# "COMPARISON OF LOSS OF RESISTANCE TO AIR TECHNIQUE AND SALINE INFUSION TECHNIQUE USING MICRO - DRIP SET FOR IDENTIFICATION OF EPIDURAL SPACE - A ONE YEAR RANDOMIZED STUDY"

## DISSERTATION

**BY**

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**ANAESTHESIOLOGY**

**UNDER THE GUIDANCE OF**

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**Dr. Akshaya N. Shetti.**

# ABBREVIATIONS

| | |
|---|---|
| ASA | American society of Anaesthesiologists |
| µg | Micrograms |
| Adr | Adrenaline |
| BP | Blood pressure |
| cm | Centimeters |
| CNS | Central nervous system |
| CSF | Cerebrospinal fluid |
| CVS | Cardio vascular system |
| ES | Epidural space |
| HR | Heart rate |
| Hr | Hour |
| Kg | Kilograms |
| LOR | Loss Of Resistance |
| mg | Milligrams |
| min | Minute |
| PR | Pulse rate |
| RR | Respiratory rate |
| RS | Respiratory System |
| USG | Ultrasonography |

# ABSTRACT

**Objective:** Epidural anaesthesia is most commonly performed technique in routine anaesthesia practice. The technique of identification of epidural space is very much important step, as it determines the success rate and quality of analgesia. Complications are associated with incorrect identification of epidural space. Different techniques have been suggested for identification of epidural space. This study compares the success rate and quality of analgesia by using two different techniques i.e. loss of resistance with air and saline infusion technique using micro-drip infusion set for identification of epidural space in patients undergoing lower abdominal and lower limb surgeries.

**Study design:** A randomized controlled trial.

**Methods:** Two hundred and twenty patients posted for lower limb or lower abdominal surgeries were randomly allocated into two groups of hundred and ten in each. Tuohy needle was used for identification of epidural space. We identified epidural space, in Group A by loss of resistance technique using air as a medium and in Group S by saline infusion technique using micro-drip infusion set. The success rate and quality of analgesia were noted. Identification of epidural space considered successful when no dripping of CSF, no increase in the heart rate on administration of epidural test dose, adequate sensory ( loss of sensation to pin prick with 22G hypodermic needle in the anterior axillary line) and motor blockade (Bromage scale>3) were noticed. The level of significance was taken as 0.05.

**Results:** The success rate of identification of epidural space in air group is 98.18% where as for saline group it is 100%. The good quality of analgesia in saline group was found to be 100% when compared to 96.36% in air group. There is no statistically significant difference between

two groups with the P values 0.498 and 0.122 for success rate and good quality of analgesia respectively.

**Conclusion:** There is no statistically difference between two groups for success rate and good quality of analgesia. The choice of technique for identification of epidural space largely depends up on personal preference of anaesthesia provider. The potential complications observed with loss of resistance technique using air as a medium are absent with saline infusion technique. Hence it is considered to be safe, simple and reliable objective technique.

**Key words:** Epidural anaesthesia, loss of resistance technique with air, saline infusion technique using micro-drip infusion set.

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# INTRODUCTION

Spinal and epidural anaesthesia are most commonly practised central neuraxial blockade. Although the caudal anaesthesia is a variant of epidural anaesthesia it has got limited applications. Regional anaesthesia has an important place in the modern anaesthetic practice as it provides safe and efficient anaesthesia. Neuraxial anaesthesia has had a great impact on all surgical fields. These actually expand the anaesthesiologist's armamentarium, providing alternatives to general anaesthesia when appropriate.

Both spinal and epidural anaesthesia have got their own advantages and disadvantages. Ideally, an anaesthetic technique should not involve undue hypotension, hypertension, bradycardia and tachycardia.

Unfortunately spinal anaesthesia is often associated with hypotension and bradycardia which may be rapid in onset and sometimes profound. To treat such adverse events rapid administration of large amount of intravenous fluids, anticholinergics or inotropic support are required.

Earlier epidural anaesthesia was considered a technique only for an enthusiastic anaesthesiologist. The epidural anaesthesia later on became popular as it has got advantage of slower onset of hypotension and Bradycardia. In this technique the anaesthesiologist gets enough time to correct hemodynamic changes. It has got several advantages like, ability to achieve segmental block, scope for postoperative analgesia even for neck and thoracic surgeries and ability to differentiate acute and chronic pain.

The added advantage is ability to maintain continuous anaesthesia with the placement of an epidural catheter.

The ability of epidural anaesthesia to produce neuraxial anaesthesia and analgesia without dural puncture provides an attractive option to spinal anaesthesia. With respect to risk of infection and potential neurotoxicity of anaesthetic and analgesic drug, the epidural space is far more forgiving than the subarachnoid space.

With these advantages of epidural anaesthesia, spinal anaesthesia fell out of fashion. Improvement in equipment, drugs and technique have made it still popular and versatile anaesthetic technique. Epidural anaesthesia has now gained such popularity that it has now become an essential part of the training of all anaesthesiologists.

The acceptance of epidural blockade is very much limited by the fear of failure, fear of neurological complications, slow onset and shorter duration of action. A large part of the success of epidural anaesthesia rests on correct identification of the epidural space. Since a hundred years numerous techniques for locating the epidural space have been demonstrated. Some of them are Odoms's indicator,[1] Macintosh's balloon,[2] loss of resistance to air or saline, combination of both air and saline, Baraka's running saline infusion technique,[3] hanging-drop technique,[4] compuflo technique,[5,6] loss of resistance using Lignocaine, combination of Lignocaine and air technique,[7] etc..

Out of all these, the manual loss of resistance technique is widely used by anaesthesiologists. This technique is easy to perform, cost effective and has got high success rate. In this technique either air or saline is used as a medium. This technique is

not free from disadvantages. Use of air to locate epidural space increases the incidence of unblocked segments, [8] emphysema of the neck, [9, 10] shoulder pain, [11] and venous air embolism [12] and persistent neurological deficits. [13] In contrast the use of saline is reported to slow the onset and reduce the quality of epidural analgesia [7]. This is mainly seen if larger amount of saline is used.

We should adapt such a technique that is be easy to perform and cost effective, with higher success in identifying epidural space, does not affect the quality of analgesia and has no complications.

Out of all above mentioned techniques, Baraka's saline infusion technique meets the criteria with an added advantage of good epidural needle stability while inserting the needle throughout the procedure, as both hands are used for directing the needle.

As there is no comparative study for successful identification of epidural space and quality of analgesia between the loss of resistance technique using air as a medium and saline infusion technique using micro-drip infusion set in our population, an attempt is made to compare in this study.

# AIMS AND OBJECTIVES

The aim of the study is to compare the loss of resistance to air and saline infusion technique using micro-drip infusion set for identifying the lumbar epidural space for lower abdominal and lower limb surgeries with regards to the,

1. Success rate of identification of epidural space.
2. Quality of analgesia.

# REVIEW OF LITERATURE

Ever since Karl Koller gave the first clinical demonstration of the local anaesthetic properties of cocaine in 1884, [14] local and regional anaesthesia has had erratic clinical application. Following this great milestone, regional anaesthesia became a widely used technique by anaesthesiologists, realising its advantages over general anaesthesia. From ancient time, science has attempted various methods of pain relief. The development of epidural anaesthesia and analgesia plays a significant role in man's triumph over pain which undoubtedly is one of the most fascinating chapters in the history of medicine.

In October 1885, the New York City physician James Leonard Corning published an article under the title "Spinal Anaesthesia and local medication of the spinal cord", an event regarded for many years as the first spinal blockade. As the lumbar puncture had not been reported before, even the appearance of cerebrospinal fluid was not mentioned; despite the large dosages of cocaine that were injected, the onset of action was slow and the extent of analgesia limited. Most likely, the injections were made into peridural space and hence Corning was considered as the pioneer of peridural anaesthesia.

Two French physicians Jean Athanase Sicard and Fernand Cathelin independently did a lot of research on analgesia via the epidural space. Cathelin was the first to report on experiences by blocking the last sacral and coccygeal nerves by an anaesthetic solution in 1901. [14]

It was applied to clinical surgery by Pages in 1921 and by Dogliotti and Aburel in 1931 and popularised by Massey Dawfin. [14]

The adaptation of Tuohy's needle for use in the epidural blockade was done in 1945. [14]

Curbelo of Cuba was the first worker to insert a catheter into the peridural space in 1949. This played a major part in enabling improvement to be made in epidural blockade both by caudal and lumbar approach. [14]

In routine practice, epidural block is a popular method of anaesthesia as well as analgesia. Because it is safe, effective and has ability to prolong the duration of action depending upon duration of surgery and post operative pain relief, it is now a worldwide accepted technique by all anaesthesiologists.

The success of the epidural block mainly depends upon the correct identification of epidural space. Skill, experience and the technique are most important factors in identifying epidural space. It is essential not to promote a method which has more disadvantages and causes serious complications, when there is a safer alternative technique is available. [15]

Many investigators studied the anatomical and physiological aspects of the epidural space based on which, they tried to develop newer and safer techniques to identify the epidural space.

Most of these techniques are based on the principle of demonstration of subatmospheric pressure or sudden loss of resistance. These techniques extend from simple loss of resistance technique to ultrasound guided epidural space identification. To

name such techniques are, Odoms's indicatior, Macintosh's balloon, loss of resistance to air or saline, combination of both air and saline, loss of resistance using Lignocaine, combination of Lignocaine and air technique, Baraka's running saline infusion technique, hanging-drop technique, compuflo technique, etc.

Out of these above mentioned techniques, loss of resistance, [16] Baraka's saline infusion technique and hanging drop technique are widely practiced. The hanging drop technique may be regarded as an illogical choice for identification of epidural space because of absence of true negative pressure in this region. [17]

Loss of resistance to air is preferred over saline for the reason that air is readily available and cannot be confused for another substance and may permit easier detection of dural tap as compared to saline.

Using the above technique is associated with numerous complications that include venous air embolism, nerve root compression, subcutaneous emphysema, pneumocephalus and a greater incidence of incomplete analgesia and higher incidence of paraesthesia. [15]

Dogliotti in 1933 described loss of resistance technique for identification of epidural space, using fluid as a medium and was based on the different densities of tissues encountered as the needle tip passes through the thick, fibrous ligamentum flavum into the epidural space. [18]

Shenouda *et al*. compared complication rates between loss of resistance to air and normal saline in both adult and paediatric patients. They found multiple complications

with air compared to salineand concluded that there is not only improved analgesia but also decreased morbidity with loss of resistance to saline compared to air. [19]

Samuel Evron et al. Studied loss of resistance to air, Lidocaine, or the combination of air and Lidocaine and found, 17% occurrence of intravascular cannulation in air group compared with only 6% in the Lidocaine group—a statistically and clinically significant difference. [7]

During the LOR technique, 2 to 5 ml of air or saline is drawn into the syringeand continuous or intermittent pressure is applied to the plunger as the epidural needle is advanced toward the epidural space. [20] On entry into the epidural space, the syringe contents are injected due to a loss in resistance. Air and saline are the most widely used medium in the LOR technique.

There are several advantages and disadvantages of using air in LOR technique. The advantages being, air is easily availableand technique is easy to perform. Even though the incidences of complications are minimal while using air in LORT, several disadvantages are noted. The potential complications associated with the use of air for identifying the epidural space with loss of resistance technique may outweigh the benefits. The use of saline to identify the epidural space may help to reduce the incidence of these complications.

In an early study, the author concluded that using 0.9% saline for the LOR technique is associated with better analgesia as compared to air for labor analgesiaand this advantage should be considered when selecting the medium for the LOR technique. [20]

Valentine SJ et.al conducted a comparative study of the effects of air or saline to identify the extradural space and concluded that air is more likely than saline to produce unblocked segments in the initiation of extradural analgesia in labour. [21]

A study was conducted in 50 labouring parturients in whom either air or saline was used to identify epidural space. Quality of analgesia and onset of analgesic effect were assured. There was no significant difference between the groups in onset or quality of analgesia. There was a significant increase in the number of subjects who experienced segmental blocks after receiving air during the LOR technique. [22]

There are several advantages of using saline as a medium for identifying epidural space by LOR technique. Injecting 10 mL of saline through the epidural needle after intrathecal opioid injection and before threading the catheter significantly decreases accidental venous catheter placement without any apparent increase in complications from excessive cephalad intrathecal opioid spread.[23] Distension of the epidural space with 5 mL saline before epidural catheter insertion decreased the incidence of number of unblocked segments. [24]

Even though there are some advantages with the use of saline over air in LOR technique, it is not free from disadvantages. Okutomi T, Hoka S. Conducted a study on 70 patients using 2ml, 5ml and 10ml saline for its effect on spread of local anaesthetic. The results suggest that a large volume of saline solution injected in the epidural space to elicit loss-of-resistance dilutes the local anaesthetic solution, resulting in reduced spread of the block. [25]

In another study the authors concluded that the injection of 10 ml of physiological saline into the epidural space does not facilitate the advancement of an epidural catheter.[21] A false loss-of-resistance, in which the tip of the epidural needle lies within subcutaneous tissue, is the likely cause of most of the complete epidural failures. [26]

With the understanding of disadvantages of both air and saline as a medium in LOR technique, new techniques have been developed to prevent or to decrease these disadvantages. In a study conducted by Evron S et al where in, air, Lidocaine or both air and Lidocaine were used to identify the epidural space showed that unblocked segments using the air for LOR technique was about 6.6% than in the Lidocaine 3.2% or air plus Lidocaine 2.2% groups .[25]

Now most of the anaesthesiologists are curiously involved in finding an easy, safe, accurate and cost effective technique with minimal or no side effects for identification of epidural space. Newer methods like USG guided technique, [27, 28] and injection pump technology [5] are coming up to identify the epidural space.

The saline infusion technique has earlier been studied by Baraka for identification of thoracic epidural space in paediatric patients,[3] but we studied it in adult lumbar space using micro-drip infusion set to overcome most of its disadvantages. This is simple, cost effective, objective technique which is used to locate epidural space.

