min.**	**45 min.**	**1 hr**	**1 hr. 15min.**	**1 hr. 30 min.**
PULSE							
B.P							
RR PATERN							
SPO2							
ECG							
SIDE EFFECTS							
REMARKS							

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patient number	name	age	gender	weight	ASA	duration	BHR	10HR	20HR	30HR	40HR	50HR	60HR	peHR	BMBP	10MBP	20MBP	30MBP	40MBP	50MBP	60MBP	peMAP	BSO2	10SO2	20SO2	30SO2	40SO2	50SO2	60SO2	peSO2	peVAS	30VAS	1VAS	2VAS	4VAS	6VAS	Opioid	DOSE	bowel
1	Mahalakshmi	22	M	64	1	60	76	80	82	78	80	78	75	80	80	82	84	86	85	80	78	84	100	100	100	100	100	100	100	100	0	0	0	1	1	2	0	0	22
2	Pandeeswarn	34	F	72	1	65	68	74	76	75	70	68	70	76	85	88	90	90	89	85	88	91	100	100	100	100	100	100	100	100	0	0	0	0	0	1	0	0	24
3	Mohammad Ismail	45	F	56	2	70	80	80	81	76	75	74	76	80	94	96	96	94	90	95	89	90	100	100	100	100	100	100	100	100	1	1	1	1	2	2	0	0	22
4	Dinesh	34	M	58	1	70	82	78	76	80	67	69	80	82	88	90	92	94	98	90	88	86	100	100	100	100	100	100	100	100	0	0	0	1	1	1	0	0	22
5	Dhivya	44	F	71	1	75	78	82	76	80	77	76	75	80	85	88	90	85	88	87	89	90	100	100	100	100	100	100	100	100	0	0	0	0	0	1	0	0	22
6	Mohan	54	M	49	2	65	77	76	74	80	82	80	72	76	92	90	94	92	94	94	90	93	99	99	100	100	100	100	100	99	2	2	2	2	3	4	1	15	23
7	Savitha	34	M	79	1	70	82	80	80	79	84	80	76	80	95	91	96	96	90	90	93	95	100	100	100	100	100	100	100	100	3	5	0	2	5	1	2	30	30
8	Andrews	38	F	58	1	75	90	88	92	86	79	75	88	89	96	91	94	90	94	96	99	98	100	100	100	100	100	100	100	100	2	2	2	3	5	1	0	0	22
9	Sangeetha	53	F	55	1	70	84	89	85	79	82	82	84	88	100	104	94	96	96	100	98	103	100	100	100	100	100	100	100	100	1	1	1	1	3	5	1	15	29
10	Preethi	52	M	66	1	65	70	76	72	77	75	68	71	75	88	90	92	92	95	93	90	91	98	99	99	100	100	100	100	99	1	1	1	1	1	2	0	0	22
11	Chandran	44	F	67	2	60	70	76	74	74	77	68	72	76	85	88	87	88	89	88	90	86	100	100	100	100	100	100	100	100	0	0	0	0	0	0	0	0	22
12	Sivakumar	55	M	72	2	65	69	73	70	69	72	70	69	72	86	88	88	90	87	90	93	90	100	100	100	100	100	100	100	100	0	0	0	1	1	2	0	0	22
13	Diyanesh	67	M	56	2	60	81	76	80	76	75	76	76	77	88	94	86	84	88	89	90	92	100	100	100	100	100	100	100	100	1	1	1	1	1	1	0	0	24
