"COMBINED SPINAL EPIDURAL FOR LABOUR ANALGESIA COMPARISON OF TWO DIFFERENT DOSES OF INTRATHECAL BUPIVACAINE 1.25mg AND FENTANYL 25µg WITH BUPIVACAINE 2.5mg AND FENTANYL 25µg "

BY

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M.B.B.S.,

Dissertation submitted to the

Rajiv Gandhi University of Health Sciences, Karnataka, Bangalore.

In Partial fulfillment

Of the requirement for the degree of

DOCTOR OF MEDICINE

IN

ANAESTHESIOLOGY

Under the guidance of

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ABSTRACT

BACKGROUND AND OBJECTIVE: The responsibility of the anaesthetist in obstetrics is arguably greater than in any other field of anaesthesia. This study compares two different low doses of intrathecal Bupivacaine 1.25 mg and 2.5 mg along with 25 µg Fentanyl as the spinal component of combined spinal epidural analgesia in the early part of labor , followed by epidural top up .

METHODOLOGY: Approval was obtained from the institutional review board and written informed consent was obtained from 60 healthy term primigravida or second gravid parturients, with cephalic singleton pregnancy between 36-42 weeks, ASA grade I/II patients. The study was conducted using low dose intrathecal Bupivacaine 1.25mg and Fentanyl 25 µg (group I) with Bupivacaine 2.5mg and Fentanyl 25 µg (group II) as the spinal component of combined spinal epidural analgesia in the early part of labor . We compared the two with respect to their onset, duration of sensory and motor block, quality of analgesia during early part of labor and the side effects of the drugs.

RESULTS: The onset of analgesia was equally rapid with both groups within 5 min, lower incidence of motor block with Group I compared to Group II. Duration of analgesia was longer in Group II, associated with higher dermatome levels of sensory block with longer time for regression of the block. However many required subsequent use of their epidural catheter to continue analgesia.

CONCLUSION: We found that bupivacaine 1.25 mg was as effective as bupivacaine 2.5 mg when added to fentanyl 25 µg for combined spinal epidural

analgesia in early part of labour, with less motor and sensory block and hypotension. Onset of analgesia was rapid and achieved within 5-10 min.

Key words: combined spinal epidural anesthesia, labor analgesia, spinal, epidural, fentanyl, bupivacaine, visual analogue scale.

CONTENTS

LIST OF ABBREVIATIONS USED

μg	-	Microgram
ASA	-	American Society of Anaesthesiologists (classification)
BP	-	Blood Pressure
BT	-	Bleeding Time
cm	-	centimeter
CSE	-	Combined Spinal Epidural
CSEA	-	Combined Spinal Epidural Analgesia
CSF	-	Cerebro Spinal Fluid
CT	-	Clotting Time
ECG	-	Electro Cardio Gram
gm	-	gram
HS	-	Highly Significant
IV	-	Intravenous
Kg	-	Kilogram
L	-	Liters
LSCS	-	Lower Segment Cesarean Section
Min	-	Minutes
ml	-	milliliter
mm of Hg	-	millimeter of mercury
NS	-	Not Significant
PDPH	-	Post Dural Puncture Headache
pKa	-	Dissociation constant
S	-	Significant
S.D.	-	Standard Deviation
T	-	Thoracic segment
TENS	-	Transcutaneous Electrical Nerve Stimulation
VAS	-	Visual Analogue Scale

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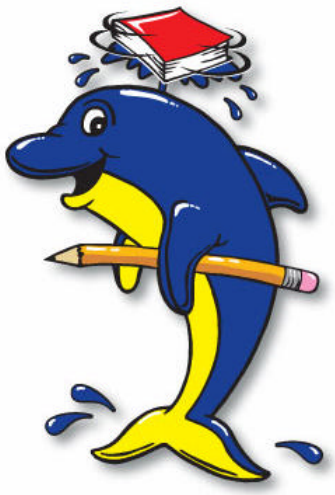


Introduction

INTRODUCTION

"The delivery of the infant into the arms of a conscious and pain free mother is one of the most exciting and rewarding moments in medicine".