**ANATOMY**

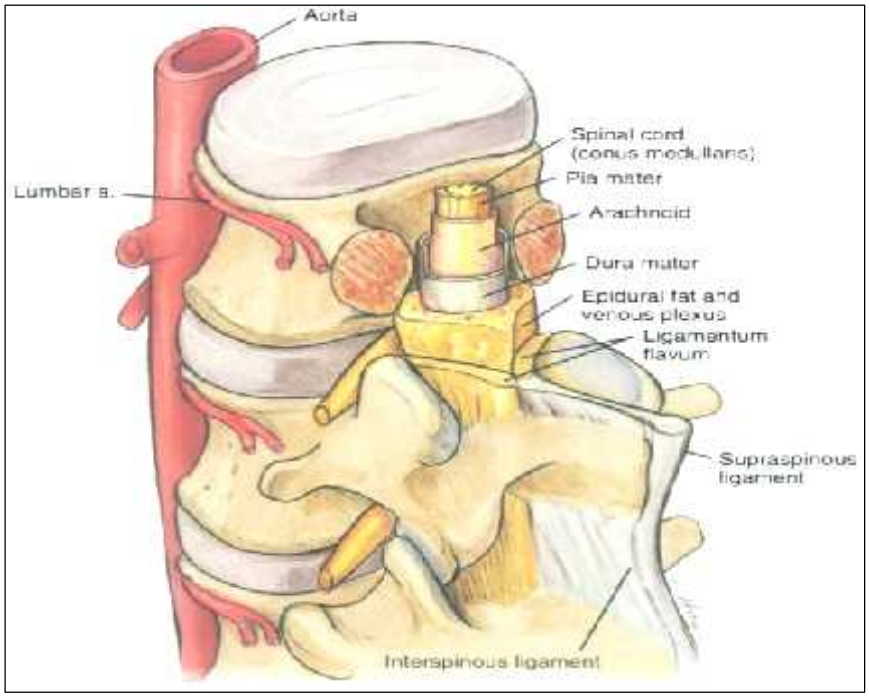



**Figure 1: Posterior view of lumbar spine, contents of epidural space and ligaments**.

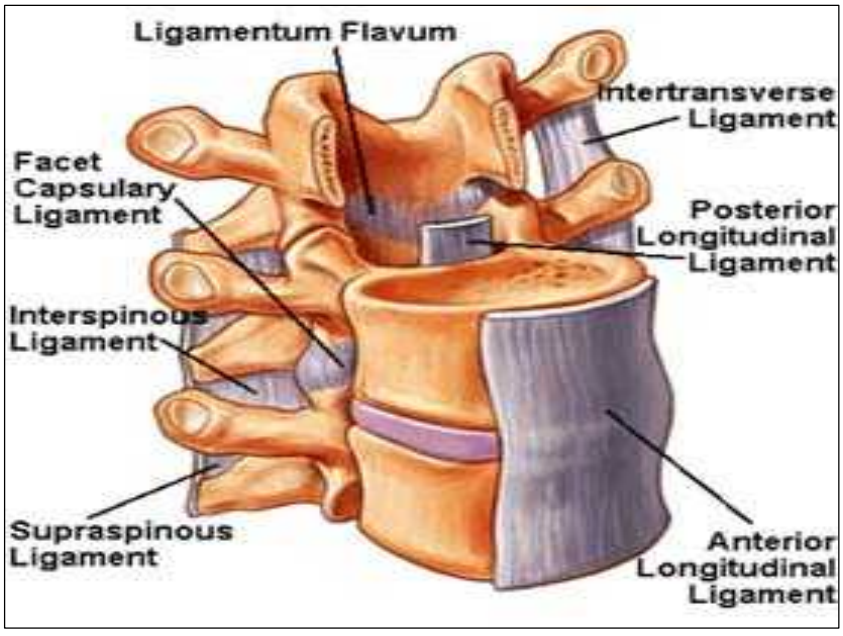



**Figure 2: Various ligaments and their attachments to vertebrae**

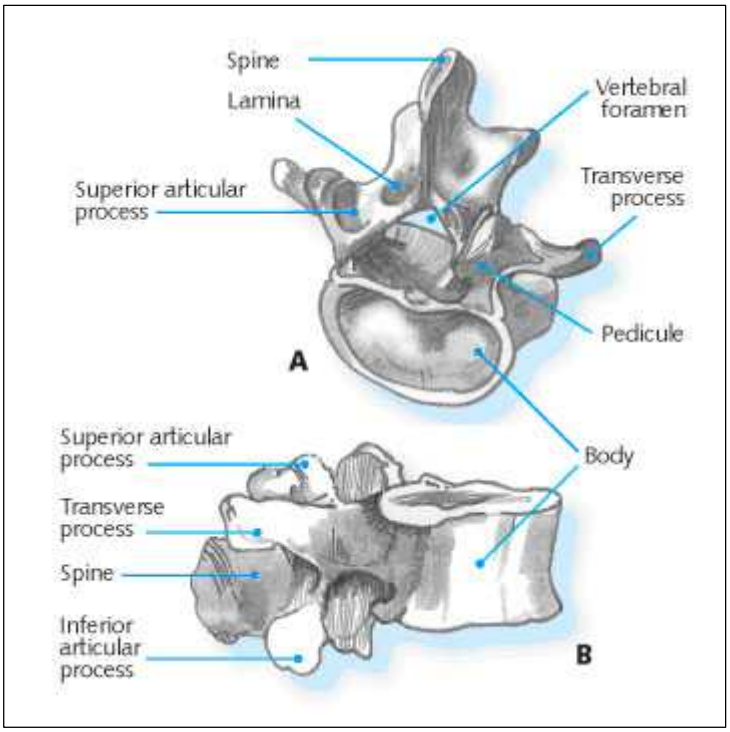



**Figure 3: Typical lumbar vertebrae and its parts**

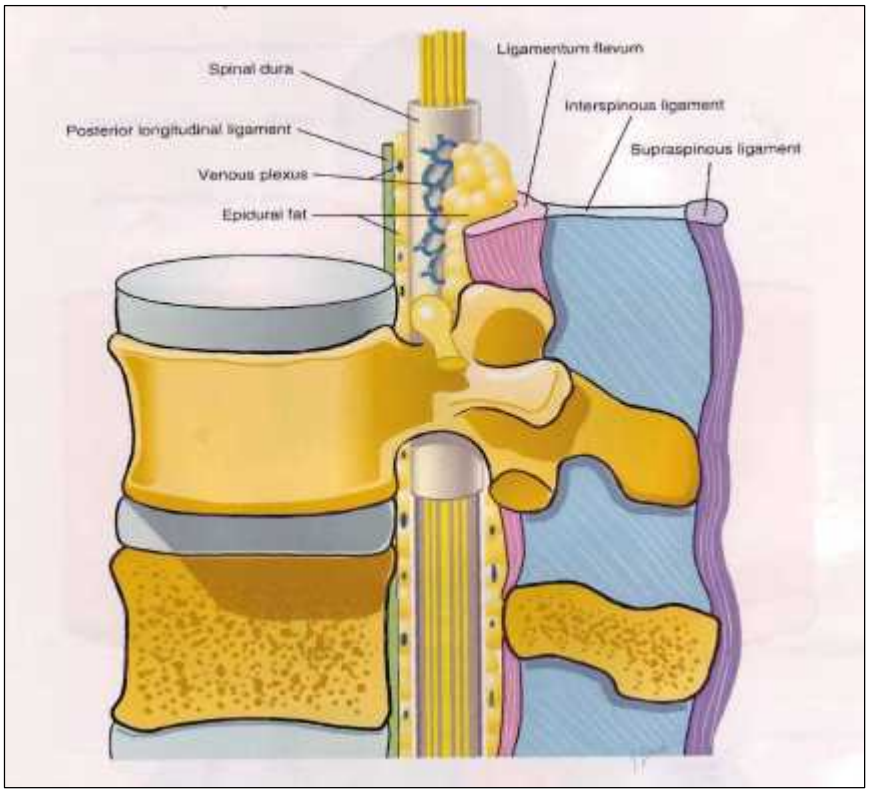



**Figure 4: Epidural space and its contents**

**Applied anatomy of epidural space**

A comprehensive understanding of the epidural space anatomy is of paramount importance for the anaesthesiologist.

The epidural space forms part of vertebral column lying between the spinal duramater internally and the periosteal lining of the vertebral canal externally. The spinal duramater forms the meningeal layer of the duramater of the brain. The periosteal lining of the vertebral canal represents the endosteal layer of duramater of the brain.

The epidural space is not as voluminous as the subarachnoid space. It extends from the base of the skull to sacrococcygeal membrane and has complicated direct communications with paravertebral space and indirect communications with cerebrospinal fluid.

The vertebral column is made up of vertebrae which offer protection to the spinal cord.

**Vertebral column**

The vertebral column comprises total of 33 vertebrae and includes 7 cervical, 12 thoracic, 5 lumbar, 5 fused sacral and 4 coccygeal vertebrae. The vertebral column has 4 curves which have significant effect on spread of drugs in subarachnoid space. Cervical and lumbar curves are convex anteriorly whereas thoracic and sacral curves are convex posteriorly. The highest points of cervical and lumbar curves in supine position are at C5 and L5; lowest points of thoracic and sacral are at T5 and S2 respectively

The vertebrae:

It is imperative for anaesthesiologist to have a thorough knowledge of the lumbar and thoracic vertebrae since epidural anaesthesia and analgesia are most commonly practised in these regions.

A typical thoracic vertebra consists of:

- Presence of articular facets on the bodies and articulation of transverse processes for articulation with the heads and tubercles respectively of the ribs.
- A Heart shaped body
- A small costal demi-facet on the lateral side of the body at superior border
- Larger demi-facet in the same lateral location but on the inferior border
- The superior vertebral notches are shallower and inferior vertebral notches are deep
- The transverse processes are directed backwards and laterally, each carrying a costal facet on its anterior aspect for articulation with the respective tubercle of the rib
- Spinous processes are long, slender, point downward and end in a single tubercle.

A typical lumbar vertebra consists of:

- Kidney shaped body
- Two pedicles, which are directed backwards from the upper part of the body.
- Two slender transverse processes.
- Two laminae meeting posteriorly and enclosing a triangular vertebral foramen.
- Thick and broad spinous process.
- Two upper and lower articular processes which prevents rotation but allows limited flexion and extension between contiguous vertebrae.

Deficiencies are present in the lateral and posterior walls of the vertebral canal, the former being the inter-vertebral foramen while the later is interlaminal foramen. In

extension the interlaminal foramen is small and triangular but elongates in flexion and provides access for the passage of epidural needle.

The spinous process of the cervical, the first two thoracic and the last four lumbar vertebrae are all horizontal and are opposite the bodies of their respective vertebrae. The other spinous processes are inclined downwards, their tips being opposite the bodies of the vertebrae next below. The direction of the spinous process determines the direction in which the epidural needle should be inserted.

**The intervertebral discs:**

One quarter of the length of the vertebral column is made up of these discs, each of which consists of an outer cover, the nucleus pulposus. The disc gives flexibility to the column and act as a shock absorber.

**The vertebral canal**

It is bounded by bodies of the vertebrae and intervertebral discs, posteriorly by the laminae, ligamentum flavum and the arch which bears spinous processes and by ligaments between them called the interspinous, laterally by pedicles and laminae.

**Contents**:

- Spinal nerve roots
- Spinal membranes with their enclosed cord and cerebrospinal fluid.
- Structures- vessels, fat and areolar tissues of epidural space
- The narrowest part of the vertebral canal is between T4-T9.

**The vertebral ligaments:**

The vertebrae are held together by a series of overlapping ligaments which bind the vertebral column and assist in protecting the spinal cord.

**Supraspinous Ligament**: It passes longitudinally over tips of the spinous processes from C7 to sacrum. It varies in width directly with the width of the spinous process; in lumbar region it may be as much as 1 cm wide. In labourers and elderly people this ligament becomes calcified making midline approach difficult.

**Interspinous Ligament**: Runs obliquely between the spinous process and continues anteriorly with ligamentum flavum and posteriorly with the supraspinous ligament. They join the spinous processes together from their roots to the tips. In the lumbar region the ligament is rectangular in shape leading to identifiable resistance to air or saline.

**Ligamentum flavum**: It runs from lamina to lamina and is composed of yellow elastic fibres. Because of its tough elasticity and thickness especially in the lumbar region, it imparts a characteristic 'springy' resistance. It runs from anterior and inferior aspects of the lamina below. Laterally, the ligament narrows as it blends with the capsule of the joint between the articular processes. Half of the substance of the posterior wall of the vertebral canal is composed of the bony lamina and the other half by ligamentum flavum.

**Posterior Longitudinal Ligament**: It lies within the vertebral canal on the posterior surface of the bodies of vertebrae, from which it is separated by the basivertebral veins.

**Anterior longitudinal ligament**: it runs along the front of the vertebral bodies and is adherent to both the body and the disc.

For epidural anaesthesia the needle pierces the first three of these. In lateral approach only ligamentum flavum is encountered.

**The epidural space:**

The epidural space forms part of the vertebral column lying between the spinal duramater internally and the periosteal lining of the vertebral canal externally. The spinal duramater represents the meningeal layer of the duramater of the brain. The periosteal lining vertebral canal represents the endosteal layer of duramater of brain.

**Boundaries**: superiorly – foramen magnum, where the periosteum of the spinal canal and spinal dura fuse together to form the endosteal and meningeal layer of the cerebral dura.

**Inferiorly**: the sacrococcygeal membrane and sacral hiatus.

**Anteriorly**: Posterior longitudinal ligament covering the vertebral bodies and intervertebral disc.

**Posteriorly** : The periosteum of anterior surface of laminae, articular processes and their connecting ligaments, roots of vertebral spines and the interlaminar space filled by ligamentum flavum.

**Laterally**: The periosteum of pedicles of the vertebrae and inter vertebral foramina.

In cross section it is circular in cervical and upper thoracic region and becomes triangular with apex dorso-medial in lower thoracic and lumbar region.

Occasionally a dorso-medial fold of duramater divides the space into ventral and dorso-medial compartments which do not always communicate freely with each other. Such abnormality may cause patchy analgesia, unilateral analgesia and missed segments and inadvertent dural puncture when mid line approach is used during lumbar epidural blockade.

**Size of epidural space**:

At cervical region: 1.5mm

At upper thoracic region: 2.5-3mm

At lower thoracic region: 4-5mm

At lumbar region: 5-6mm

Contents of the epidural space;

This includes extradural plexus of veins, spinal nerve roots, spinal arteries, lymphatics and fat, dural sac.

**Epidural veins**: The large valve - less epidural veins form the internal vertebral venous plexus, draining the neural tissue of the spinal cord, the cerebrospinal fluid and the bony spinal canal. They connect by the way of sacral venous plexus to the uterine and the iliac vessels below and to the intra-cranial veins above which includes occipital, sigmoid and basilar venous sinuses. The main trunks communicate by venous rings at the vertebral body level with the ascending and deep cervical, intercostals, iliolumbar and lateral sacral veins.

In case of large abdominal tumours and in advanced pregnancy, the epidural veins are distended due to compression on inferior vena cava by the tumour and gravid uterus.

The epidural veins are most prominent along the lateral walls of the spinal canal and so it is out of reach of the correctly placed epidural needle in the midline approach. The dose and rate of local anaesthetic should be reduced in case of inferior vena cava obstruction as it will diminish the effective volume of epidural space with the result that injected local anaesthetics spreads more widely up or down the epidural space.

**The epidural fat**: It is a semifluid, lobulated, areolar tissue which extends throughout spinal and caudal space. It is most abundant posteriorly. It is very vascular with small capillaries that form a rich network in its substance. Fat has great affinity for drugs with high lipid solubility like Bupivacaine and Etidocaine which may remain in the epidural fat for a long time. Uptake of local anaesthetic into epidural fat competes with vascular and neural uptake.

**Spinal arteries**: These arteries arise from the vertebral, ascending cervical, deep cervical, intercostals, lumbar and ilio lumbar arteries. They anastomose with their neighbours above and below, also lie chiefly in the lateral parts of the epidural space.

Its significance to epidural block is that, the spinal branches of the subclavian, aortic and iliac arteries cross the epidural space and enter the subarachnoid space in the region of dural cuffs. The largest feeding artery to spinal cord is artery of Adamkiewicz which supplies the anterior spinal artery in the area of lumbar enlargement of the cord. It enters by the way of single intervertebral foramen, usually on the left side between T8 and L3 foramen. Damage to this artery by epidural needle can result in ischemia to the entire lumbar enlargement of the cord.

Blood vessels supplying spinal cord pass through the epidural space. The anterior spinal artery is more susceptible, because it is not paired, has three distinct level of supply with little anastomoses between them and has only minimal horizontal anastomosis with the posterior spinal arteries. Thus, even epidural puncture below L2 level avoid direct trauma to spinal cord. Dangerous trauma to anterior spinal artery can be minimized by puncture at L3-L4.

**Lymphatics**: The dural cuff region is supplied with a rich lymphatic network that rapidly conveys debris from arachnoid villi out through intervertebral foramina to reach lymph channels in front of the vertebral bodies. It is reassuring that foreign material can be carried away rapidly by an efficient system that runs in a direction away from spinal fluid and spinal cord.

**Dural sac**: It contains dura, arachnoid, CSF, spinal nerves and spinal cord.

**Some useful surface markings:**

The inferior angle of the scapula corresponds to T7, the root of the spine of the scapula to T3 and the vertebra prominence C7. A line drawn between the highest points of both iliac crests (Tuffier's line) usually crosses either the body of L4 or the L4–L5 interspace. The dimples overlying the posterior superior iliac spine are on a line crossing the second posterior sacral foramina and at this level the dural sac in the adult usually ends. The lower end of the spinal cord terminates at the level of the upper border of the body L2. The line through posterior superior iliac spines crosses the S 2 level.

**Physiology of Epidural Blockade**

The physiological responses to epidural blockade are mainly because of sympathetic blockade accompanied by somatic blockade which may involve sensory blockade with or without motor blockade.

The major sympathetic blockade can be avoided by restricting the level of analgesia at T10 level. This level of sympathetic blockade is a very practical approach in considering the physiologic effects of epidural blockade because lower abdominal, inguinal, perineal, urological and lower limb surgical procedures can be carried out satisfactorily and will only produce peripheral sympathetic blockade.

In all regional anaesthesia there exists competition of uptake of local anaesthetic between nerves and blood vessels and this is more clearly observed and easily demonstrable in epidural anaesthesia. In subarachnoid block the local anaesthetics directly come in contact with the nerves and a high proportion of it is taken up by the nerves. Within the epidural space the venous plexus which is present in large surface area which makes local anaesthetic, to get absorbed in higher concentrations.

As large doses are used for extensive epidural blockade with or without Epinephrine, this may cause physiological changes as a result of the direct pharmacological effects of circulating blood concentrations. Direct intravascular injection may result in the rapid attainment of very high concentrations of local anaesthetic in the brain and or heart resulting in potential for convulsions and or sudden depression of cardiac output.

Erlanger and Gasser showed that the action of local anaesthetics on nerve fibres is by “Differential conduction blockade”. The nerve fibres are of three types viz, A, B, C types.

Type A and B are myelinated while C fibres are unmyelinated. Differential conduction blockade can be illustrated by selective blockade of preganglionic sympathetic nervous system B fibres with low concentrations of local anaesthetics. Slightly higher concentrations of local anaesthetics interrupt conduction in small C and small and medium- sized A fibres, with loss of sensation of pain and temperature. Touch, proprioception and motor functions are still present such that the patient will sense

pressure but not pain with surgical stimulation. In anxious patient, however, any sensation may be interpreted as failure of the block.

In contrast to spinal anaesthesia, during epidural anaesthesia, there is no zone of differential sympathetic nervous system blockade and the zone of differential motor blockade may average up to four rather than two segments below the sensory level.

A minimum length of myelinated nerve fibre should get exposed to local anaesthetic for conduction blockade. Blockade occurs at the nodes of Ranvier of myelinated fibres. Three adjacent nodes must be blocked for nerve conduction to be completely interrupted. Preganglionic B fibres are more readily blocked by LA than any fibre, even though these are myelinated fibres. Both types of pain conducting myelinated A-delta fibres and nonmyelinated C fibres are blocked with similar Cm of local anaesthetics, despite the differences in the diameters of these nerve fibres.

The minimum concentration of local anaesthetic necessary to produce conduction blockade of nerve impulses is termed as the Cm. The Cm is analogous with minimum alveolar concentration for inhaled anaesthetics. Nerve fibre diameter influence Cm, with larger nerve fibres requiring higher concentrations of local anaesthetic for conduction blockade. The Cm of motor fibres is approximately twice that of sensory fibres; thus, sensory anaesthesia may not always be accompanied by skeletal muscle paralysis. An increased tissue pH or high frequency of nerve stimulation decreases Cm. Each local anaesthetic has a unique Cm, reflecting differing potencies of each drug.

Local anaesthetics injected epidurally, block nerve conduction depending upon the concentration and volume injected, the sensitivity of nerve fibres and type of drug used.

Local anaesthetic in the epidural space may gain access to the site of action by diffusing through the dural cuffs into the roots round which they are injected, by entering into the paravertebral spaces, or by diffusing into the cerebrospinal fluid.

**Factors influencing height and distribution of local anaesthetic:**

**Patient characteristics:**

Age

Weight

Height

Intra abdominal pressure

Posture

Gender

**Technique of injection:**

Site of injection

Direction of the bevel

Rate of injection

**Characteristics of anaesthetic solutions**

Amount

Concentration

Density

Temperature

Additives

1. Age: Bromage in his study found that there is a correlation between age and dose requirements. An increase in the dose requirement was found from the age 4 to 18 yrs and gradual decrease in the dose from the age 19 to 104 yrs.

   A thumb rule for 2% Lignocaine solution and Epinephrine or equivalent

   20-40yrs – 1 to1.5ml/segment

   40-60yrs – 0.5 to 1ml/segment

   60-80yrs – 0.3 to 0.6ml/segment

   Further adjustment should be made on the basis of height, site of injection and pathophysiological state.

2. Height: A dose of 1 ml per segment is adequate for the majority of patients of height 150 cm, while a dose of 1.6 ml per segment is sufficient for the majority of patients of height 180 cm. A simple thumb rule is to use 1 ml per segment for height of 150 cm and then add 0.1 ml per segment for each 5 cm over 150 cm.

3. Weight: There is little correlation between the spread of analgesia and the weight of the patient. However, in morbidly obese patients, there may be compression of the epidural space secondary to increased intraabdominal pressure, creating a higher block for a given dose of local anaesthetic.

4. Intraabdominal pressure: Seen in obesity, intraabdominal tumour and in the pregnant patient, venous engorgement in the epidural veins increases the risk of entry into a vessel.

5. Posture: It has a mild but significant effect on spread of analgesia. Bromage found, in sitting position caudal spread of analgesia was favoured and dose requirements were slightly increased in comparison to the horizontal position. Gravity has some influence on anaesthetic solutions as it tends to travel downwards in the epidural space and posture affects segmental spread and dose requirements.

6. Site of injection: Rapid onset of action and intense blockade was observed if the local anaesthetics were injected nearer to the nerve roots. When compared to caudal, lumbar epidural injection shows greater cranial spread.