14	Karpagavalli	61	F	50	2	65	75	80	74	78	72	78	76	80	85	90	90	94	88	85	81	89	99	100	100	100	100	100	100	100	0	0	0	0	0	0	0	0	23
15	Vellai	28	F	57	1	65	70	72	70	75	65	69	74	80	86	90	89	84	88	87	91	90	100	100	100	100	100	100	100	100	1	1	1	1	2	2	0	0	23
16	Saroja	27	F	55	1	70	65	66	68	70	68	66	66	75	79	80	84	85	81	82	82	78	100	100	100	100	100	100	100	100	0	0	0	0	0	2	0	0	23
17	Neelagandan	34	M	68	1	65	79	78	74	75	80	75	75	74	80	84	83	85	82	79	80	84	99	100	100	100	100	100	100	100	0	0	1	1	3	5	1	15	28
18	Shanmuganatan	33	M	77	1	70	80	80	83	85	76	76	78	73	95	90	93	99	92	88	90	92	99	99	100	100	100	100	100	100	1	1	1	2	3	3	0	0	22
19	Manikandan	29	F	72	1	75	73	75	77	74	75	78	71	70	94	92	95	99	93	90	94	95	100	100	100	100	100	100	100	100	0	0	0	0	0	1	0	0	22
20	Venkatesh	45	F	74	1	75	85	88	75	85	80	85	82	80	89	90	92	94	90	90	88	90	100	100	100	100	100	100	100	100	1	1	1	1	1	1	0	0	24
21	Muthukaruppan	41	F	55	1	70	90	92	87	86	85	90	84	80	99	100	97	98	103	100	97	98	100	100	100	100	100	100	100	100	0	0	0	0	0	0	0	0	25
22	David	44	F	70	1	65	58	64	79	64	59	64	63	65	98	101	100	96	99	98	104	99	99	99	100	100	100	99	100	99	0	0	0	0	0	0	0	0	22
23	Begam	32	M	80	2	65	66	68	68	63	65	68	64	68	93	90	91	88	94	94	90	94	100	100	100	100	100	100	100	100	0	0	1	1	2	4	1	15	24
24	Sampath	33	M	68	1	70	70	73	74	71	74	74	68	70	89	92	97	94	90	91	92	94	100	100	100	100	100	100	100	100	0	0	0	1	3	5	1	15	26
25	Sharmila	34	M	67	1	60	70	73	77	68	73	68	65	66	91	90	88	88	92	95	93	93	98	99	100	100	100	100	100	99	1	1	1	1	2	2	0	0	22
26	Kannan	50	M	59	1	65	58	62	65	59	57	64	60	64	86	90	92	95	90	88	85	86	100	100	100	100	100	100	100	100	0	0	0	0	1	1	0	0	22
27	Muthulakshmi	47	F	60	2	65	68	70	68	70	67	61	64	68	81	84	84	83	81	78	82	83	100	100	100	100	100	100	100	100	0	0	0	0	0	0	0	0	22
28	Maharajan	51	M	60	2	70	70	72	72	69	71	68	72	70	89	92	94	91	89	90	92	92	99	100	99	100	99	100	100	100	0	0	0	0	1	1	0	0	23
29	Renuka	45	F	58	1	70	74	74	72	70	74	70	70	73	90	92	90	90	94	89	92	93	100	100	100	100	100	100	100	100	1	1	2	3	4	5	1	15	26
30	Karuppaiah	58	M	56	1	65	73	75	74	72	76	70	76	78	94	90	88	93	95	90	94	92	99	100	100	100	99	100	100	99	0	0	0	0	0	1	0	0	22