- Moir DD

Labor is defined as a series of events that take place in the genital organs in an effort to expel the viable products of conception out of the womb through the vagina into the outer world[1]

Pain is a noxious and unpleasant stimulus which produces fear and anxiety. Pathophysiological responses occur in the body during pain. In the respiratory system there is hyperventilation during contraction, increasing the work of breathing and oxygen consumption.[2] Unrelieved stress in labour produces increased plasma cortisol and catecholamines concentrations which reduces utero-placental blood flow[3] by 35-70% compounding the effects of hyperventilation on the oxygen supply to the foetus.

Metabolic acidosis as a result of increased metabolic rate especially in the second stage of labour is transferred to the foetus. There is delayed gastric emptying and urinary emptying.[2]

Effective pain relief reduces plasma noradrenaline[4], prevents the rise during first and second stage of labour of 11-hydroxy corticosteroid[6], prevents metabolic acidosis by reducing the rate of rise of lactate and pyruvate.[5] It decreases maternal oxygen consumption by upto 14%.[6]

The pain-induced hyperventilation and hypocapnia[7] , reduces utero-placental blood flow by upto 25%. The respiratory alkalosis further impairs foeto-maternal gas exchange by shifting the oxyhaemoglobin dissociation curve to the left and fetal PaO_2 may fall upto 23%.[7]

Various techniques available for pain relief during labour include.[8]

1. Non-pharmacological methods:

1. Prepared child birth
2. Hypnosis
3. Acupuncture
4. Transcutaneous electrical nerve stimulation (TENS)

2. Pharmacological methods:

A. Inhalational analgesics

- Gaseous – nitrous oxide, oxygen (entonox)

B. Systemic analgesics

- Opioid analgesics
- Opioid Agonist – antagonist analgesics.
- Non-opioid analgesics

  Tranquilizers

  Sedatives

  Ketamine

C. Regional techniques

- Epidural analgesia

- Spinal analgesia
- Combined spinal epidural
- Alternative regional techniques
    - Lumbar sympathetic block
    - Paracervical block
    - Pudendal nerve block

Advantages of regional techniques are that there is no risk of gastric aspiration, avoids the use of depressant general anaesthetic drugs and allows the mother to remain awake and participate during delivery.

It has been suggested that confining women to bed during labour may cause the labour to be longer and more pain feel with an increase in abnormal presentation, instrumental deliveries and fetal distress.[10]

Epidural analgesia has been used extensively using mixture of low dose local anaesthetics and opioids to provide pain relief in labour, but has the drawbacks of delayed onset and motor blockade. Low dose combined spinal epidural (CSE) analgesia has gained wide spread acceptance as an approach to labour analgesia. The rapid onset of analgesia is one of the major advantages of combined spinal epidural analgesia and with its increased association with maternal satisfaction.[9]

Combined spinal epidural analgesia consists of identification of epidural space and insertion of an epidural catheter plus the initial intentional placements of an intrathecal dose of opioids, local anaesthetic or both all as a single procedure.[11]

Combined spinal epidural analgesia is an effective method of analgesia in labour. Intrathecal administration of combination of local anaesthetic and lipophilic opioid provides rapid analgesia. Principle drug providing the intrathecal component of analgesia is the lipid soluble opioid, synergism has been demonstrated when a local anaesthetic is administered together with an opioid allowing enhanced pain relief with fewer adverse effects.[9]

The present study compares the efficacy of low dose of bupivacaine 1.25mg and 2.5 mg with Fentanyl 25 µg intrathecally (single dose) in terms of onset, duration of block and quality of analgesia during labour, followed by epidural analgesia.