7. Rate of injection: A rapid injection of local anaesthetic produces an incomplete but rapid onset and more extensive block. Injection rate of 0.3-0.75ml per sec results in the most reliable spread of analgesia.

8. Volume, concentration and dose of local anaesthetic agent: Earlier the epidural block was considered as a "multiple paravertebral block" which led to use of larger volume of diluted local anaesthetic. Extensive studies by Bromage showed that the dosage of a drug determine the spread of analgesia. Increasing dosage results in linear increase in degree of sensory block and duration of epidural block, while increasing the concentration increases the intensity of motor blockade.

Dose spread = volume of Anaesthetic solution injected / number of dermatomes blocked.

**Effects of epidural block on various systems:**

**1. Cardiovascular system:**

- Block Below T4

The effect of epidural anaesthesia on the cardiovascular system depends on the level and the degree of sympathetic blockade. Vasomotor tone is maintained by sympathetic fibres from T5 to L1 that innervate vascular smooth muscles. Blockade of these fibres causes venodilation with venous pooling as well as arterial vasodilation with decreased systemic vascular resistance. The venous pooling leads to a marked decrease in venous return, right atrial pressure and subsequently, cardiac output. The decrease in venous return can then lead to an increase in cardiac vagal tone, especially for blocks near the T5 level. Clinically, the patient can be hypotensive without a change or a decrease in heart rate.

Normal cardiac output is maintained either by volume loading or by physiologic mechanism, (i.e, physiologic release of catecholamine and vasoconstriction in unblocked area), the total peripheral vascular resistance will only decrease by approximately 15%, a value well tolerated by a healthy patient. In an elderly patient with cardiovascular disease, a more ominous decrease in blood pressure with significant hypotension can develop.

- Block Above T4

The cardiovascular effects of a block above T4 are the result of a high sympathetic block. The cardiac sympathetic fibres arise from T1 to T4 and when blocked, profound hypotension (the result of a decrease in cardiac contractility) and bradycardia can occur. In addition to the cardiac effects, a high level of sympathetic blockade causes:

Increased central venous pressure without an increase in stroke volume

- Vasoconstriction in the head, neck and upper limbs

- Splanchnic nerve blockade with blockade of medullary secretion of catecholamines
- Blockade of vasoconstrictive effect on the capacitance vessels of the lower limbs

When a sympathetic block occurs at such a high level, the cardiovascular system may be left without its mechanisms for responding to low cardiac output states. This can be detrimental to a patient with limited cardiac reserve because profound hypotension with bradycardia and decreased contractility can result.

**2. Respiratory**

Epidural blockade to midthoracic levels have minimal effect on patients with adequate lung function. Lung volumes (tidal volume, vital capacity), resting minute ventilation and dead space are basically unchanged even with a higher thoracic epidural. Even with abdominal or intercostal muscle paralysis by a high thoracic block, major alteration in pulmonary function is not seen.

There is concern regarding the use of epidural blockade in patients with severe chronic lung disease dependent on accessory muscle function to maintain adequate ventilation, because paralysis of respiratory muscles and changes in bronchial tone from epidural analgesia can occur. In a study by Gruber and colleagues, thoracic epidurals were placed in patients with end-stage chronic obstructive pulmonary disease undergoing lung volume reduction surgery. Thoracic epidural analgesia with 0.25% Bupivacaine did not adversely affect ventilator mechanics, breathing pattern, gas exchange and inspiratory muscle force generation in these patients.

**Gastrointestinal**

The gastrointestinal effects of epidural anaesthesia are largely the result of blockage of the sympathetic splanchnic fibres from the T5 through L1 level. Unopposed

vagal dominance leads to an increase in secretions, peristalsis and a small contracted gut. Postoperatively, gastrointestinal motility returns more quickly when epidural analgesia with a local anaesthetic is instituted. Several studies have been conducted demonstrating the positive effect of thoracic epidural anaesthesia on visceral perfusion. Christopherson and colleagues used intramucosal pH measurements (pHi) as an indicator of stable visceral perfusion during abdominal surgery. They suggested that thoracic epidural anaesthesia prevented the decrease of intramucosal pH during major abdominal surgery as an effect of stable visceral perfusion. When thoracic epidural anaesthesia is used as an adjunct to general anaesthesia for major thoracic, cardiac, or abdominal surgery, a segmental block of T1 through T5 is typically the goal. Segmental sympatholysis creating an increase of sympathetic activity in segments below the block leading to impaired splanchnic blood flow has been a concern. In a study in awake and anesthetized dogs, an upper thoracic epidural block had no compromising effect on gastrointestinal perfusion.

Nausea is a common problem following neuraxial anaesthesia. It has been reported to occur in up to 20% of patients undergoing neuraxial blocks. It is thought to be related to increase in gastric peristalsis secondary to unopposed vagal activity. It can be prevented by promptly treating hypotension with a fluid bolus, ephedrine, or phenylephrine.

**Renal/Genitourinary**

Since renal blood flow is maintained through autoregulation, an epidural has very little effect on renal function. Neuraxial blockade at the lumbar level has been postulated to impair control of bladder function secondary to blockade of the S2 to S4 segments. Urinary retention may occur until the block wears off. The clinician should avoid giving

excessive intravenous crystalloids if a urinary catheter is not in place. If a continuous epidural is used, then urinary catheterization may be necessary. More recent studies have questioned the validity of this belief.

**Neuroendocrine**

Surgical stress produces a variety of changes in endocrine and metabolic function. Increased protein catabolism and oxygen consumption are common. Increased plasma concentrations of catecholamines, vasopressin, growth hormone, renin, angiotensin, cortisol, glucose, antidiuretic hormone and thyroid-stimulating hormone have been documented and referred to as the surgical stress response. Intraoperative manifestations of the response are demonstrated as hypertension, tachycardia, hyperglycemia, suppressed immune function and altered renal function. Afferent sensory information from the surgical site is thought to play a pivotal role in the response. The response can be completely abolished by an appropriate level of sensory blockade produced by regional anaesthesia. The inhibitory effect is greatest with lower abdominal and lower extremity surgery and slightly less effective in upper abdominal and thoracic surgery, probably because the epidural cannot completely block all nociceptive afferent pathways.

The most critical effect of neuroendocrine activation in the perioperative period is the increase in plasma Norepinephrine, which peaks about 18 h after the surgical stimulus is initiated. The increase in plasma Norepinephrine is associated with activation of nitric oxide in the endothelium of patients with atherosclerotic disease, producing paradoxic vasospasm. Thus, in patients with significant atherosclerotic disease, the combination of paradoxic vasospasm and the hypercoagulable state may be the reason underlying the

cardioprotective effects of thoracic epidural anaesthesia and analgesia in patients with cardiac disease

**Site of action of local anaesthetic agent in the epidural space:**

1. The site of action may be on anterior and posterior nerve roots with their ganglia, in the epidural space.

2. On the mixed nerve roots in the paraverteral space after they have shed their dural sheaths.

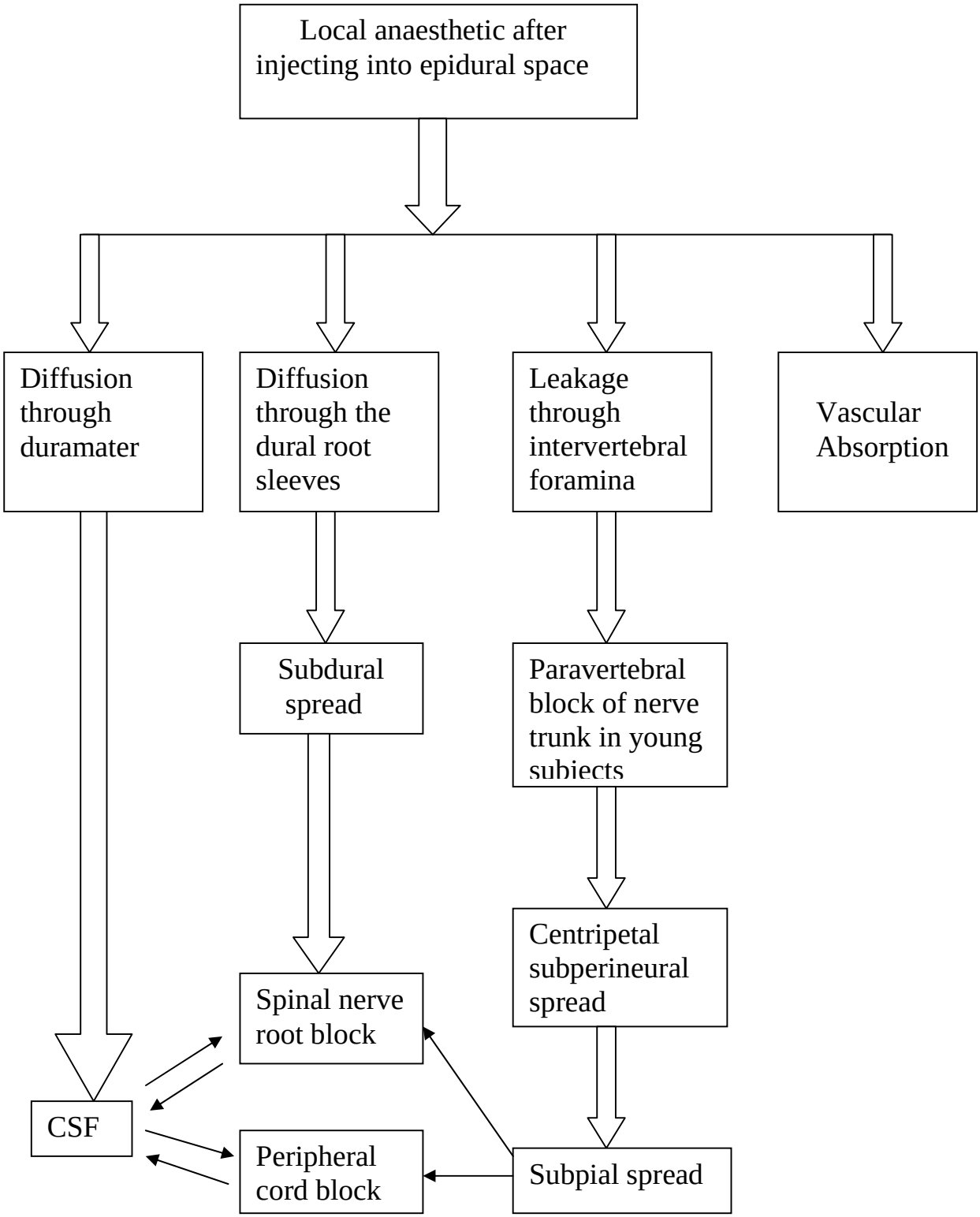
3. On the visceral afferents accompanying sympathetic fibres, white and grey communicans.

4. On the nerve roots in the intradural or sub arachnoid space after inward diffusion of the drug across the dura.

5. Diffusion into the subperineural and subpial spaces from the so called 'ink cuff' zone where the anterior and posterior nerve roots fuse and it may eventually pass centripetally and reach the substance of the cord and diffuse from these into the CSF.

6. Peripherally placed ascending and sympathetic excitatory tracts.

## Fate of local anaesthetics in epidural space



## Pharmacology

To be successful with epidural blockade, the clinician must understand the physiology of nerve conduction and the pharmacology of the local anaesthetics. Potency and duration of the drugs, their ability to preferentially block sensory and motor fibres, as well as the anticipated duration of surgery or need for postoperative analgesia are factors to be considered before instituting epidural blockade.

Local anaesthetics are drugs that produce reversible conduction blockade of impulses along central and peripheral nerve pathways after regional anaesthesia. With progressive increase in concentration of local anaesthetics, the transmission of autonomic, sensory and motor impulses are interrupted leading to autonomic nervous system blockade, sensory anaesthesia and skeletal muscle paralysis in the area innervated by the affected nerve.

The basic properties of an ideal local anaesthetic agent are:

High potency

Short latency

Highly penetrating

Completely reversible

Prolonged duration of action

Low toxicity

No tachyphylaxis

Easy to sterilize

Easily available

Cost effective

**Lignocaine hydrochloride (Lidocaine, Xylocaine)**

This tertiary amide was synthesized in 1945 in Sweden by Lofgren and Lundquist. It came in to clinical practice in 1948, which was introduced by Gordh of Karolinska hospital, Stockholm.

Chemistry: It is 2 diethylamine – 2, 6, acetoxylidide hydrochloride.

Physio-chemical properties:

a. Solubility: It is freely soluble in water. pH of 1% solution in 0.9% saline is 6.5-7.
b. Stability: Lignocaine solutions are stable and are not decomposed by boiling, or mixing with acids or alkalis. It can be autoclaved. The stability of Lignocaine is absolute within a wide range of temperature and pH.

Anaesthetic properties:

a. Potency: It is more potent than Procaine. Onset of analgesia is at least two times faster than with procaine. Depth of anaesthesia is more compared to procaine. Since Lignocaine has got high margin of safety it is used safely.
b. pKa: Lignocaine is a weak base pKa of 7.8. At pH of 7.4 the 35% of Lignocaine is in basic form.
c. Anaesthetic index: It is defined as the ratio of potency to the toxicity of a local anaesthetic drug. For Lignocaine, it is 2-3 when used for infiltration with 0.5% and 1 for 2% solution.
d. Dosage: It depends upon individual patient. Maximum dose of Lignocaine without Epinephrine is 3mg/kg and with Epinephrine is 7mg/kg.
e. Duration of nerve block: 60-120 min.

Side effects of Lignocaine: The principle side effects related to the use of local anaesthetic are allergic reactions and systemic toxicity due to excessive plasma and tissue concentrations of the local anaesthetic.

1. Central Nervous System: The most frequent side effect of Lignocaine involves the central nervous system. Following absorption it causes stimulation of CNS leading to confusion, restlessness, drowsiness, tinnitus, dizziness.

   As the dose increases seizures, coma, respiratory depression and cardiopulmonary arrest may occur.

   CNS stimulation is followed by CNS depression. Death is usually by respiratory failure. The apparent stimulation and subsequent depression produced by local anaesthetics to CNS presume to be due to depression of neuronal activity. A selective depression of inhibitory neurons is thought to account for the excitatory phase in vivo. Rapid administration of local anaesthetic may produce death with or only transient sign of CNS stimulation. Under these circumstances the concentration of the drug probably increases so rapidly that all neurons are depressed simultaneously.

   Drowsiness is the most frequent complaint which results from the CNS actions of Lignocaine. It may produce dysphoria, euphoria, loss of consciousness that is preceded by symptoms of sedation.

2. Neuromuscular junction and ganglionic synapse: Local anaesthetics affect transmission of impulse at neuromuscular junction. Procaine for example can block the response of the skeletal muscle to maximal motor nerve and to acetylcholine at a concentration where the muscle responds normally to direct electrical stimulation. Similar effect occurs at autonomic ganglia.

3. Cardiovascular system: Primary site of action is on the myocardium. Lignocaine decreases conduction rate, decreases electrical excitability, decreases force of contraction. In addition most of the local anaesthetics cause arteriolar dilatation. The cardiovascular effects are usually seen only after attaining high plasma concentration and effects on CNS are produced.
4. Smooth muscle: Lignocaine depresses the contraction in strips of isolated bowel. They also relax as vascular and bronchial smooth muscle although low concentration may initially produce constriction.
5. Toxicity and blood Lignocaine concentration response relationship: Subjective toxic effects of Lignocaine on the CNS occurs at blood Lignocaine concentration of 3-5 mcg/ml and objective adverse manifestation including muscular irritability, convulsions and coma appear at blood Lignocaine concentration of 6-10 mcg/ml. At 20 mcg/ml respiratory arrest occurs. At 26 mcg/ml cardiovascular depression occurs.

The blood concentration of Lignocaine from 1.5-5.5mcg/ml is the therapeutic range.

Metabolism and excretion: Lignocaine is predominantly metabolised in liver. The amide linked local anaesthetic are in general degraded by hepatic endoplasmic reticulum, the initial reaction involving N-dealkylation and subsequent hydrolysis.

There are two major metabolites of Lignocaine.

a) Monoethyl glycine xylidide (MEGx)
b) Glycine xylidide (Gx)

These are found in significant concentrations in the blood of patients receiving Lignocaine therapeutically. These metabolites also have antiarrhythmic and convulsant

activity in animals, with monoethyl glycine xylidine appearing to have potencies similar to Lignocaine.

Monoethyl glycine xylidide is metabolised in liver and has, a half life of 120 min which is similar to that of Lignocaine. Glycine xylidide is (10-26%) as potent as Lignocaine. Glycine xylidide is metabolised and excreted by kidney and unlike Lignocaine and Monoethyl glycine xylidide, Glycine xylidide has long half life of 10 hrs. Thus the metabolites of Lignocaine also contribute to the toxicity while the blood Lignocaine levels are within the therapeutic range.

**Renal excretion**: Lignocaine is excreted rapidly from the body. Total body clearance is 0.95% litres/min owing to rapid extraction by liver. Renal clearance of the drug is related inversely to its protein binding capacity and to the pH of urine.

**Distribution**: At the usual therapeutic blood concentration, Lignocaine is bound extensively (55-95%) to plasma protein particularly to alfa1 acid glycoprotein. It is less bound to erythrocyte than to plasma protein and concentration in whole blood differs from that in plasma.

Partition co-efficient of Lignocaine also varies. Spleen has high affinity (tissue to plasma co-efficient 3.5).

Lignocaine is a weak base with pKa 7.85 near physiologic pH observed in tissues. In acid environment more Lignocaine is ionised and trapped. Age related changes in the protein binding also occur. The neonate is relatively deficient in plasma proteins that bind local anaesthetics and thereby has great susceptibility to toxicity. Plasma proteins are not sole determinants of local anaesthetic availability. Uptake by the lungs also plays an important role in the distribution of amide linked local anaesthetics in the body.

## Bupivacaine

This local anaesthetic is one of the homologous series synthesized by Bo of Ekenstam in 1957 and its first clinical use was made in 1963 by L.J.Telivuo.

Presently it is available in the form of 0.5%, 0.25% and 0.75%.

**Dosage**: 2mg/kg for single shot epidural blockade.

**Basic pharmacology**: It is 2-pyridine carboxamide, 1-butyl-N-2, 6- dimethyl phenyl, monohydrochloride and monohydrate. Bupivacaine molecule is a tertiary amine separated from an aromatic ring system that is a benzene ring by an intermediate chain. The tertiary amine is a base that is a proton acceptor. The chain contains an amide linkage (-NHCo-) therefore it is classified as an amionoamide compound. This amide linkage contributes to the anaesthetic potency.