Patient Number	name	Age	Sex	Wt	ASA	duration	BHR	10HR	20HR	30HR	40HR	50HR	60HR	peHR	BMBP	10MBP	20MBP	30MBP	40MBP	50MBP	60MBP	peMAP	BSO2	10SO2	20SO2	30SO2	40SO2	50SO2	60SO2	peSO2	peVAS	30VAS	1VAS	2VAS	4VAS	6VAS	Opioid	DOSE
1	Murugan	26	F	66	1	65	77	80	86	90	94	90	96	96	81	82	84	86	90	95	93	98	100	100	100	100	100	100	100	100	0	0	0	1	1	2	0	0
2	Selvam	45	M	70	1	75	70	74	76	78	80	82	83	84	82	88	90	93	95	95	99	101	100	100	100	100	100	100	100	100	2	3	3	5	1	1	1	15
3	Idumban	48	M	56	2	70	81	85	85	88	85	88	88	92	94	96	96	100	98	99	101	101	100	100	100	100	100	100	100	100	1	1	1	1	2	2	0	0
4	Paramasivam	56	F	60	1	70	80	88	87	90	90	92	90	94	88	90	92	94	98	99	98	100	100	100	100	100	100	100	100	100	2	2	5	1	1	1	1	15
5	Ranjini	51	F	71	1	70	79	82	86	88	88	90	88	92	85	88	90	95	93	97	98	97	100	100	100	100	100	100	100	100	1	2	2	2	3	4	1	15
6	Vennila	23	M	68	2	80	76	86	84	82	84	84	88	88	86	90	94	92	98	98	97	96	99	100	100	100	100	100	100	99	2	2	2	2	3	3	0	0
7	Jayakumar	38	M	59	1	70	83	88	85	83	84	80	86	88	85	91	96	95	91	90	93	95	100	100	100	100	100	100	100	100	3	5	0	0	0	1	1	15
8	Arun	32	F	58	1	75	88	88	92	96	89	88	90	92	86	91	94	95	94	96	99	98	99	99	100	100	100	100	100	100	2	2	2	3	5	1	1	15
9	Sathyan	60	F	60	2	70	85	89	85	89	88	90	94	92	93	98	94	98	96	100	98	103	100	100	100	100	100	100	100	100	2	4	1	1	3	5	2	30
10	Bharathi	40	M	66	1	70	73	76	76	77	80	82	80	86	88	90	92	92	95	98	94	90	98	99	99	100	100	100	100	99	1	1	2	2	3	3	0	0
11	Vijayakumar	49	F	65	1	60	71	76	84	84	87	82	82	86	82	88	87	88	89	92	90	84	100	100	100	100	100	100	100	100	2	5	1	2	3	5	2	30
12	Paichiammal	52	M	72	2	65	73	73	78	80	82	78	84	84	84	90	88	91	90	90	93	94	100	100	100	100	100	100	100	100	1	1	1	1	2	2	0	0
13	Vettai	46	M	60	2	60	82	86	90	88	90	88	90	92	88	94	86	84	88	89	95	92	100	100	100	100	100	100	100	100	1	1	2	2	4	3	1	15
14	Prabhu	54	F	50	2	70	70	80	84	88	84	88	86	86	88	90	90	94	98	96	96	95	100	100	100	100	100	100	100	100	0	2	4	2	5	3	2	30
15	Dhanam	27	M	61	1	65	71	72	76	80	82	83	84	88	86	90	93	93	92	95	91	94	100	100	100	100	100	100	100	100	1	1	2	2	4	2	1	15
16	Jagadish	60	F	55	1	70	66	68	70	70	72	74	76	80	85	86	88	88	93	95	95	94	100	100	100	100	100	99	100	99	0	0	1	1	3	5	1	15
17	Vembu	39	M	68	2	65	68	78	74	76	80	82	88	86	82	84	88	88	92	95	93	94	99	100	100	100	100	100	100	100	2	2	3	3	5	3	1	15
18	Karuppaee	26	M	75	1	70	78	80	83	85	86	88	88	90	95	96	100	101	101	98	99	103	99	99	100	100	100	100	100	100	1	1	2	2	3	3	0	0
19	Karmegam	43	F	72	1	65	74	75	77	78	80	80	82	82	84	92	95	99	96	90	94	95	100	100	100	100	100	100	100	100	3	4	5	1	2	2	2	30
20	Stella	38	F	71	1	75	83	88	85	85	86	85	88	88	89	90	92	94	95	93	98	100	100	100	100	100	100	100	100	100	1	2	2	2	2	3	0	0
21	Venkatesh Babu	54	F	55	2	70	88	92	87	88	88	90	94	90	99	100	97	98	103	100	97	103	100	100	100	100	100	100	100	100	2	3	5	5	2	2	3	45
22	Muthukumar	26	F	70	1	60	70	74	79	74	79	75	80	83	98	101	100	110	103	105	104	104	99	100	100	100	100	99	100	99	4	0	2	2	4	3	2	30
23	Hafeena	31	M	76	1	65	67	68	70	73	75	78	80	80	88	90	95	95	94	94	98	99	100	100	100	100	100	100	100	100	0	0	1	1	3	3	0	0
24	Muthupandi	38	F	68	1	70	65	73	74	75	75	74	78	80	85	92	97	94	95	91	92	98	100	100	100	100	100	100	100	100	0	1	2	5	3	5	2	30
25	Esther	33	M	66	1	60	71	73	77	78	73	78	75	76	86	90	94	98	98	95	97	98	98	99	100	100	100	100	100	99	1	2	5	5	2	2	2	30