Objectives

OBJECTIVES

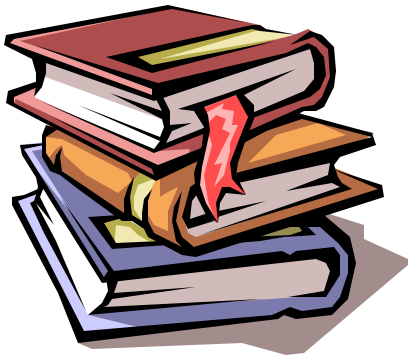
Objectives of the study :

1. To study the onset and duration of sensory and motor block in early part of labour with two different low doses of intrathecal Bupivacaine (1.25 mg and 2.5 mg) along with 25 µg Fentanyl.

2. To study quality of analgesia during early part of labour.

3. To study the side effects of the drugs

    - Pruritis
    - Sedation
    - Nausea vomiting
    - Hypotension
    - Fetal bradycardia.



Review of Literature

REVIEW OF LITERATURE

ANATOMY OF VERTEBRAL COLUMN AND EPIDURAL SPACE:

Vertebral column :

The vertebral canal extends from the foramen magnum to the sacral hiatus. It is composed of 33 vertebrae. (7 cervical, 12 thoracic, 5 lumbar, 5 fused sacral and 4 coccygeal) It has four curves, the cervical and lumbar curves are convex anteriorly while the thorax and sacral curves are convex posteriorly. These curves have a significant influence on the spread of local anaesthetic in the subarachnoid and epidural space.

The canal is bounded in front by bodies of the vertebrae and intervertebral disc with the posterior longitudinal ligament, posteriorly by the laminae and ligamentum flava; laterally by the pedicle and laminae. The vertebral canal is narrow at the thoracic level and considerably wider at the cervical and lumbar levels.[12,13]

The vertebral column is bounded together by several ligaments, which give its stability and elasticity.

a) Supraspinous ligament
b) Interspinous ligament
c) Ligamentum flavum
d) Longitudinal ligaments