The aromatic ring system gives lipophylic character to its portion if molecules, where as the tertiary amine end is relatively hydrophilic.

**Structure-activity relationship**: It binds to protein, soluble in lipid and pKa of 8.1 these account for many of the positive attributes, minimal placental transfer, long duration of action and potency.

**Mechanism of action**: The uptake of the drug by the tissues is largely due to lipophyllic adsorption. This shifts effective pKa downwards and thereby favouring the neutral base form.

Bupivacaine blocks impulses by reducing the currents through voltage activated $Na^+$ channels. The inhibition is not specific; however $K^+$ currents are also reduced. Binding of Bupivacaine to sites on voltage gated $Na^+$ channels presents opening of the channels by inhibiting conformational changes.

**Clinical Pharmacology:**

1. Anaesthetic Potency: Hydrophobicity is a major determinant of intrinsic anaesthetic potency and Bupivacaine is highly hydrophobic and is very potent.
2. Onset of Action: It depends upon the pH of the drug and its concentration.
3. Differential and sensory / motor blockade: Bupivacaine 0.25- 0.75% produces adequate analgesia with less of motor blockade.

**Factors influencing anaesthetic activity:**

1. Dosage of Bupivacaine: As dosage of Bupivacaine is increased, the probability and duration of satisfactory analgesia will increase and the onset of block will be shortened. The dosage can be increased by administering either large volume or more concentrated solutions.
2. Addition of vasoconstrictor: Addition of Epinephrine does not significantly increase the duration of action of Bupivacaine.
3. Site of action: the latencies and duration are long when given for brachial block, epidural block, subarachnoid block.
4. Compounding of local anaesthetic: the basis for this practice is rapid onset of one agent e.g. Lignocaine and longer duration of action of other agent e.g. Bupivacaine.

5. Pregnancy: The spread and depth of spinal and epidural analgesia are greater in pregnant than in non-pregnant women.
6. Carbonation: The success of any local anaesthetic depends upon the quantity of drug that can be absorbed on to the axon membrane of the target nerves. This in turn depends upon the ability of the drug to penetrate tissue barrier around the nerve. Alkalinization of local anaesthetic solution improves the penetration power and more availability of diffusible base of the local analgesic.

The addition of sodium bicarbonate to Bupivacaine increases the pH of the solution without affecting its chemical stability.

**Pharmacokinetics of Bupivacaine**: The concentration of Bupivacaine in the blood is determined by the amount injected, the rate of absorption from the site of injection, the rate of tissue distribution and the rate of biotransformation and excretion of the drug.

**Absorption**: The systemic absorption of Bupivacaine is determined by the site of injection, dosage and addition of vasoconstrictor agent. The maximal blood level of Bupivacaine is related to the total dose of the drug administered for any particular site of injection.

**Distribution**: The distribution of Bupivacaine can be described by a two or three compartment model. The rapid distribution phase (alpha) phase is believed to be related to uptake by rapidly equilibrating tissues, that is, tissues which have high vascular perfusion. The slower (beta) phase is mainly a function of distribution to slowly equilibrating tissues and the biotransformation and excretion of the compound.

Most highly perfused organs show higher concentration of the drug. Bupivacaine is rapidly extracted by lung tissues. The highest percentage of injected dose of the local anaesthetic is found in skeletal muscle.

**Biotransformation and excretion:**

Bupivacaine undergoes enzymatic degradation primarily in the liver. The excretion of Bupivacaine occurs via the kidney. Less than 5% of the unchanged drug is excreted via this route. The major portion of the drug appears in the urine in the form of various metabolites. 2, 6 pipecoloxylidine (PPX) is a N-dealkylated metabolite of Bupivacaine. Renal clearance of the drug is related to its protein binding capacity and to the pH of urine.

**Pharmacokinetic alteration by patient status:**

Age of the patient: Half life of Bupivacaine significantly increases in old age.

Hepatic factors: In patients in whom liver blood flow is abnormally low or nonexistent, significantly higher blood levels of Bupivacaine occur.

**Toxicity of Bupivacaine**: It is relatively free of side effects if administered in an appropriate dosage and in the appropriate anatomic location. However, systemic reactions can occur.

**CNS toxicity**: The CNS is more susceptible to Bupivacaine. The initial symptoms involve feeling of light headedness and dizziness followed by visual and auditory disturbances. Disorientation and occasional feeling of drowsiness may occur. Objective signs are usually of excitatory nature and include shivering, muscular twitching and

tremors initially involving muscles of face and distal parts of the extremities. Ultimately generalized convulsions of tonic-clonic nature may occur. At still higher dose CNS depression and respiratory arrest occurs.

**CVS toxicity**: It depresses the rapid phase of depolarisation in purkinje fibres and the ventricular muscles to a greater extent than Lignocaine. Also the rate of recovery from a block is slower with Bupivacaine than Lignocaine. This results in an incomplete restoration of Vmax between action potentials particularly at high heart rates in contrast to complete recovery with Lignocaine. This explains the antiarrhythmic properties of Lignocaine and the arrhythmogenic potential of Bupivacaine.

High levels of Bupivacaine prolong conduction time through various parts of the heart. Extremely high concentrations will depress spontaneous pacemaker activity, resulting in bradycardia and cardiac arrest. Cardiac resuscitation is more difficult following Bupivacaine induced cardiovascular collapse. Acidosis and hypoxia markedly potentiate the cardiotoxicity.

Pregnancy enhances cardiotoxic effects of Bupivacaine. 0.75% solution is no longer recommended for use in labour analgesia.

Cardiovascular collapse/ CNS ratio: The ratio of the dosage required for irreversible cardiovascular collapse and the dosage that will produce CNS toxicity (convulsion). The CC/CNS ratio is lower for Bupivacaine (3.7±0.5) than for Lignocaine (7.1±1.1).

Many reports are published about the toxicity of Bupivacaine. Continuous infusions of Bupivacaine intravenously in conscious man and dog showed that up to 2.1 mcg/ml Bupivacaine was not toxic to conscious man, while 4 mcg/ml of Bupivacaine can cause convulsion in conscious man.

The plasma concentration of Bupivacaine of 5.1 mcg/ml to 5.4 mcg/ml causes convulsion and it ceased when the concentration decreased to 3.5mcg/ml.

The threshold for CNS toxicity is 0.24 mcg/ml of free Bupivacaine, 3.5 mcg/ml for total Bupivacaine concentration in a patient with normal AAGP (Alpha1 acid glycoprotein).

## Epinephrine:

It holds a special place as an adjuvant of local anaesthetic solutions. The vasodilator property of local anaesthetics enhances their absorption, addition of a vasoconstrictor decreases vascular absorption, resulting in the drug being in contact with nerve tissue for a longer period of time thus prolonging the period of conduction blockade.

It has alpha and beta stimulating property. It is rapidly metabolized in the body, first by catechol-o-methyl transferase (COMT) to metanephrine and then by monoamino oxidase (MAO) in the liver and blood stream.

Patients on antihypertensive drugs, like alpha methyldopa, MAO inhibitors, tricyclic antidepressants the systemic effect of Epinephrine may be potentiated.

In epidural anaesthesia it increases the chance of “Anterior spinal artery syndrome” a rare complication of epidural block.

Local anaesthetic solutions usually contain Epinephrine, at a concentration of 5 microgram/ml i.e 1 in 2, 00,000 dilutions.

# METHODOLOGY

A randomized clinical trial was conducted on 220 patients undergoing lower abdominal and lower limb surgery under epidural anaesthesia from December 2007 to 2008.

Using previously conducted study results and standard statistical formula the sample size was calculated. We considered alpha error as 0.05 and β error as 0.2, with the power of 80% and p1 as 98 and p2 as 90.

After obtaining institutional ethical committee clearance, informed consent was taken from all the patients. This study was carried out in K.L.E's Dr.Prabhakar Kore hospital Belgaum.

**Inclusion criteria:**

220 patients belonged to ASA grade I and II categories who were scheduled for general surgery, orthopaedic or gynaecological operations in the age group of 15-65yrs were included. All these patients required central neuraxial blockade below umbilicus.

**Exclusion criteria:**

1. Patient refusal.
2. Infection at the site of epidural needle insertion.
3. Severe spine abnormalities like spina bifida, meningocele
4. Vertebral implant
5. Raised intracranial tension.
6. Known case of hydrocephalus

7. Severe convulsive disorders.

Apart from routine hemogram, chest x-ray and ECG were also done in patients over 40yrs of age. The patient's height and weight were noted. The patients were randomly divided into two groups, A and S, 110 in each group using a computerized randomization.

Group A: Loss of resistance to air technique used for identification of epidural space.

Group S: Saline infusion technique using micro-drip infusion set used for identification of epidural space.

Base line heart rate, blood pressure and respiratory rate were recorded. A large bore intravenous cannula (18G) was secured. All the patients were preloaded with crystalloids 10-15 ml/kg prior to the block.

**Technique of epidural blockade:**

**In group A:** Loss of resistance to air technique

The patient was positioned in left lateral position on the operation table, with a pillow under the head. With all aseptic precautions, namely painting and draping the area L2-L3 or L3-L4 interspace was chosen. The skin, subcutaneous tissue, supraspinous and interspinous ligaments were infiltrated with 2 ml plain Lignocaine with 23 gauge needle. After three minutes the skin was punctured with a 16 gauge needle up to the subcutaneous tissue in the midline at the chosen interspace. The 18G Tuohy's needle with stylet was introduced through the skin puncture and advanced in the midline with a slight cephalic inclination till it reached interspinous ligament and the stylet was removed. A 10

ml loss of resistance syringe filled with 5 ml of air was fitted to the hub of Tuohy's needle. The needle was held by Bromage grip with the left hand while right hand maintains continuous pressure on the piston of the syringe, as the needle was being advanced. As soon as ligamentum flavum was pierced and the epidural space entered, there was sudden loss of resistance to the syringe plunger and the needle advancement stopped. The syringe was disconnected and after ruling out any out flow of CSF and blood, 3 ml of Injection 2% Lignocaine with Epinephrine (1:2, 00,000) was injected. Heart rate for five minutes and development of spinal anaesthesia were monitored. Absence of tachycardia rules out possible intravenous placement of epidural needle tip. The main local anaesthetic mixture was then injected.

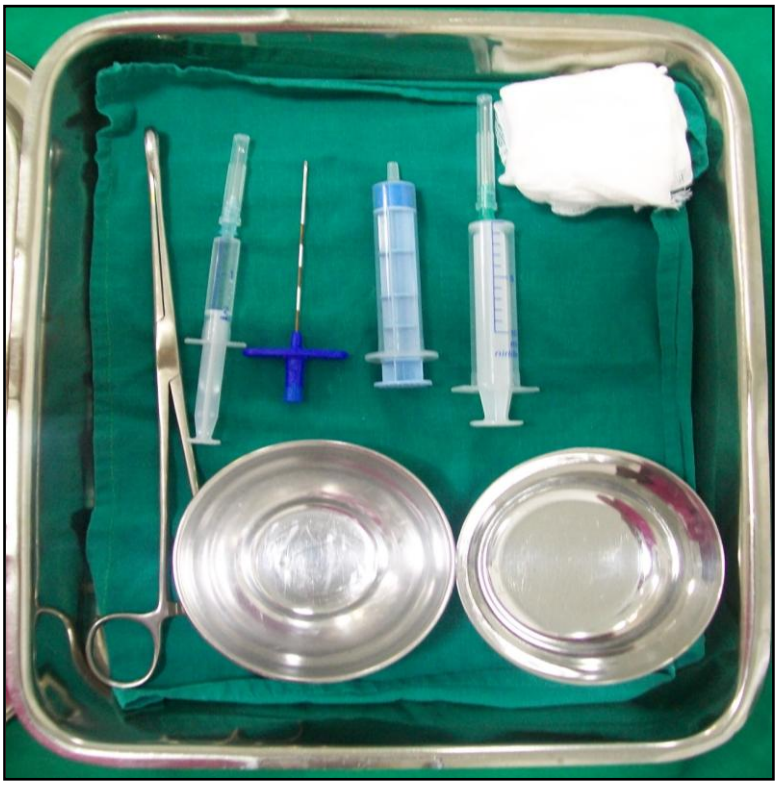


**Figure 5: Epidural kit for loss of resistance to air technique**

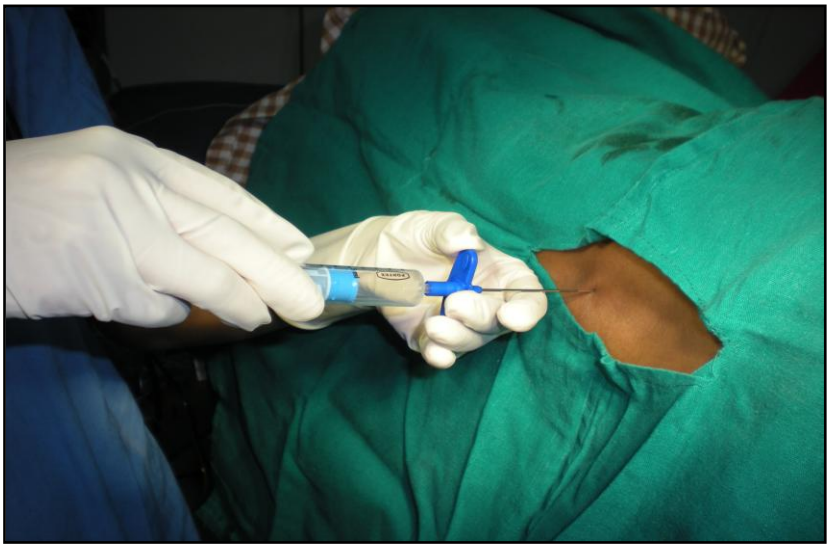


**Figure 6: LOR technique using air as a medium**

**In group S:** Saline infusion technique.

The patient was positioned in the left lateral position with a pillow under the head. After preparing the back with proper aseptic precautions, a18 gauge Tuohy epidural needle was inserted in the midline at the desired lumbar level. As in the loss of resistance to air technique, the needle was advanced through the skin, subcutaneous tissue, supraspinous ligament and interspinous ligament using both the hands. The stylet was then removed and the sterile end of micro-drip infusion tubing was carefully connected to the hub of the needle. The drip was then left open. The infusion sometimes drips slowly when the tip of the needle is in the loose interspinous ligament. When the needle engaged the ligamentum flavum, marked resistance was felt and the infusion always stopped running. The needle was then advanced very slowly through the ligamentum flavum. [(3)]

As soon as the bevel of the needle entered the epidural space, the infusion started to flow, thus identifying the epidural space. Entry into the space was usually associated with a "give way" feeling and advancement of the needle is stopped immediately. The infusion was then detached from the needle. [(3)]