Ref.No.6506/E1/5/2014

Madurai Medical College,
Maduai -20. Dated: 31.07.2014.

Institutional Review Board/Independent Ethics Committee
Capt.Dr.B.Santhakumar,MD (FM). deanmdu@gmail.com
Dean, Madurai Medical College &
Government Rajaji Hospital, Madurai 625 020 . Convenor

Sub: Establishment – Madurai Medical College, Madurai-20 –
Ethics Committee Meeting – Meeting Minutes - for July 2014 –
Approved list – reg.

The Ethics Committee meeting of the Madurai Medical College, Madurai was held on 22nd July 2014 at 10.00 Am to 12.00 Noon at Anaesthesia Seminar Hall at Govt. Rajaji Hospital, Madurai . The following members of the Ethics Committee have attended the meeting.

1.Dr.V.Nagarajan,M.D.,D.M(Neuro) Ph: 0452-2629629 Cell No.9843052029 nag9999@gmail.com.	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2.Dr.Mohan Prasad, MS.M.Ch. Cell.No.9843050822 (Oncology) drbkcmp@gmail.com	Professor & H.O.D of Surgical Oncology (Retired) D.No.32, West Avani Moola Street, Madurai.-1	Member Secretary
3. Dr.L.Santhanalakshmi, MD (Physiology) Cell No.9842593412 dr.l.santhanalakshmi@gmail.com.	Vice Principal, Prof. & H.O.D. Institute of Physiology Madurai Medical College	Member
4.Dr.K.Parameswari, MD(Pharmacology) Cell No.9994026056 drparameswari@yahoo.com.	Director of Pharmacology Madurai Medical College.	Member
5.Dr.S.Vadivel Murugan, MD., (Gen.Medicine) Cell No.9566543048 svadivelmurugan_2007@rediffmail.com.	Professor & H.O.D of Medicine Madurai Medical College	Member
6.Dr.A.Sankaramahalingam, MS., (Gen. Surgery) Cell.No.9443367312 chandrahospitalmdu@gmail.com	Professor & H.O.D. Surgery Madurai Medical College.	Member
7.Mrs.Mercy Immaculate Rubalatha, M.A., Med., Cell.No.9367792650 lathadevadoss86@gmail.com	50/5, Corporation Officer's Quarters, Gandhi Museum Road, Thamukam, Madurai-20.	Member
8.Thiru.Pala.Ramasamy, B.A.,B.L., Cell.No.9842165127 palaramasamy2011@gmail.com	Advocate, D.No.72,Palam Station Road, Sellur, Madurai-20.	Member
9.Thiru.P.K.M.Chelliah, B.A., Cell No.9894349599 pkmandco@gmail.com	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20.	Member

The following project was approved by the committee

Name of the PG Student	Course	Name of the Project	Remarks
Dr.P.Ravi drravi82@yahoo.com	PG in M.D (Anaesthesia) Madurai Medical College, Madurai	To study the impact of intravenous lidocaine on intraoperative haemodynamics, recovery characteristics, post OP pain scores, post OP Analgesic requirement and return of bowel function.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance.
   She/He should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary
Ethical Committee

Chairman
Ethical committee

31.7.18
DEAN/Convenor
Madurai Medical College & Govt.
Rajaji Hospital, Madurai.

To
The above Applicant
-thro. Head of the Department concerned

Dr. Ravi

DIRECTOR
INSTITUTE OF ANAESTHESIOLOGY
Madurai Medical College &
Govt. Rajaji Hospital
Madurai-625 020