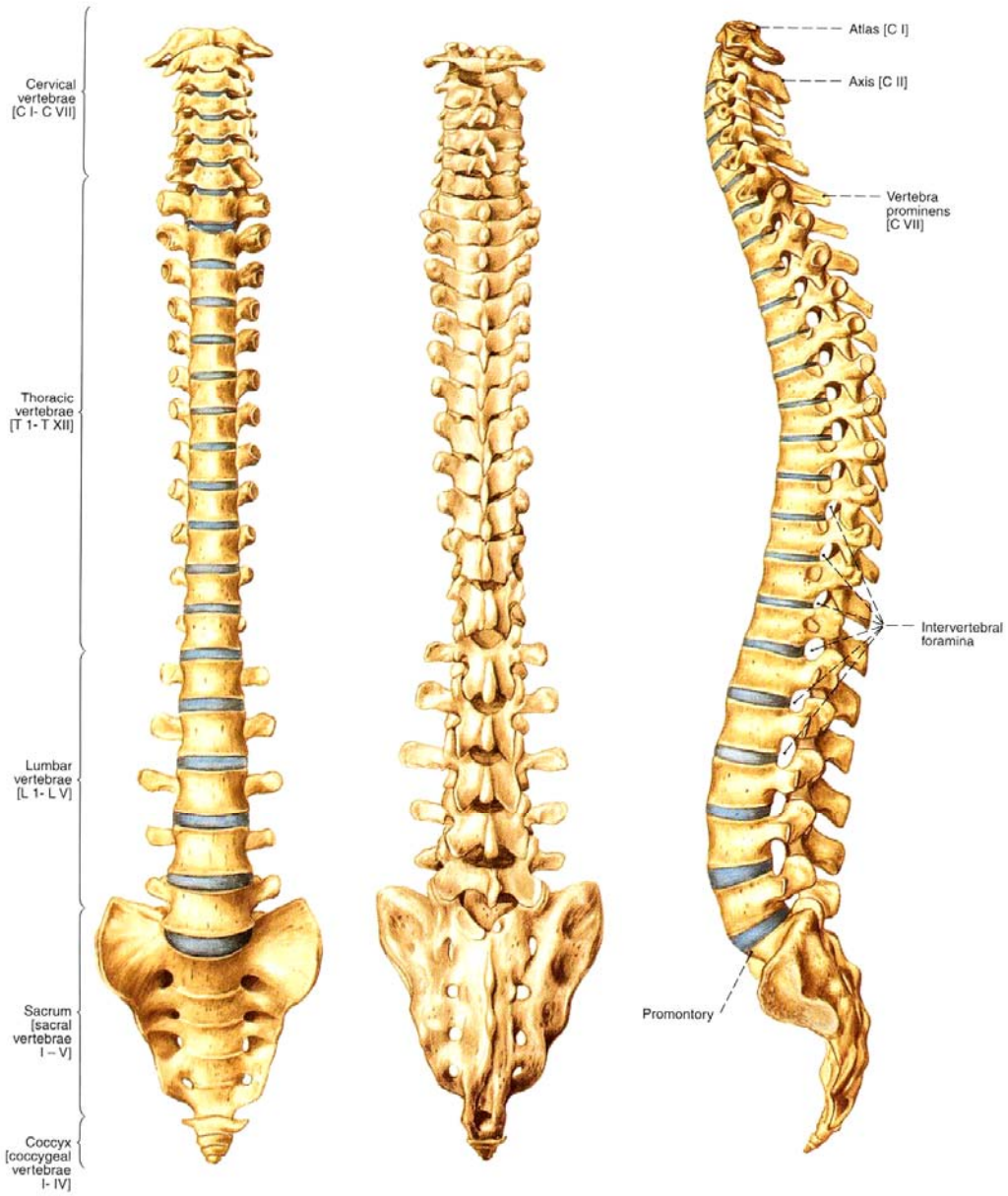


Fig. 1 : Vertebral column

a) **Supraspinous ligament:** It is a strong fibrous ligament that connects the apices of the spinous processes from the sacrum to C7, where it is continued upwards to the external occipital protrubcrancc as the ligamenlum nuchae. It is thickest and broadest in the lumbar region and varies with patient age and sex.

b) **Intcrspinous ligament:** It is a thin membranous ligament that connects the spinous processes together, uniting the lower border of one with the upper border of its caudal neighbour. Therefore the ligament is rectangular in shape. It blends anteriorly with the ligamentum flavum and posteriorly with supraspinous ligament.

c) **Ligamentum flavum :** This ligament is composed of yellow elastic fibers and connects adjacent laminae that run from the caudal edge of the vertebrae above to the cephalad edge of the lamina below, laterally this ligament begins at the roots of the articular processes and extends posteriorly and medially to the point where the laminae join to form the spinous process. Here the two compartments of the ligaments are united, thus covering the interlaminar space.

They cover the capsules of the articular facets, the lower part of the upper laminae and the inter laminar spaces. The ligamentum flava constitute slightly more than half of the posterior wall of the vertebral canal. They arc thickest and strongest in the lumbar region where powerful stresses and strain have to be countered. Site thickness of ligamentum flava :