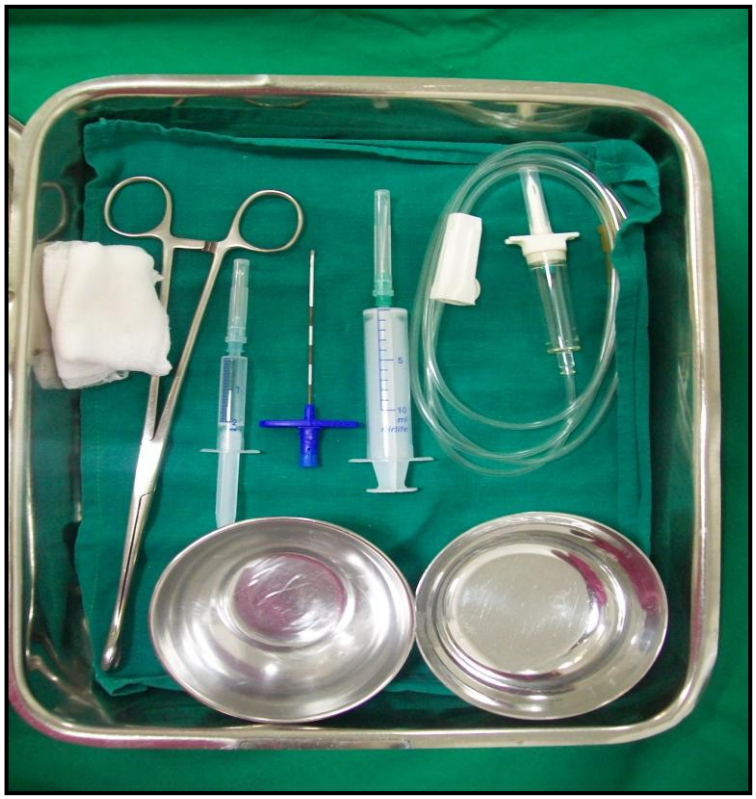


**Figure 7: Epidural kit for saline infusion technique using micro-drip infusion set**

**Figure 8: Saline infusion technique using micro-drip set**

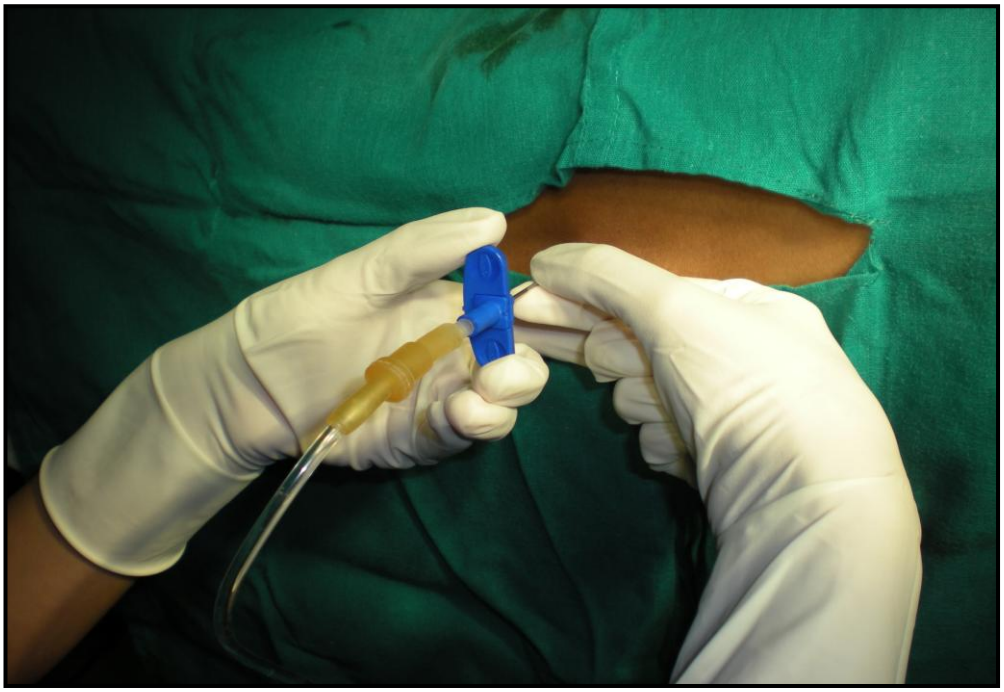


**Figure 9: Saline infusion technique. (Note: both hands are used to grip the needle.)**

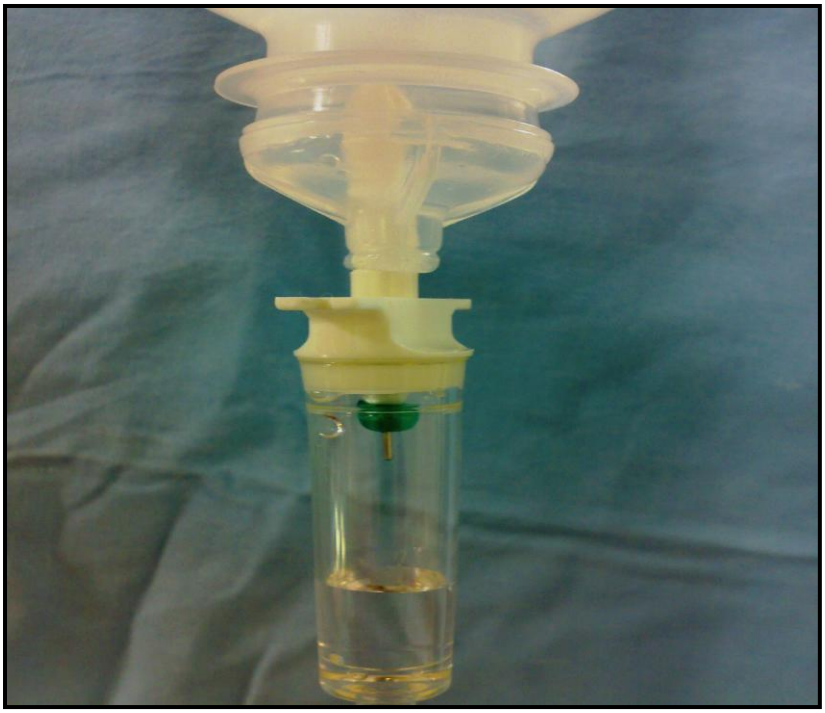


**Figure 10: Before entering into epidural space. (Note: that no fluid runs in the chamber.)**

**Figure 11: After entering into epidural space. (Note: continuous fluid runs in the chamber.)**

After ruling out any out flow of CSF and blood, 3ml of Injection 2% Lignocaine with Epinephrine (1:2, 00,000) was injected. Heart rate for five minutes and development of spinal anaesthesia were monitored. Absence of tachycardia rules out possible intravenous placement of epidural needle tip. The main local anaesthetic mixture was then injected. Following injection in both groups the patient was placed in supine position.

The following parameters were then evaluated:

1) Successfulness in identification of epidural space:

- No dripping of CSF.
- No increase in heart rate (above 20% of the basal) on administration of 3 cc of 2% Lignocaine with Epinephrine (1 in 2, 00,000) during monitoring for 5 minutes.
- Adequate sensory blockade (i.e loss of sensation to pin prick with 22G hypodermic needle in the anterior axillary line) and adequate motor blockade (i.e by Grade 3 Bromage scale).

**Bromage scale:**

- Grade 0- No impairment of movement of leg and feet.
- Grade 1- Barely able to flex knees, no impairment of movement of feet.
- Grade 2- Unable to flex knees, barely able to move feet.
- Grade 3- Unable to move knees and feet.

2) Quality of analgesia: The quality of anaesthesia is judged good if there is no additional requirement of intravenous analgesics or any intravenous anaesthetic drugs during intraoperative period.

Depending upon requirement of level of block and duration of the surgery the epidural anaesthesia was induced with a compound mixture of Lignocaine (2%) with a maximum

dose of 5 mg/kg body weight and Bupivacaine (0.5%) with a maximum dose of 3 mg/kg body weight in 1:4 ratios by weight.

As for lumbar region, 1.6 ml/segment (adjusted to body weight) the mixture was injected.

The intraoperative hypotension (mean arterial blood pressure < 20%) below baseline was treated with Ephedrine 5-10 mg IV and bradycardia (heart rate < 50/ min) was treated with Atropine 0.6mg I.V. Intra operative blood loss was replaced with crystalloid fluids at a 3:1 ratio or allogenic blood transfusion at a 1:1 ratio.

# RESULTS

The present study was undertaken in 220 ASA grade I and grade II patients of either sex aged 15 to 65 years scheduled for general surgery, orthopaedic or gynaecological surgeries. All patients enrolled completed the study.

**Table No. 1: Gender Distribution**

| | Male | Female | Total |
|---|---|---|---|
| Saline | 61 | 49 | 110 |
| Air | 55 | 55 | 110 |
| $X^2$=0.656 | DF=1 | P=0.418 | |

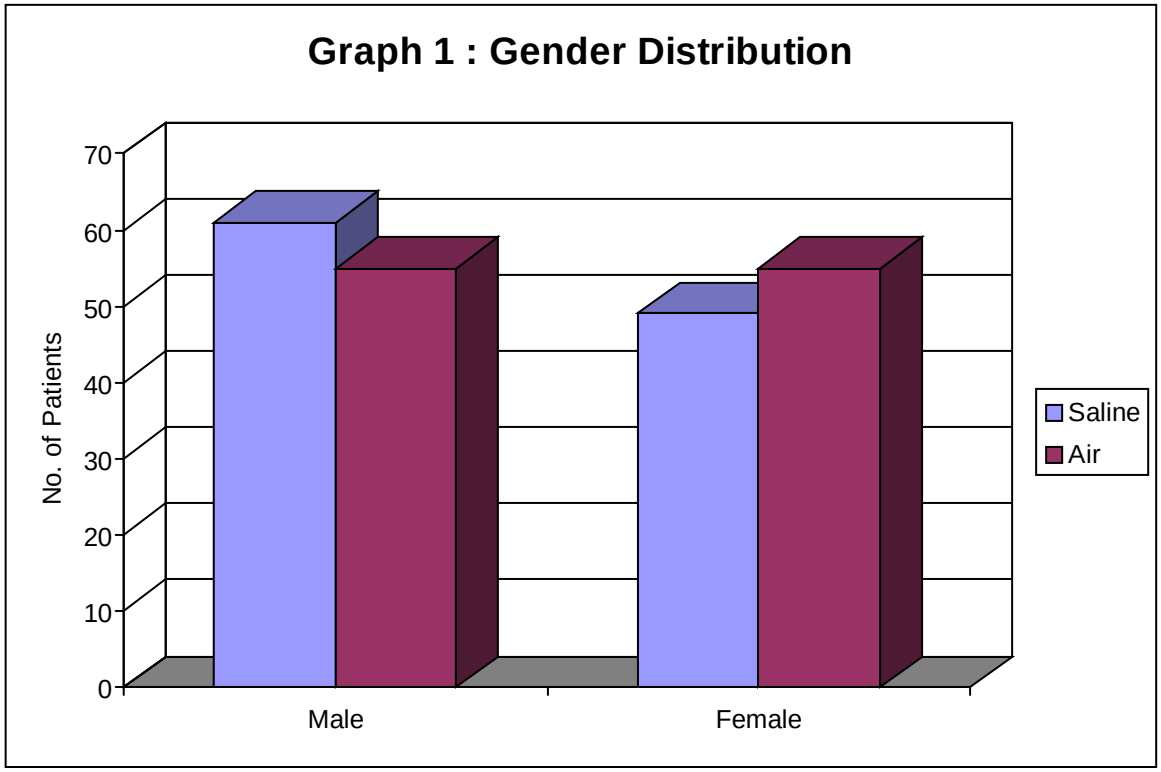
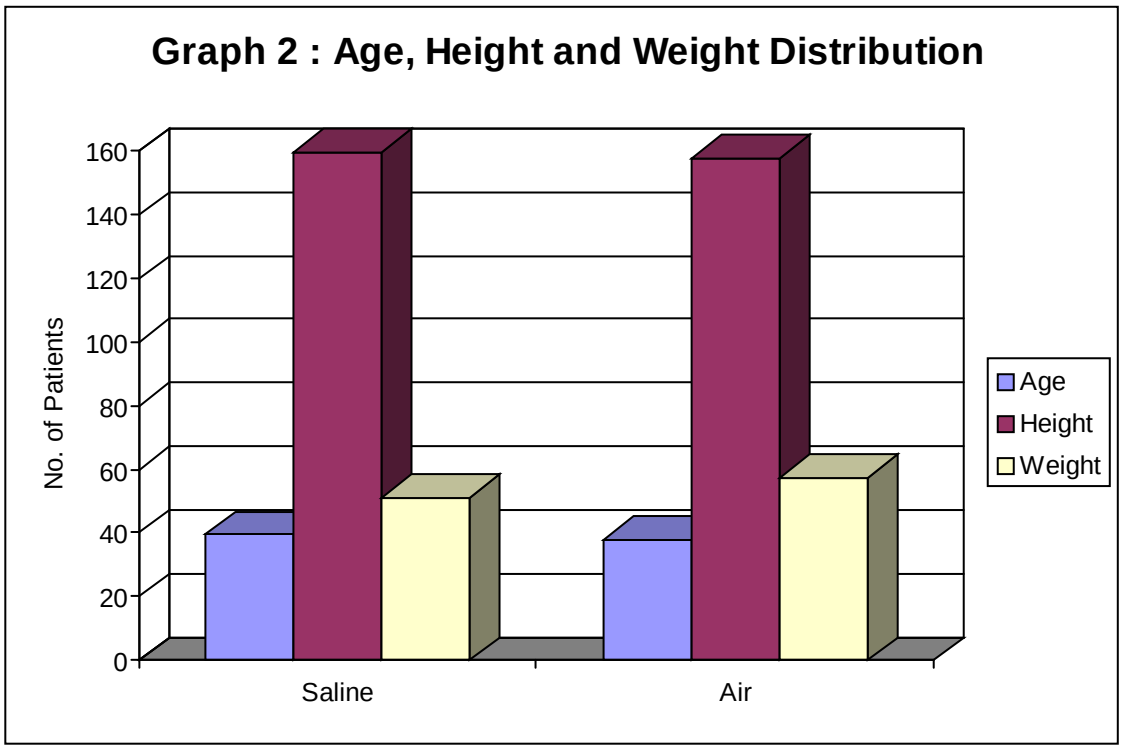


**Table No. 2: Age, Height, Weight distribution (Mean ±S.D)**

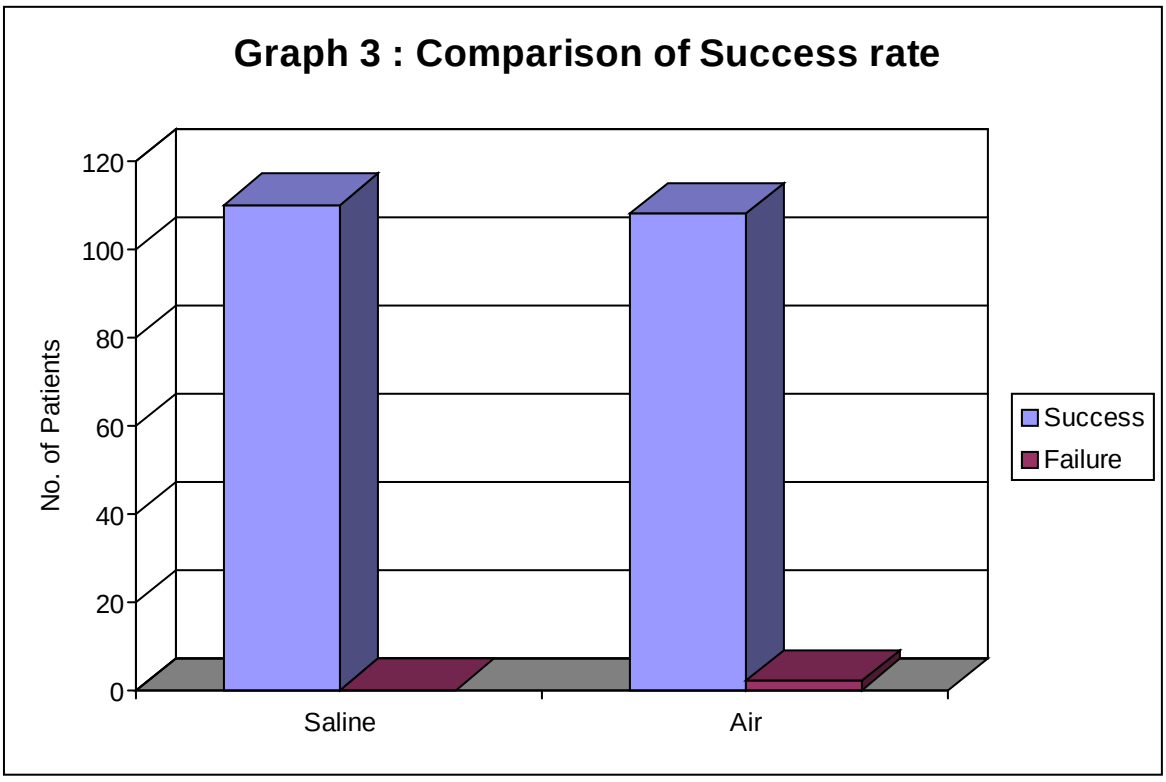
| | AGE | HEIGHT | WEIGHT |
|---|---|---|---|
| **Saline** | 39.5±13.16 | 159.8±7.85 | 51±10.4 |
| **Air** | 38±11.67 | 158±6.91 | 57.5±8.46 |
| DF=218 | t=0.927<br>p=0.355 | t=1.801<br>p=0.073 | t=0.391<br>p=0.696 |




The demographic data with respect to gender age, height and weight were comparable between the two groups as shown in table 1 and 2.

**Table No. 3: Comparison of success rate**

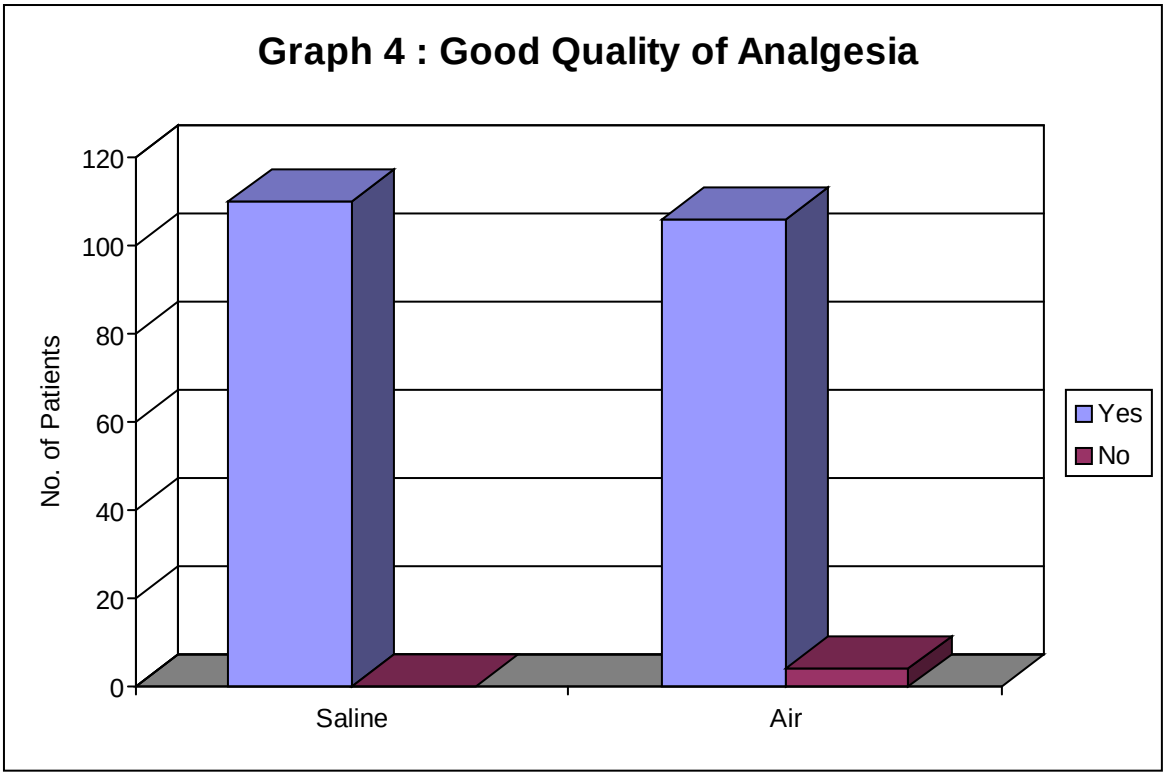
| OUTCOME | | | |
|---|---|---|---|
| **GROUP** | **SUCCESS** | **FAILURE** | **TOTAL** |
| **SALINE** | 110 | 0 | 110 |
| **AIR** | 108 | 2 | 110 |
| Fisher's exact test , p=0.498 | | | |




There is no significant difference in the success rates for identification of epidural space in both the groups.