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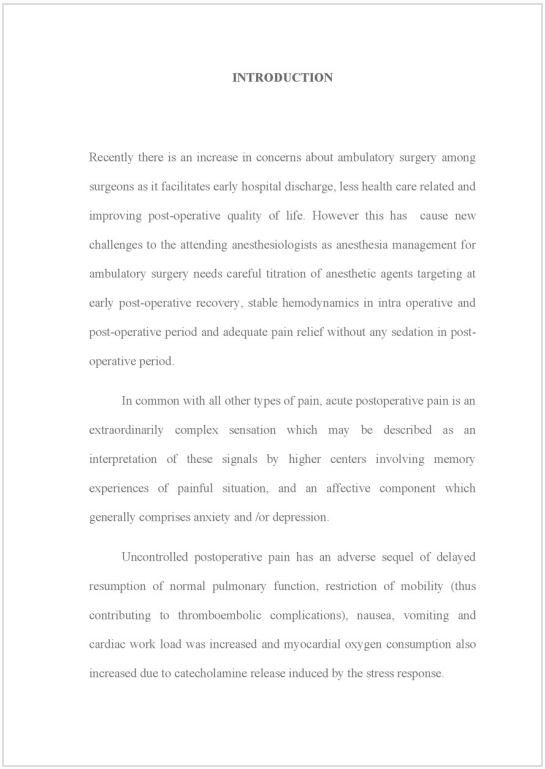
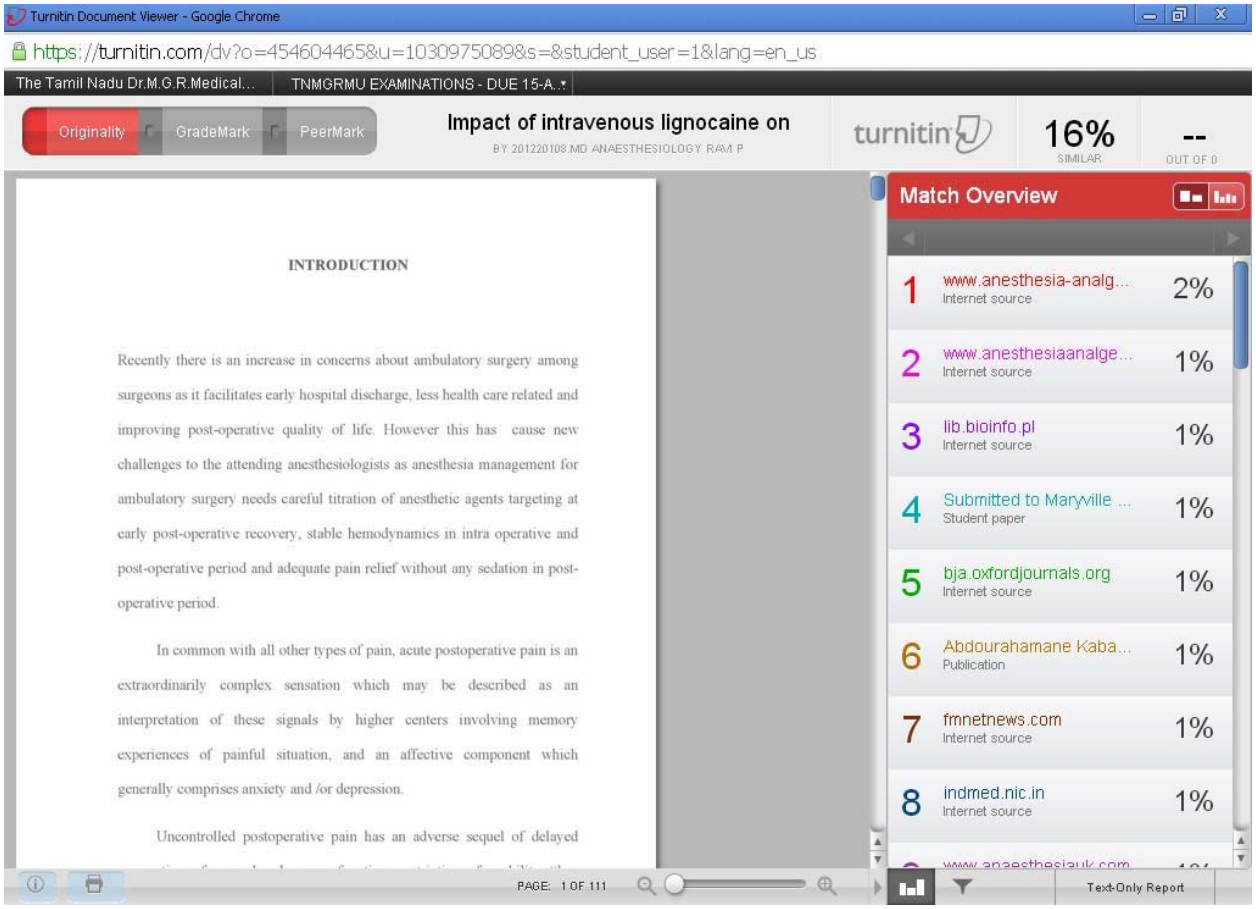


INTRODUCTION

Recently there is an increase in concerns about ambulatory surgery among surgeons as it facilitates early hospital discharge, less health care related and improving post-operative quality of life. However this has cause new challenges to the attending anesthesiologists as anesthesia management for ambulatory surgery needs careful titration of anesthetic agents targeting at early post-operative recovery, stable hemodynamics in intra operative and post-operative period and adequate pain relief without any sedation in post-operative period.

In common with all other types of pain, acute postoperative pain is an extraordinarily complex sensation which may be described as an interpretation of these signals by higher centers involving memory experiences of painful situation, and an affective component which generally comprises anxiety and /or depression.

Uncontrolled postoperative pain has an adverse sequel of delayed resumption of normal pulmonary function, restriction of mobility (thus contributing to thromboembolic complications), nausea, vomiting and cardiac work load was increased and myocardial oxygen consumption also increased due to catecholamine release induced by the stress response.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