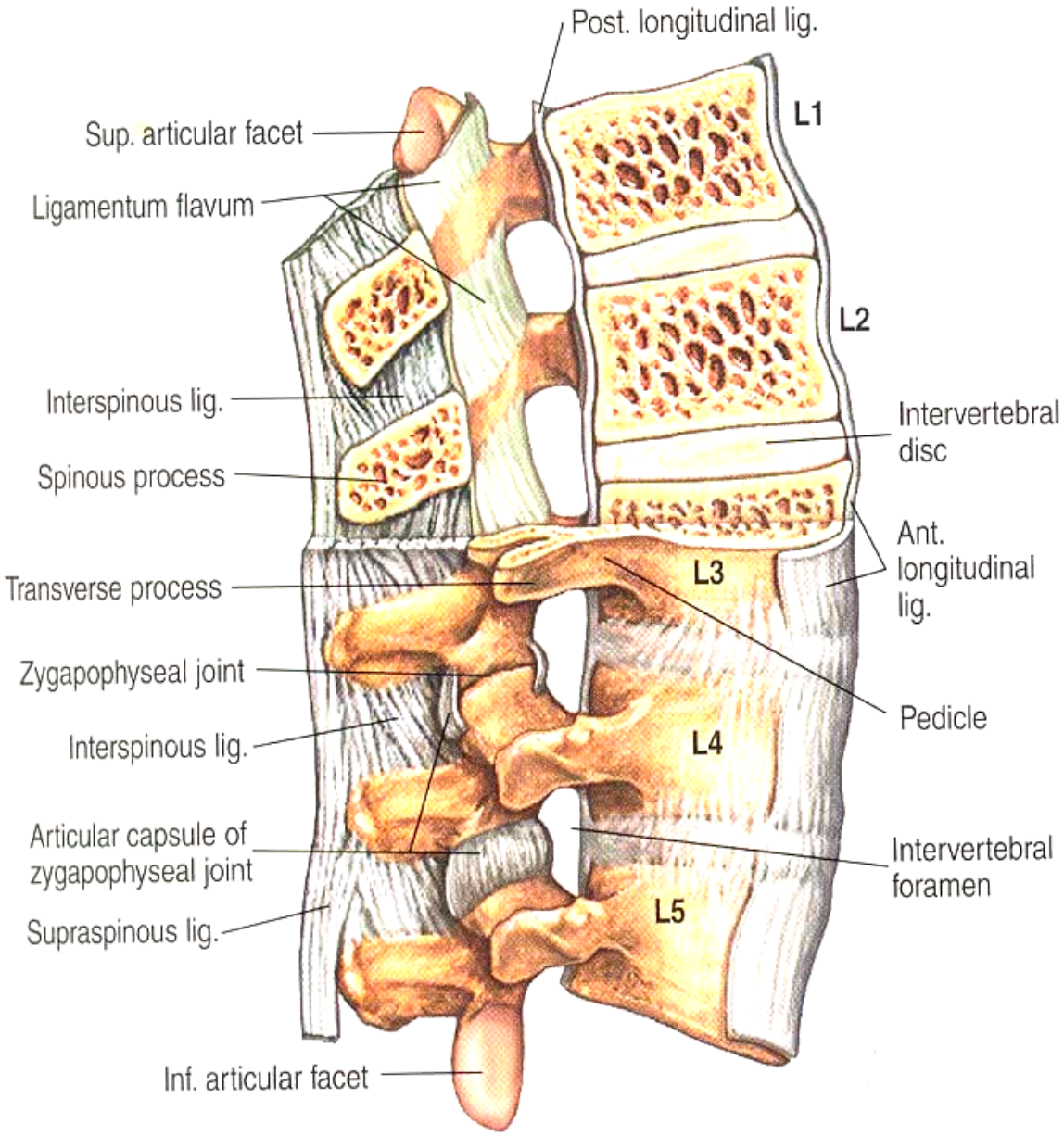


Fig.2 : Lateral view of lumbar vertebral ligaments

1. Cervical 1.5 to 3mm
2. Thoracic 3 to 5mm
3. Lumbar 5 to 6 mm
4. Sacral 2 to 6mm

d) **Longitudinal ligaments:** The anterior and posterior longitudinal ligaments bind the vertebral bodies together.

Posterior Longitudinal ligament:

Posterior longitudinal ligament: Lies with in the canal on posterior surfaces of bodies of vertebra from which it is separated by basivertebral veins. This ligament is thinnest in cervical and lumbar region.

Anterior longitudinal ligament: It is more of anatomical interest than anaesthetic interest. It runs along the front of vertebral bodies.

The cervical, thoracic and lumbar vertebrae have certain differentiating features. Cervical vertebrae differ from the thoracic and lumbar vertebrae, the former have foramina in their transverse process. The thoracic vertebrae differs from the lumbar and cervical vertebrae, the former have articular facets for ribs on their bodies. The spine of the thoracic vertebrae slopes steeply downwards, while those of lumbar vertebra are horizontal.