**Table No. 4: Good quality of analgesia**

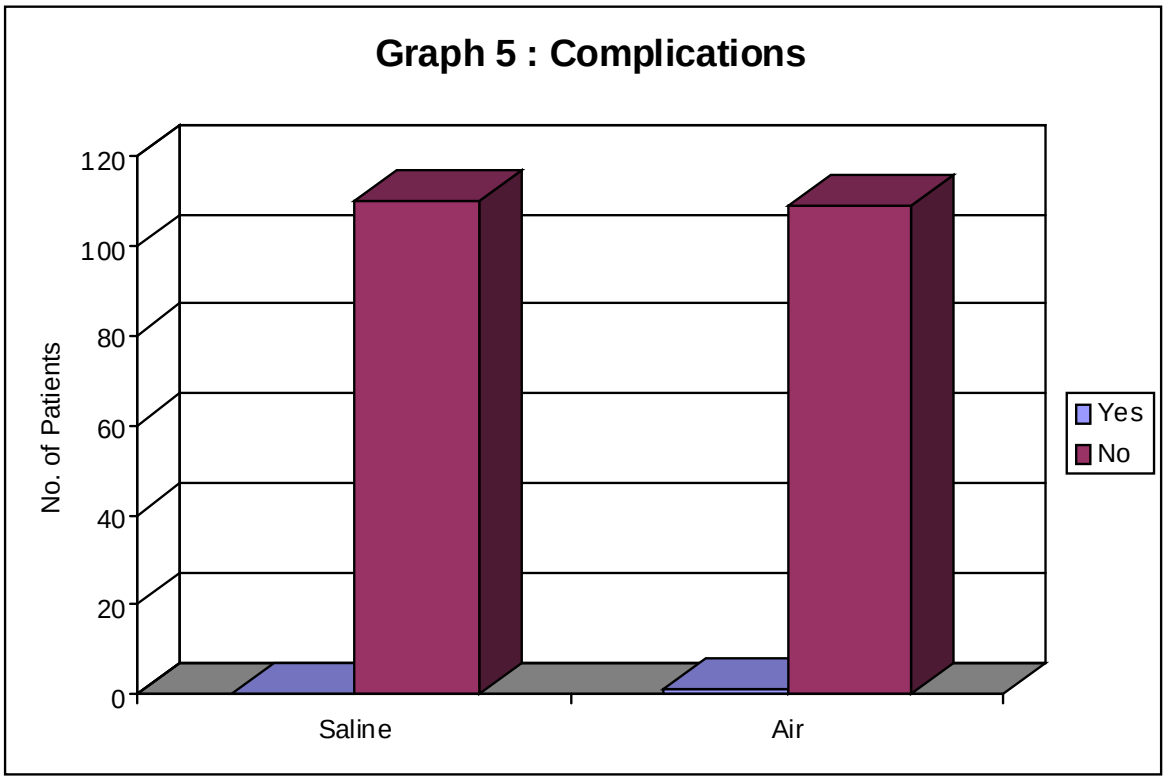
| GOOD QUALITY OF ANALGESIA | | | |
|---|---|---|---|
| **GROUP** | **YES** | **NO** | **TOTAL** |
| **SALINE** | 110 | 0 | 110 |
| **AIR** | 106 | 4 | 110 |
| Fisher's exact test , p=0.122 | | | |




There is no statistically significant difference in the quality of analgesia in both the group.

**Table No. 5: Complications**

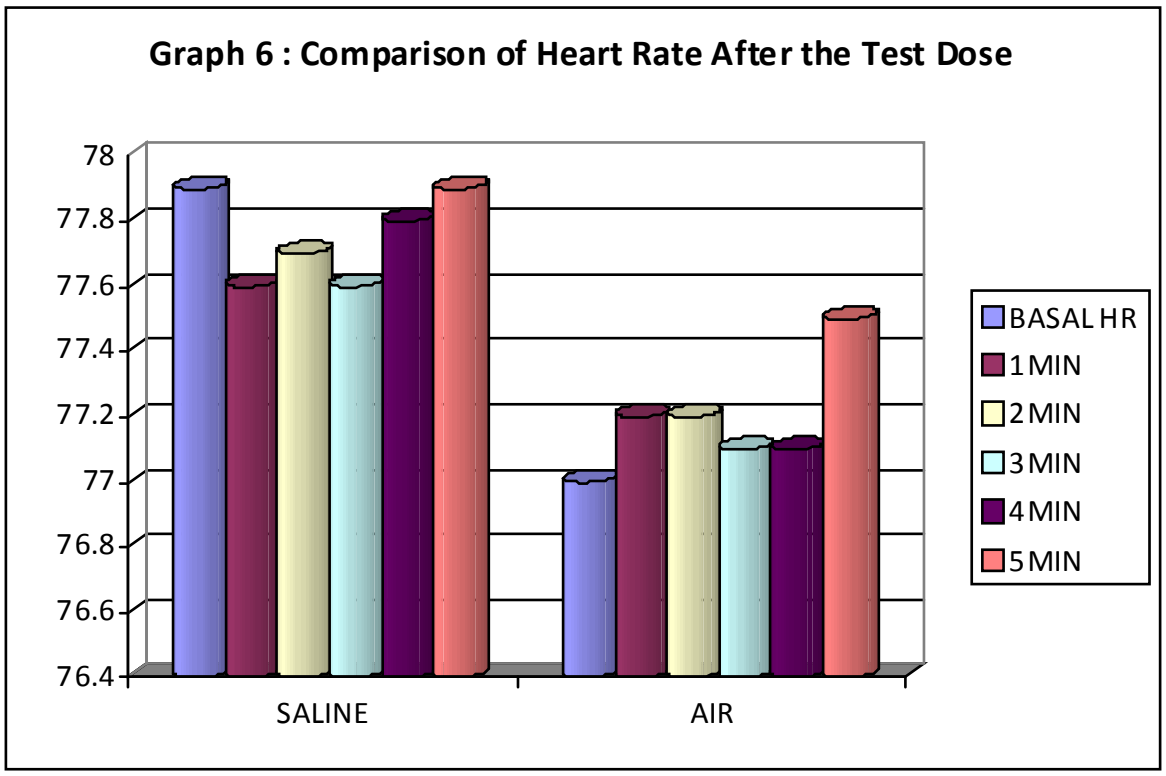
| COMPLICTIONS | | | |
|---|---|---|---|
| **GROUP** | **YES** | **NO** | **TOTAL** |
| **SALINE** | 0 | 110 | 110 |
| **AIR** | 1 | 109 | 110 |
| Fisher's exact test , p=1 | | | |




There is no statistically significant difference in the complications occurring in both the groups.

**Table No. 6: Comparison of heart rate changes after the test dose (Mean ±S.D)**

| COMPARISON OF HEART RATE AFTER THE TEST DOSE | | | | | | |
|---|---|---|---|---|---|---|
| **GROUP** | **BASAL HR** | **1MIN** | **2MIN** | **3MIN** | **4MIN** | **5MIN** |
| **SALINE** | 77.9±7.46 | 77.6±7.06 | 77.7±7.02 | 77.6±7.42 | 77.8±7.51 | 77.9±7.19 |
| **AIR** | 77±7.74 | 77.2±7.74 | 77.2±7.38 | 77.1±7.35 | 77.1±7.41 | 77.5±7.19 |
| **t** | 0.833 | 0.437 | 0.468 | 0.365 | 0.750 | 0.384 |
| **DF** | 218 | 218 | 218 | 218 | 218 | 218 |
| **p** | 0.406 | 0.663 | 0.640 | 0.715 | 0.454 | 0.701 |




There is no significant rise in the heart rate after the injection of test dose in both the groups.

# DISCUSSION

Abdominal and lower limb surgeries are the most commonly performed surgeries. As regional anaesthesia has various advantages over general anaesthesia, it is preferred whenever applicable. Many of the lower limb and abdominal surgeries can be performed under central neuraxial blockade. Amongst the central neural blockades epidural block is preferred over subdural block in day to day clinical anaesthetic practice. This is mainly due to less rapid hemodynamic changes, less danger of neurological sequelae, absence of postdural puncture headache and availability of rapid onset safer local anaesthetics. No other neural blockade techniques are used as extensively in each of the fields of surgical anaesthesia, obstetric anaesthesia and diagnosis and management of acute and chronic pain.

The success of an epidural block mainly depends upon the correct identification of epidural space. So, identification of the epidural space is an important step during epidural block.

There has always been a debate on a better and effective technique for identification of epidural space; newer techniques have developed over the years. An ideal technique should be easy to perform, have a high success rate in identifying epidural space, should not affect the onset of action, with no patchy or unilateral block and should have minimal chances of puncturing the dura.

Among the various techniques used the most common method for determining entry into the epidural space is the loss of resistance technique. During this technique, 2

to 5 ml of air is drawn into the syringe and continuous or intermittent pressure is applied to the plunger as the epidural needle is advanced toward the epidural space. On entry into epidural space, the air is injected due to a sudden loss of resistance. Air is most widely used medium in the loss of resistance technique, but this technique is not free from disadvantages.

In view of the advantages and disadvantages of the most commonly used techniques for identifying epidural space, we conducted a clinical study to compare the success rate and quality of analgesia with loss of resistance to air with that of saline infusion technique using micro-drip infusion set in patients undergoing lower abdominal and lower limb surgeries.

As the saline infusion technique has not yet been studied in adult patients and not compared with the most commonly used loss of resistance to air technique, we prospectively collected the data to compare and evaluate the success rate, quality of analgesia and complications in both the groups.

Two hundred twenty ASA I and II patients were randomly divided into two groups. Patients scheduled for elective general surgical, gynaecological and orthopaedic operations were included. All these patients required neural blockade below the level of umbilicus. In Group A (n=110) loss of resistance to air technique was used and for Group S (n=110) saline infusion technique using micro-drip set was used.

In our study the demographic data were comparable with respect to age, gender, weight and height in both the group.

Baseline heart rate was recorded in both the groups. Mean value of heart rate was 77.9±7.46 in saline group and in air group it is 7.7±7.74 with p value of 0.406 suggestive of no significant difference in both groups.

In this study the success rate for identification of epidural space was carried out. The success rate in saline group was found to be 100%. This is consistent with a study conducted by M.Yashshita and M.Tsuji, in which the success rate in identification of epidural space by saline infusion technique in children of different age groups was between 97-100%.[29]

The success rate in identification of epidural space using loss of resistance to air was found 98.18% in our study.

We observed 100%, of good quality of analgesia in saline group. Even though some studies show that the use of saline in epidural space does affect the quality of analgesia due to the dilutional effect of the local anaesthetic in the epidural space, we observed 100% good quality of analgesia with saline infusion technique. This may be explained by use of micro-drip infusion set which might have led to entry of minimal saline into the epidural space and lesser amount of dilution.

Good quality of analgesia in air group was about 96.36%. Out of 110 cases in Group A, two cases had patchy block to which we administered intravenous opioids to overcome the pain initially but later on there was no additional requirement of opioids. This could be due to the formation of bubbles in epidural space as air was used as a medium for this technique. The possibility of bubbles of air collecting within the epidural space following use of air for loss of resistance technique was confirmed by Dalens,

Bazin and Haberer. [8] A study in which 12 ml of air was injected into the epidural space of patients in labour reported no segmental defect in subsequent analgesia, but no details were given. [30] The results of our study are similar as they do not show that using air in loss of resistance technique leads to persistently unblocked segments. We experienced use of air has lead to patchy analgesia following initial injection of local anaesthetics.

Inspite of apparently clear signs, failure to obtain effective epidural block was not infrequent, especially in the elderly patients. In our study we experienced a "false" loss of resistance in two patients of air group which led to a false positive result. This may be due to cavity formation within the interspinous ligament which was described by Rissanen. [31] Degeneration of interspinous ligament with resultant cavity formation is frequent in elderly patients. In our study, false positive loss of resistance was observed in adult patients. The cavitation of interspinous ligament is usually asymptomatic and radiologically there is no sign of cyst formation. Rissanen in his study found that 27% of adults had interspinous cavitation. This could explain the false loss of resistance encountered even in adult population. [31] General anaesthesia was administered in two patients, as epidural block was unsuccessful.

There was no bloody tap in our study. Many studies found no difference in the incidence of intravascular cannulation when loss of resistance to air was compared with loss of resistance to normal saline. The role of fluid volume injected before catheter insertion has made clearer by Shmuel Evron et.al [24] who demonstrated 10 ml of normal saline decreases the incidence of venous cannulation, but at the expense of possible impairment of the quality of analgesia. He also concluded that predistension of epidural space with 5 ml of normal saline was associated with lower incidence of intravascular

catheter insertion and fewer unblocked segments. In a study performed by Mehmet Cesur et. al [32] it was revealed that, giving a single injection dose via the epidural needle before catheter placement improves the quality of epidural anaesthesia and reduces catheter related complications. Thus saline infusion technique using micro-drip infusion set also reduces the risk of such an important complication and will improve several aspects of epidural analgesia.

The loss of resistance technique with air is associated with several adverse effects. For example in our study, the incidence of accidental dural puncture in air group is 0.9% as compared to zero percent in the saline group. Even though this difference is statistically insignificant, practically it is important to prevent dural puncture and post dural puncture headache. This difference may be explained by a good stability over the epidural needle in saline infusion technique as both the hands were used to grip the needle and its controlled insertion while localizing epidural space. Therefore, we believe that the loss of resistance technique with air is less reliable for locating the epidural space. Air in the epidural space appears to increase the occurrence of post dural puncture headache. The association between pneumocephalus and post dural puncture headache is clear. [33]

In conclusion saline infusion using micro-drip infusion set is a better technique for correct identification of epidural space. Our results demonstrate a reduced incidence of unblocked segments and complications when compared to loss of resistance to air technique.

**Future scope of the study:**

1. It is important to compare the onset of the epidural block, since each technique has effect on the onset of the block; this aspect needs to be studied in detail.

2. A change in the quality of analgesia by epidural catheter insertion or single shot epidural technique needs to be studied.

3. In group S, we have not calculated how much amount of saline is infused during identification of epidural space. As the quality of analgesia is 100% which can explain possible entry of minimal amount of saline into epidural space. But further study is required to calculate the amount of saline that is infused and its effect on quality of analgesia.

# CONCLUSION

In conclusion, this study suggests that saline infusion technique using micro-drip infusion set, which is a modification of older techniques, is a better choice when compared to loss of resistance to air technique for the identification of epidural space. A larger randomized study, however confirms this suggestion.

# SUMMARY

The present study was conducted on 220 patients of ASA grade I and II patients in the age group between 15- 65 years, scheduled for elective lower abdominal and lower limb surgeries. These patients were divided into Group A and S in which loss of resistance technique with air as medium and saline infusion technique using micro drip set for the identification of epidural space respectively.

This study was conducted after institutional ethical committee approval and informed consent was obtained from all patients. Baseline heart rate, blood pressure were noted. Under strict aseptic condition the epidural space was identified using 18 gauge Tuohy's epidural needle. Depending upon computer generated randomization either of the technique, was used to identify the epidural space.

The success rate of identification of epidural space, quality of anaesthesia and complications that occurred during the procedures was studied.

In this study there was no statistically significant difference between success rate for identification of epidural space, quality of analgesia and complications was observed.

Before making a premature conclusion that either of the technique is the cause of the failure to identify the epidural space or the cause of complications, it might also be important to consider the insertion skills of the provider and the ease or difficulty of the insertion procedure.

Thus either of the two techniques for identification of epidural space can be recommended for routine practice depending upon the choice and convenience of anaesthesiologist.

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# INFORMED CONSENT

You Mr/Mrs/Ms. _____ I.P No. _____ are asked to participate in the research study titled **"COMPARISON OF LOSS OF RESISTANCE TO AIR TECHNIQUE AND SALINE INFUSION TECHNIQUE USING MICRO - DRIP SET FOR IDENTIFICATION OF EPIDURAL SPACE - A ONE YEAR RANDOMIZED STUDY."**

A one year randomized trial conducted by Dr. Akshaya. N. Shetti, post graduate student. Department of Anaesthesiology, JNMC, Belgaum. You are eligible after looking into inclusion criteria. You may read this form and ask any questions you may have before agreeing to participate.

**RESEARCH BEING DONE**

To compare the two techniques with respect to success rate in identifying epidural space and quality of analgesia.

**Purpose of the research**

To compare the two different techniques for locating epidural space , where in, we study which technique is more successful in identifying epidural spaceand in which technique the quality of analgesia is good, so that in future we can proceed with one of the above techniques which gives high success in identifying epidural space and also which gives good quality of analgesia.

**Procedures Involved:**

You will be randomly allocated either into study Group A or Group S. If you are in Group A then your epidural space will be identified by loss of resistance to air technique and if you are in Group S then your epidural space will be identified by using saline infusion technique. The success rate and the quality of analgesia will be compared.

**Potential Risks and discomforts:**

- Air embolism, pneumocephalus, with air technique and dural puncture in both techniques.

**Benefits of taking part in this research:**

- We can avoid general anaesthesia
- Good quality of analgesia

**Other options:**

You can go for general anaesthesia or spinal anaesthesia if there are no obvious contraindications for these.

**Decline from participation:**

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

**New information:**

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

**Privacy and confidentiality:**

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

**Injury as a result of participation**

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

**Costs of participation in research**

Participation is free of cost

**Reimbursement for any expenses for participation in research**

No Reimbursement for any of your expenditures.

**Withdrawal or be removed**

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

**Whom to contact**

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

- DR.V.D.PATIL. Principal, JNMC, Belgaum.
- DR.M.G.DHORIGOL M.D. Professor, Dept of Anaesthesiology, JNMC Belgaum. (Ph: 9844778096).
- DR.AKSHAYA.N.SHETTI, Post graduate student in Anaesthesiology, JNMC, Belgaum. (Ph: 9742502464).

Signature of the participant or legally authorized person

Participant name:

Signature / thumb impression:

Witness name:

Signature / thumb impression:

Date:

Place:

# PROFORMA

**Title: "COMPARISON OF LOSS OF RESISTANCE TO AIR TECHNIQUE AND SALINE INFUSION TECHNIQUE USING MICRO - DRIP SET FOR IDENTIFICATION OF EPIDURAL SPACE- A ONE YEAR RANDOMIZED STUDY."**

Patient's Name: I.P.No:

Age: Weight:

Height:

Sex: Date of operation:

Occupation: Address:

Anaesthesiologist:

**PRE-ANAESTHETIC EVALUATION:**

**Chief complaints:**

**Past History:**

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

b) Drug therapy

c) Previous exposure to anaesthesia

**Family history:**

**General Physical examination:**

Pallor / Icterus / Clubbing / Lymphadenopathy / Oedema

P.R: B.P:

R.R:

**Musculoskeletal system examination:**

Jaw movements: Teeth:

Airway assessment: Spine:

**Systemic examination:**

a. R.S: b. CNS:

c. C.V.S: d. Per abdomen:

**Investigations:**

**Hb%: Total count: Differential count:**

**Bleeding time:**

**Clotting time:**

**PT: aPTT: INR:**

**Urine routine:**

**Any others:**

**Preoperative physical status: ASA grade I II III IV V**

**Inclusion criteria**

i. Patients of either sex aged between 15-65 years undergoing lower abdominal surgery.

ii. Patients undergoing lower limb surgery.

**Exclusion criteria**

1. Patient refusal
2. Infection at the site of epidural needle insertion
3. Severe spine abnormalities like spina bifida, meningocele
4. Vertebral implant
5. Raised intracranial tension
6. Known case of hydrocephalus
7. Severe convulsive disorders

**Diagnosis**:

**Proposed surgery:**

Patients will be allocated by computer generated randomization into group A and groupS. On the day of surgery I.V line is secured in a peripheral vein.

**Preoperative baseline:**

Heart rate:

Blood pressure:

**Monitors attached:**

Pulse oximeter:

Noninvasive blood pressure:

ECG:

18G Tuohy needle used for the study. Identification of epidural space is done in Group A by loss of resistance to air technique and in group S saline infusion using sterile micro-drip set. Once the space is identified then a test dose, 3ml of Inj 2% Lignocaine with Epinephrine (1in 2, 00,000) administered.

The patients of both the groups will be observed immediately after giving the above mentioned drug and heart rate will be recorded for five minutes with the interval of 1min. A change in the heart rate above 20% of the basal rate, dripping of cerebrospinal fluid or blood through needle will be considered as failure.

| Group | Time (Interval- 1minute) | Heart rate |
|---|---|---|
| | 1min | |
| | 2min | |
| | 3min | |
| | 4min | |
| | 5min | |

Intraoperative:

1. Is identification of epidural space successful? (Defined successful when no dripping of CSF through the needle, no rise in the heart rate above 20% of the basal on administration of test dose, adequate sensory and motor blockade.)
    a. Yes
    b. No
2. Is the quality of analgesia provided good? ( Defined good if there is no additional requirement of intravenous analgesics or an intravenous anaesthetic drugs during intraoperative period)
    a. Yes
    b. No
3. If any complications have occurred then mention them

Signature of the staff in charge:

# SALINE GROUP

| S/N | I.P. No | Age (Yrs) | Sex | Height (cm) | Weight (kg) | Operation | H.R (min)B | H.R (min) | H.R (min) | H.R (min) | H.R (min) | H.R (min) | Successful | Good Quality of Analgesia | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 251129 | 23 | m | 170 | 55 | DCP of lt tibia | 86 | 84 | 80 | 86 | 82 | 80 | y | y | nil |
| 2 | 250284 | 63 | m | 169 | 65 | meshplasty | 84 | 82 | 84 | 83 | 80 | 78 | y | y | nil |
| 3 | 253082 | 19 | m | 158 | 38 | secondary suturing | 82 | 80 | 82 | 80 | 82 | 81 | y | y | nil |
| 4 | 253210 | 32 | f | 163 | 58 | BAT | 73 | 74 | 74 | 72 | 70 | 73 | y | y | nil |
| 5 | 253119 | 48 | f | 166 | 54 | B/L varicose vein stripping | 70 | 68 | 68 | 70 | 66 | 69 | y | y | nil |
| 6 | 253032 | 28 | f | 158 | 40 | appendicectomy | 80 | 82 | 80 | 84 | 82 | 80 | y | y | nil |
| 7 | 254070 | 28 | m | 160 | 50 | debridement of wound lt leg | 86 | 84 | 82 | 84 | 88 | 78 | y | y | nil |
| 8 | 254870 | 60 | m | 168 | 56 | debridement | 86 | 80 | 82 | 78 | 86 | 84 | y | y | nil |
| 9 | 254789 | 50 | f | 168 | 50 | BAT | 86 | 84 | 83 | 84 | 88 | 80 | y | y | nil |
| 10 | 256196 | 45 | m | 162 | 45 | Rt inguinal hernia repair | 76 | 74 | 72 | 70 | 74 | 77 | y | y | nil |
| 11 | 256945 | 62 | m | 168 | 58 | Lt inguinal hernia repair | 76 | 74 | 75 | 74 | 73 | 75 | y | y | nil |
| 12 | 257350 | 30 | f | 149 | 45 | vaginal hysterectomy | 96 | 92 | 90 | 88 | 85 | 94 | y | y | nil |
| 13 | 256844 | 60 | m | 158 | 59 | Rt inguinal hernia repair | 80 | 83 | 78 | 75 | 80 | 81 | y | y | nil |
| 14 | 256950 | 36 | m | 170 | 62 | Rt inguinal hernia repair | 70 | 72 | 74 | 68 | 69 | 72 | y | y | nil |
| 15 | 257242 | 26 | m | 168 | 40 | Appendicectomy | 86 | 87 | 88 | 86 | 88 | 89 | y | y | nil |
| 16 | 256945 | 62 | m | 170 | 60 | Lt inguinal hernia repair | 88 | 86 | 84 | 87 | 82 | 85 | y | y | nil |
| 17 | 256985 | 30 | m | 168 | 45 | Appendicectomy | 68 | 70 | 68 | 67 | 69 | 70 | y | y | nil |
| 18 | 256882 | 28 | f | 170 | 42 | BAT | 80 | 82 | 84 | 82 | 85 | 84 | y | y | nil |
| 19 | 257711 | 28 | m | 166 | 43 | Appendicectomy | 86 | 84 | 82 | 84 | 82 | 80 | y | y | nil |
| 20 | 257884 | 40 | m | 162 | 60 | Rt inguinal hernia repair | 78 | 76 | 75 | 77 | 78 | 75 | y | y | nil |
| 21 | 257320 | 46 | m | 159 | 46 | BAT | 86 | 84 | 85 | 83 | 85 | 86 | y | y | nil |
| 22 | 258280 | 16 | f | 145 | 28 | Appendicectomy | 96 | 94 | 95 | 96 | 97 | 93 | y | y | nil |
| 23 | 260982 | 21 | m | 152 | 36 | appendicectomy | 80 | 78 | 80 | 76 | 78 | 76 | y | y | nil |
| 24 | 260484 | 60 | m | 158 | 50 | debridement of wound lt leg | 78 | 76 | 79 | 74 | 76 | 78 | y | y | nil |
| 25 | 258818 | 31 | m | 166 | 56 | Lt inguinal hernia repair | 80 | 82 | 83 | 84 | 87 | 85 | Y | Y | nil |
| 26 | 261260 | 48 | m | 165 | 50 | Debridement of wound lt leg | 74 | 75 | 74 | 76 | 77 | 76 | y | y | nil |
| 27 | 261252 | 37 | f | 160 | 38 | BAT | 70 | 73 | 68 | 75 | 73 | 74 | y | y | nil |
| 28 | 259924 | 28 | m | 175 | 60 | Interlocking Rt tibia | 74 | 76 | 77 | 78 | 70 | 73 | y | y | nil |
| 29 | 260456 | 38 | f | 160 | 45 | appendicectomy | 80 | 82 | 83 | 82 | 83 | 84 | y | y | nil |
| 30 | 262584 | 46 | f | 145 | 44 | abdominal hysterectomy | 66 | 68 | 70 | 64 | 65 | 66 | y | y | nil |
| 31 | 261827 | 38 | f | 168 | 68 | vaginal hysterectomy | 76 | 72 | 76 | 75 | 72 | 76 | y | y | nil |
| 32 | 261557 | 65 | m | 160 | 60 | DCP of lt femur | 70 | 74 | 76 | 73 | 72 | 70 | y | y | nil |
| 33 | 264771 | 25 | m | 170 | 68 | Rt inguinal hernia repair | 86 | 84 | 85 | 87 | 86 | 85 | y | y | nil |
| 34 | 263148 | 28 | f | 156 | 40 | appendicectomy | 78 | 77 | 80 | 69 | 70 | 70 | y | y | nil |
| 35 | 262468 | 58 | m | 170 | 68 | debridement of wound lt leg | 80 | 82 | 78 | 80 | 80 | 80 | y | y | nil |
| 36 | 264432 | 52 | m | 168 | 65 | Lt inguinal hernia repair | 86 | 84 | 82 | 84 | 87 | 86 | y | y | nil |
| 37 | 269532 | 28 | m | 167 | 57 | debridement | 80 | 79 | 80 | 80 | 81 | 82 | y | y | nil |

| S/N | I.P. No | Age (Yrs) | Sex | Height (cm) | Weight (kg) | Operation | H.R (min)B | H.R (min) | H.R (min) | H.R (min) | H.R (min) | H.R (min) | Successful | Good Quality of Analgesia | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 38 | 264820 | 28 | m | 160 | 40 | appendicectomy | 72 | 76 | 74 | 72 | 74 | 75 | y | y | nil |
| 39 | 262780 | 42 | m | 166 | 50 | haemorrhoidectomy | 64 | 66 | 67 | 64 | 67 | 68 | y | y | nil |
| 40 | 267928 | 55 | m | 175 | 60 | debridement lt foot | 86 | 87 | 88 | 87 | 86 | 87 | y | y | nil |
| 41 | 267073 | 60 | f | 155 | 49 | vaginal hysterectomy | 90 | 92 | 89 | 90 | 88 | 90 | y | y | nil |
| 42 | 270799 | 31 | m | 175 | 40 | B/L varicose vein stripping | 76 | 74 | 75 | 76 | 76 | 77 | y | y | nil |
| 43 | 271831 | 53 | f | 160 | 42 | Abdominal hysterectomy | 80 | 78 | 79 | 80 | 82 | 80 | y | y | nil |
| 44 | 269160 | 60 | f | 162 | 68 | TAH with BSO | 80 | 82 | 78 | 80 | 82 | 78 | y | y | nil |
| 45 | 273147 | 40 | m | 169 | 65 | laparotomy | 80 | 82 | 83 | 80 | 78 | 82 | Y | Y | nil |
| 46 | 276944 | 30 | f | 158 | 40 | BAT | 70 | 68 | 71 | 70 | 68 | 70 | y | y | nil |
| 47 | 273250 | 32 | f | 152 | 50 | BAT | 82 | 84 | 82 | 84 | 82 | 83 | y | y | nil |
| 48 | 266847 | 31 | f | 154 | 45 | appendicectomy | 86 | 84 | 85 | 86 | 86 | 85 | y | y | nil |
| 49 | 274806 | 38 | f | 160 | 40 | BAT | 75 | 76 | 74 | 75 | 76 | 75 | y | y | nil |
| 50 | 274404 | 47 | f | 156 | 48 | TAH with BSO | 80 | 79 | 78 | 80 | 79 | 80 | y | y | nil |
| 51 | 274547 | 38 | f | 157 | 52 | vaginal hysterectomy | 80 | 78 | 79 | 80 | 80 | 78 | y | y | nil |
| 52 | 275717 | 35 | f | 164 | 58 | vaginal hysterectomy | 74 | 73 | 75 | 76 | 77 | 78 | y | y | nil |
| 53 | 275444 | 34 | f | 160 | 64 | BAT | 68 | 67 | 69 | 66 | 67 | 68 | y | y | nil |
| 54 | 275997 | 40 | f | 168 | 58 | vaginal hysterectomy | 76 | 74 | 75 | 76 | 74 | 76 | y | y | nil |
| 55 | 275438 | 38 | f | 162 | 36 | BAT | 66 | 67 | 68 | 66 | 67 | 67 | y | y | nil |
| 56 | 276202 | 62 | f | 148 | 57 | vaginal hysterectomy | 77 | 76 | 78 | 79 | 80 | 77 | y | y | nil |
| 57 | 276933 | 45 | f | 160 | 52 | endometrial biopsy | 88 | 86 | 87 | 87 | 88 | 89 | y | y | nil |
| 58 | 276184 | 52 | m | 157 | 52 | meshplasty | 88 | 82 | 81 | 82 | 84 | 86 | y | y | nil |
| 59 | 276705 | 66 | f | 154 | 56 | meshplasty | 68 | 70 | 68 | 70 | 68 | 69 | y | y | nil |
| 60 | 277339 | 20 | f | 168 | 40 | Appendicectomy | 80 | 80 | 82 | 86 | 87 | 80 | y | y | nil |
| 61 | 276493 | 30 | f | 160 | 50 | Appendicectomy | 78 | 78 | 80 | 82 | 76 | 80 | y | y | nil |
| 62 | 277301 | 34 | f | 158 | 48 | BAT | 72 | 70 | 68 | 69 | 70 | 70 | y | y | nil |
| 63 | 277599 | 45 | m | 165 | 70 | Rt inguinal hernia repair | 90 | 92 | 90 | 89 | 90 | 91 | y | y | nil |
| 64 | 276896 | 53 | f | 155 | 57 | vaginal hysterectomy | 68 | 66 | 64 | 68 | 66 | 67 | y | y | nil |
| 65 | 277745 | 52 | f | 162 | 60 | varicose vein stripping | 68 | 64 | 66 | 68 | 64 | 67 | y | y | nil |
| 66 | 277577 | 45 | f | 168 | 40 | vaginal hysterectomy | 84 | 86 | 86 | 87 | 86 | 85 | y | y | nil |
| 67 | 277713 | 36 | f | 156 | 46 | vaginal hysterectomy | 74 | 75 | 76 | 75 | 76 | 75 | y | y | nil |
| 68 | 277079 | 65 | f | 148 | 52 | vaginal hysterectomy | 83 | 84 | 85 | 85 | 84 | 85 | y | y | nil |
| 69 | 275483 | 24 | m | 162 | 48 | varicose vein stripping | 78 | 76 | 80 | 78 | 77 | 76 | y | y | nil |
| 70 | 278230 | 20 | f | 140 | 30 | foot abscess drainage | 79 | 80 | 82 | 79 | 82 | 82 | y | y | nil |
| 71 | 278943 | 62 | m | 150 | 56 | excision of epididamal cyst | 68 | 69 | 70 | 68 | 69 | 70 | y | y | nil |
| 72 | 277820 | 26 | m | 158 | 45 | debridement | 90 | 88 | 91 | 89 | 88 | 90 | y | y | nil |
| 73 | 277945 | 40 | m | 170 | 70 | umblical hernia | 70 | 71 | 72 | 72 | 71 | 70 | y | y | nil |
| 74 | 278120 | 24 | f | 160 | 70 | Appendicectomy | 69 | 70 | 70 | 69 | 70 | 71 | y | y | nil |
| 75 | 277982 | 62 | m | 156 | 62 | DHS plating | 70 | 70 | 69 | 68 | 69 | 70 | y | y | nil |

| S/N | I.P. No | Age (Yrs) | Sex | Height (cm) | Weight (kg) | Operation | H.R (min)B | H.R (min) | H.R (min) | H.R (min) | H.R (min) | H.R (min) | Successful | Good Quality of Analgesia | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 76 | 278328 | 44 | m | 152 | 50 | biopsy of penile growth | 80 | 78 | 79 | 77 | 79 | 80 | y | y | nil |
| 77 | 278608 | 70 | m | 153 | 62 | ORIF of lt femur | 68 | 65 | 65 | 67 | 65 | 68 | y | y | nil |
| 78 | 280562 | 27 | m | 150 | 46 | varicose vein stripping | 88 | 86 | 87 | 88 | 84 | 85 | y | y | nil |
| 79 | 278826 | 34 | m | 146 | 58 | lipoma excision | 76 | 74 | 76 | 77 | 78 | 74 | y | y | nil |
| 80 | 280744 | 30 | f | 142 | 35 | appendicectomy | 80 | 78 | 80 | 82 | 78 | 79 | y | y | nil |
| 81 | 282173 | 38 | f | 143 | 35 | BAT | 76 | 77 | 78 | 79 | 80 | 78 | y | y | nil |
| 82 | 282255 | 35 | f | 154 | 46 | abdominal hysterectomy | 86 | 84 | 85 | 85 | 86 | 87 | y | y | nil |
| 83 | 284180 | 21 | m | 156 | 40 | circumcission | 76 | 76 | 78 | 79 | 75 | 76 | y | y | nil |
| 84 | 287449 | 40 | f | 158 | 58 | laparotomy | 68 | 68 | 70 | 64 | 66 | 67 | y | y | nil |
| 85 | 284670 | 24 | f | 160 | 40 | appendicectomy | 70 | 74 | 76 | 72 | 74 | 72 | y | y | nil |
| 86 | 284699 | 58 | m | 168 | 62 | meshplasty | 62 | 66 | 60 | 62 | 64 | 66 | y | y | nil |
| 87 | 284724 | 36 | f | 155 | 60 | BAT | 80 | 74 | 76 | 77 | 79 | 80 | y | y | nil |
| 88 | 287628 | 35 | m | 160 | 50 | appendicectomy | 70 | 74 | 70 | 72 | 74 | 71 | y | y | nil |
| 89 | 287750 | 26 | f | 158 | 48 | debridement | 86 | 85 | 80 | 82 | 90 | 86 | y | y | nil |
| 90 | 287021 | 32 | m | 162 | 52 | meshplasty | 76 | 76 | 78 | 76 | 78 | 77 | y | y | nil |
| 91 | 287729 | 30 | f | 152 | 46 | BAT | 78 | 76 | 77 | 78 | 75 | 77 | y | y | nil |
| 92 | 280175 | 50 | m | 156 | 46 | debridement | 86 | 88 | 86 | 90 | 87 | 88 | y | y | nil |
| 93 | 278926 | 26 | f | 148 | 36 | Appendicectomy | 84 | 87 | 88 | 84 | 87 | 87 | y | y | nil |
| 94 | 278605 | 42 | m | 146 | 48 | tangential exision of stump | 86 | 88 | 87 | 86 | 84 | 86 | y | y | nil |
| 95 | 282818 | 28 | f | 148 | 30 | Appendicectomy | 68 | 69 | 68 | 67 | 70 | 68 | y | y | nil |
| 96 | 282012 | 30 | m | 146 | 40 | I&D of perineal abscess | 96 | 94 | 93 | 92 | 97 | 98 | y | y | nil |
| 97 | 282412 | 30 | m | 172 | 56 | debridement | 80 | 81 | 82 | 83 | 84 | 82 | y | y | nil |
| 98 | 282587 | 26 | m | 148 | 26 | Appendicectomy | 70 | 71 | 72 | 72 | 73 | 72 | y | y | nil |
| 99 | 279950 | 30 | m | 160 | 48 | Appendicectomy | 74 | 74 | 72 | 73 | 74 | 75 | y | y | nil |
| 100 | 282480 | 30 | m | 158 | 48 | BAT | 80 | 80 | 78 | 81 | 81 | 82 | y | y | nil |
| 101 | 280886 | 28 | m | 160 | 52 | debridement | 76 | 76 | 74 | 76 | 75 | 76 | y | y | nil |
| 102 | 281240 | 42 | m | 164 | 68 | rt varicose vein stripping | 78 | 76 | 78 | 77 | 76 | 77 | y | y | nil |
| 103 | 282704 | 36 | m | 168 | 58 | debridement | 70 | 69 | 70 | 68 | 71 | 71 | y | y | nil |
| 104 | 280187 | 65 | m | 152 | 62 | corn excision | 88 | 88 | 86 | 88 | 87 | 90 | y | y | nil |
| 105 | 280429 | 42 | m | 150 | 46 | Appendicectomy | 76 | 76 | 78 | 76 | 74 | 76 | y | y | nil |
| 106 | 283042 | 48 | m | 170 | 70 | B/L varicose vein stripping | 69 | 70 | 70 | 71 | 72 | 70 | y | y | nil |
| 107 | 287564 | 40 | m | 168 | 67 | Rt inguinal hernia repair | 68 | 68 | 67 | 69 | 70 | 69 | y | y | nil |
| 108 | 297973 | 48 | m | 164 | 50 | haemorrhoidectomy | 70 | 68 | 70 | 68 | 69 | 68 | y | y | nil |
| 109 | 298380 | 35 | m | 168 | 62 | hydrocelectomy | 70 | 70 | 68 | 66 | 68 | 69 | y | y | nil |
| 110 | 282378 | 38 | f | 160 | 38 | BAT | 76 | 76 | 76 | 78 | 78 | 73 | y | y | nil |

## AIR GROUP

| S/N | I.P. No | Age (Yrs) | Sex | Height (cm) | Weight (kg) | Operation | H.R (min)B | H.R (min) | H.R (min) | H.R (min) | H.R (min) | H.R (min) | Successful | Quality of Analgesia | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 257281 | 22 | m | 161 | 45 | Appendicectomy | 70 | 72 | 74 | 72 | 70 | 74 | y | y | nil |
| 2 | 253109 | 30 | f | 156 | 53 | BAT | 86 | 88 | 84 | 82 | 80 | 84 | y | y | nil |
| 3 | 253107 | 30 | f | 162 | 48 | BAT | 76 | 74 | 72 | 76 | 74 | 72 | y | y | nil |
| 4 | 253189 | 28 | f | 170 | 40 | BAT | 68 | 70 | 72 | 68 | 66 | 70 | y | y | nil |
| 5 | 253108 | 28 | f | 168 | 46 | BAT | 76 | 78 | 76 | 78 | 77 | 80 | y | y | nil |
| 6 | 253430 | 24 | f | 164 | 59 | sinus over right thigh for biopsy | 88 | 90 | 86 | 84 | 83 | 86 | y | y | nil |
| 7 | 254972 | 40 | f | 156 | 55 | Abdominal hysterectomy | 86 | 83 | 84 | 80 | 78 | 78 | y | y | nil |
| 8 | 255012 | 32 | f | 145 | 55 | Abdominal hysterectomy | 80 | 82 | 78 | 80 | 79 | 80 | y | y | nil |
| 9 | 254980 | 30 | f | 168 | 52 | B/L Abdominal tubectomy | 90 | 82 | 78 | 77 | 76 | 89 | y | No, Patchy block | nil |
| 10 | 254988 | 28 | m | 170 | 55 | debridement &skin grafting | 74 | 73 | 72 | 70 | 71 | 73 | y | y | nil |
| 11 | 250290 | 51 | f | 162 | 62 | vaginal hysterectomy | 70 | 72 | 71 | 74 | 73 | 76 | y | y | nil |
| 12 | 251824 | 37 | f | 149 | 52 | appendicectomy | 90 | 86 | 88 | 89 | 90 | 88 | y | y | nil |
| 13 | 256930 | 29 | f | 164 | 35 | BAT | 86 | 84 | 83 | 82 | 80 | 86 | y | y | nil |
| 14 | 256244 | 55 | f | 162 | 53 | DCP  Plating of rt tibia | 76 | 78 | 77 | 74 | 73 | 76 | y | y | nil |
| 15 | 258350 | 30 | m | 155 | 45 | debridement of traumatic wound | 82 | 80 | 78 | 80 | 77 | 78 | y | y | nil |
| 16 | 253016 | 25 | f | 152 | 42 | Appendicectomy | 96 | 96 | 97 | 98 | 95 | 94 | y | y | nil |
| 17 | 258418 | 22 | f | 160 | 38 | Appendicectomy | 90 | 94 | 96 | 92 | 94 | 90 | y | y | nil |
| 18 | 258410 | 28 | f | 150 | 38 | BAT | 66 | 66 | 68 | 69 | 68 | 67 | y | y | nil |
| 19 | 256704 | 30 | f | 156 | 44 | BAT | 68 | 70 | 68 | 69 | 67 | 68 | y | No,Pachy block | nil |
| 20 | 257726 | 36 | f | 159 | 43 | BAT | 77 | 76 | 78 | 79 | 75 | 76 | y | y | nil |
| 21 | 257624 | 38 | f | 155 | 38 | BAT | 78 | 76 | 73 | 75 | 77 | 78 | y | y | nil |
| 22 | 257511 | 40 | f | 160 | 60 | vaginal hysterectomy | 80 | 78 | 79 | 78 | 78 | 79 | y | y | nil |
| 23 | 259972 | 35 | m | 162 | 60 | debridement of wound lt foot | 76 | 74 | 77 | 72 | 75 | 78 | y | y | nil |
| 24 | 260448 | 30 | f | 168 | 56 | vaginal hysterectomy | 70 | 72 | 73 | 73 | 75 | 74 | y | y | nil |
| 25 | 260348 | 28 | f | 158 | 45 | Debridement rt leg | 68 | 70 | 70 | 71 | 72 | 68 | y | y | nil |
| 26 | 260268 | 25 | m | 162 | 46 | Debridement | 76 | 74 | 75 | 76 | 76 | 76 | y | y | nil |
| 27 | 262448 | 45 | m | 166 | 52 | Debridement lt leg wound | 70 | 68 | 70 | 67 | 69 | 73 | y | y | nil |
| 28 | 262414 | 30 | m | 162 | 50 | Lt tibia interlocking | 76 | 76 | 78 | 76 | 77 | 78 | no | no | converted to GA |
| 29 | 262468 | 58 | m | 174 | 64 | debridement of wound rt leg | 80 | 82 | 80 | 78 | 80 | 82 | y | y | nil |
| 30 | 265072 | 55 | m | 170 | 60 | lt inguinal hernia repair | 80 | 82 | 83 | 80 | 82 | 83 | y | y | nil |
| 31 | 264450 | 35 | m | 160 | 50 | lt inguinal hernia repair | 78 | 79 | 80 | 77 | 78 | 77 | y | y | nil |
| 32 | 264328 | 58 | m | 158 | 50 | rt inguinal hernia repair | 80 | 78 | 79 | 80 | 81 | 78 | y | y | nil |
| 33 | 261192 | 60 | m | 165 | 48 | lipoma excision | 90 | 89 | 90 | 88 | 89 | 80 | y | y | nil |
| 34 | 267491 | 25 | m | 156 | 42 | Abdominal wall abscess dranage | 80 | 79 | 80 | 80 | 79 | 80 | y | y | nil |
| 35 | 267288 | 32 | m | 178 | 80 | rt inguinal hernia repair | 70 | 72 | 72 | 70 | 72 | 70 | y | y | nil |
| 36 | 262674 | 65 | m | 160 | 76 | stsg of rt leg wound | 72 | 76 | 70 | 70 | 72 | 74 | y | y | nil |
| 37 | 267721 | 65 | f | 158 | 62 | Lt femur IT # dcp platting | 86 | 87 | 88 | 86 | 87 | 88 | y | y | nil |
| 38 | 268609 | 51 | m | 158 | 46 | ca penis for biopsy | 90 | 92 | 93 | 89 | 88 | 90 | y | y | nil |
| 39 | 267977 | 45 | f | 156 | 45 | Abdominal hysterectomy | 80 | 78 | 80 | 82 | 80 | 82 | y | y | nil |

| S/N | I.P. No | Age (Yrs) | Sex | Height (cm) | Weight (kg) | Operation | H.R (min)B | H.R (min) | H.R (min) | H.R (min) | H.R (min) | H.R (min) | Successful | Quality of Analgesia | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 40 | 271834 | 28 | f | 168 | 50 | TAH with BSO | 76 | 77 | 78 | 76 | 75 | 76 | y | y | nil |
| 41 | 270038 | 35 | f | 160 | 52 | Abdominal hysterectomy | 86 | 88 | 85 | 84 | 87 | 86 | y | y | nil |
| 42 | 273549 | 34 | f | 162 | 45 | abdominal hernia repair | 99 | 100 | 98 | 98 | 97 | 99 | Y | Y | nil |
| 43 | 253642 | 20 | m | 154 | 40 | appendicectomy | 94 | 96 | 92 | 90 | 93 | 90 | y | y | nil |
| 44 | 277426 | 28 | m | 154 | 46 | DCP Plating of rt femur | 70 | 68 | 721 | 71 | 70 | 71 | y | y | nil |
| 45 | 274808 | 35 | f | 160 | 55 | BAT | 64 | 65 | 66 | 64 | 63 | 64 | y | y | nil |
| 46 | 274805 | 28 | f | 148 | 45 | secondary suturing | 72 | 74 | 76 | 72 | 76 | 72 | y | y | nil |
| 47 | 274805 | 30 | f | 156 | 46 | TAH with BSO | 66 | 68 | 67 | 70 | 65 | 67 | y | y | nil |
| 48 | 274404 | 47 | f | 169 | 55 | TAH with BSO | 78 | 78 | 76 | 77 | 77 | 78 | y | y | nil |
| 49 | 275443 | 30 | f | 156 | 48 | BAT | 68 | 68 | 69 | 70 | 68 | 66 | y | y | nil |
| 50 | 275717 | 35 | f | 160 | 56 | vaginal hysterectomy | 66 | 65 | 68 | 69 | 65 | 67 | y | y | nil |
| 51 | 275332 | 62 | f | 160 | 62 | vaginal hysterectomy | 68 | 69 | 70 | 69 | 70 | 69 | y | y | nil |
| 52 | 275623 | 26 | m | 156 | 55 | debridement | 76 | 76 | 78 | 75 | 77 | 79 | y | y | nil |
| 53 | 276473 | 38 | m | 146 | 51 | implant removal | 76 | 75 | 77 | 78 | 76 | 78 | y | y | nil |
| 54 | 275622 | 35 | f | 158 | 48 | vaginal hysterectomy | 70 | 68 | 70 | 71 | 72 | 73 | y | y | nil |
| 55 | 275534 | 38 | f | 168 | 47 | vaginal hysterectomy | 72 | 68 | 70 | 70 | 72 | 71 | y | y | nil |
| 56 | 276445 | 26 | m | 170 | 48 | hydrocelectomy | 80 | 82 | 82 | 83 | 81 | 84 | y | y | nil |
| 57 | 276933 | 45 | m | 160 | 56 | ovarian cystectomy | 70 | 68 | 69 | 70 | 70 | 69 | y | y | nil |
| 58 | 276122 | 25 | f | 150 | 48 | Appendicectomy | 88 | 87 | 90 | 84 | 85 | 86 | y | y | nil |
| 59 | 276077 | 48 | f | 160 | 52 | vaginal hysterectomy | 76 | 76 | 78 | 80 | 76 | 78 | y | y | nil |
| 60 | 276705 | 62 | f | 156 | 60 | meshplasty | 78 | 76 | 80 | 84 | 76 | 78 | y | y | nil |
| 61 | 270778 | 56 | m | 160 | 60 | debridement | 64 | 68 | 70 | 69 | 70 | 68 | y | y | nil |
| 62 | 276244 | 28 | m | 158 | 45 | skin grafting rt leg | 68 | 68 | 70 | 72 | 68 | 70 | y | y | nil |
| 63 | 276122 | 58 | m | 158 | 50 | meshplasty | 80 | 78 | 80 | 82 | 82 | 80 | y | y | nil |
| 64 | 276320 | 46 | m | 162 | 56 | varicose vein stripping | 80 | 76 | 82 | 78 | 76 | 74 | y | y | nil |
| 65 | 277307 | 33 | f | 165 | 52 | BAT | 82 | 84 | 86 | 85 | 83 | 84 | y | y | nil |
| 66 | 275483 | 34 | m | 144 | 48 | varicose vein stripping | 80 | 78 | 82 | 84 | 80 | 79 | y | y | nil |
| 67 | 277599 | 45 | m | 165 | 70 | secondary suturing | 68 | 64 | 67 | 68 | 69 | 66 | y | y | nil |
| 68 | 277565 | 32 | m | 156 | 56 | BAT | 67 | 68 | 64 | 67 | 68 | 70 | y | y | nil |
| 69 | 277521 | 36 | m | 168 | 70 | haemorrhoidectomy | 86 | 88 | 86 | 88 | 90 | 86 | y | y | nil |
| 70 | 277579 | 45 | f | 156 | 48 | vaginal hysterectomy | 76 | 77 | 78 | 76 | 75 | 78 | y | y | nil |
| 71 | 275323 | 24 | m | 154 | 54 | debridement of wound rt leg | 74 | 75 | 74 | 76 | 75 | 77 | y | y | nil |
| 72 | 277982 | 36 | f | 156 | 56 | meshplasty | 68 | 67 | 68 | 70 | 67 | 69 | no | no | Converted to GA |
| 73 | 277828 | 40 | m | 157 | 45 | meshplasty | 76 | 78 | 77 | 76 | 75 | 78 | y | y | nil |
| 74 | 277863 | 46 | f | 160 | 56 | appendicectomy | 80 | 84 | 85 | 86 | 87 | 85 | y | y | nil |
| 75 | 277882 | 47 | m | 158 | 60 | meshplasty | 80 | 78 | 76 | 79 | 80 | 79 | y | y | nil |
| 76 | 277955 | 32 | f | 162 | 48 | appendicectomy | 67 | 68 | 70 | 68 | 69 | 70 | y | y | nil |
| 77 | 277689 | 55 | f | 158 | 60 | varicose vein stripping | 68 | 70 | 68 | 69 | 72 | 68 | y | y | nil |
| 78 | 280180 | 23 | m | 146 | 36 | Hydrocelectomy | 86 | 86 | 84 | 86 | 86 | 87 | y | y | nil |
| 79 | 280392 | 42 | m | 148 | 50 | appendicectomy | 90 | 89 | 88 | 91 | 92 | 89 | y | y | nil |

| S/N | I.P. No | Age (Yrs) | Sex | Height (cm) | Weight (kg) | Operation | H.R (min)B | H.R (min) | H.R (min) | H.R (min) | H.R (min) | H.R (min) | Successful | Quality of Analgesia | Complications |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 80 | 280773 | 60 | m | 153 | 58 | lt inguinal hernia repair | 88 | 86 | 84 | 88 | 86 | 88 | y | y | nil |
| 81 | 282078 | 28 | f | 148 | 30 | BAT | 80 | 78 | 80 | 77 | 79 | 80 | y | y | nil |
| 82 | 280635 | 54 | m | 149 | 48 | lt inguinal hernia repair | 84 | 86 | 84 | 82 | 86 | 88 | y | y | nil |
| 83 | 281920 | 50 | m | 156 | 60 | haemorrhoidectomy | 78 | 78 | 76 | 78 | 80 | 76 | y | y | nil |
| 84 | 282891 | 35 | m | 148 | 56 | paraumblical hernia repair | 88 | 92 | 90 | 96 | 94 | 92 | y | y | nil |
| 85 | 282120 | 24 | f | 160 | 40 | appendicectomy | 70 | 71 | 72 | 73 | 74 | 75 | y | y | nil |
| 86 | 284264 | 45 | f | 162 | 62 | B/L varicose vein stripping | 78 | 78 | 76 | 74 | 78 | 78 | y | y | nil |
| 87 | 284895 | 62 | m | 162 | 60 | excision of epididamal cyst | 70 | 70 | 68 | 64 | 66 | 70 | y | y | nil |
| 88 | 288278 | 30 | f | 152 | 50 | appendicectomy | 70 | 74 | 70 | 76 | 70 | 74 | y | y | nil |
| 89 | 287256 | 56 | m | 158 | 60 | B/L varicose vein stripping | 68 | 68 | 70 | 72 | 68 | 68 | y | y | nil |
| 90 | 287457 | 30 | f | 160 | 52 | B/L varicose vein stripping | 66 | 70 | 68 | 69 | 70 | 71 | y | y | nil |
| 91 | 287564 | 60 | m | 148 | 58 | Debridement | 86 | 84 | 83 | 82 | 86 | 86 | y | y | nil |
| 92 | 290057 | 45 | m | 156 | 55 | appendicectomy | 80 | 78 | 76 | 78 | 79 | 80 | y | y | nil |
| 93 | 279203 | 25 | m | 145 | 48 | varicocelectomy | 78 | 80 | 78 | 77 | 79 | 78 | y | y | nil |
| 94 | 277570 | 35 | f | 146 | 42 | DHS Plating | 80 | 84 | 82 | 83 | 84 | 80 | y | y | nil |
| 95 | 282245 | 35 | f | 146 | 37 | appendicectomy | 84 | 83 | 85 | 84 | 83 | 85 | y | y | nil |
| 96 | 282200 | 46 | m | 148 | 48 | appendicectomy | 80 | 84 | 82 | 86 | 85 | 84 | y | y | nil |
| 97 | 278329 | 52 | m | 146 | 52 | penile growth for biopsy | 80 | 81 | 82 | 82 | 81 | 86 | y | y | nil |
| 98 | 279941 | 30 | m | 150 | 60 | appendicectomy | 82 | 80 | 82 | 86 | 82 | 78 | y | y | nil |
| 99 | 280140 | 32 | f | 158 | 50 | appendicectomy | 78 | 76 | 78 | 76 | 77 | 76 | y | y | nil |
| 100 | 280748 | 38 | f | 146 | 50 | meshplasty | 68 | 70 | 71 | 72 | 70 | 70 | y | y | nil |
| 101 | 281440 | 28 | m | 156 | 42 | STSG | 78 | 78 | 76 | 75 | 78 | 78 | y | y | nil |
| 102 | 281288 | 30 | m | 168 | 52 | appendicectomy | 68 | 70 | 68 | 69 | 70 | 69 | y | y | nil |
| 103 | 282824 | 30 | m | 158 | 40 | Appendicectomy | 80 | 78 | 79 | 80 | 80 | 79 | y | y | nil |
| 104 | 283014 | 45 | f | 152 | 50 | vaginal hysterectomy | 70 | 69 | 70 | 70 | 68 | 71 | y | y | nil |
| 105 | 283326 | 38 | m | 159 | 50 | lt inguinal hernia repair | 70 | 70 | 69 | 68 | 70 | 68 | y | y | nil |
| 106 | 281426 | 52 | m | 156 | 68 | debridement | 70 | 70 | 69 | 70 | 72 | 74 | y | y | nil |
| 107 | 282743 | 32 | m | 157 | 48 | appendicectomy | 80 | 80 | 78 | 77 | 79 | 80 | y | y | nil |
| 108 | 282840 | 28 | m | 160 | 48 | corn excision rt foot | 76 | 76 | 74 | 76 | 77 | 78 | y | y | nil |
| 109 | 298668 | 28 | m | 160 | 50 | appendicectomy | 70 | 68 | 70 | 68 | 70 | 70 | y | y | nil |
| 110 | 288798 | 30 | f | 159 | 65 | uv prolapse | 69 | 68 | 72 | 71 | 72 | 70 | y | y | nil |