LUMBAR VERTEBRA

A typical lumbar vertebra is made up of following pans :

1. The body
2. Vertebral arch
3. Transverse and spinous processes
4. Superior and inferior articular processes

BODY:

It is kidney shaped. They are weight bearing. The flat articular surfaces are covered with hyaline cartilage, which is firmly united to the fibrocartilagenous intervertebral discs (annulus fibrosus and nucleus pulposus). The anterior and posterior longitudinal ligaments reinforce the union between the bodies. The broad anterior longitudinal ligament is firmly attached to the intervertebral discs and loosely attached to bodies. The posterior longitudinal ligament is narrower and is similarly attached. It sends a few irregular slender fibers to join the anterior surface of the spinal duramater.

Vertebral arch :

Composed of pedicles and laminae which surround and protect the spinal cord and its coverings.

Each half of the vertebral arch is divided into two parts by the root of the transverse process. Anteriorly, the arch is formed by the powerful rounded pedicle, whose function is to transmit stress. Posteriorly it is completed by the lamina, which is flat and is mainly protective in function. From the vertebral

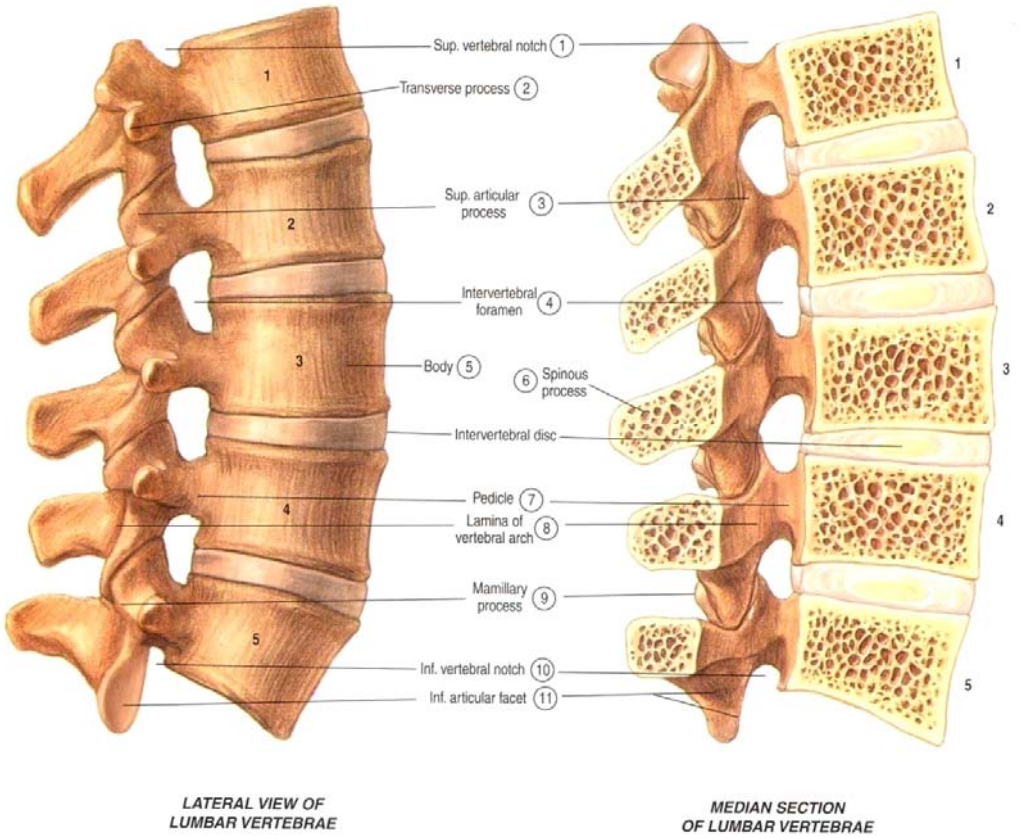


Fig. 3 : Lateral view of lumbar vertebral column

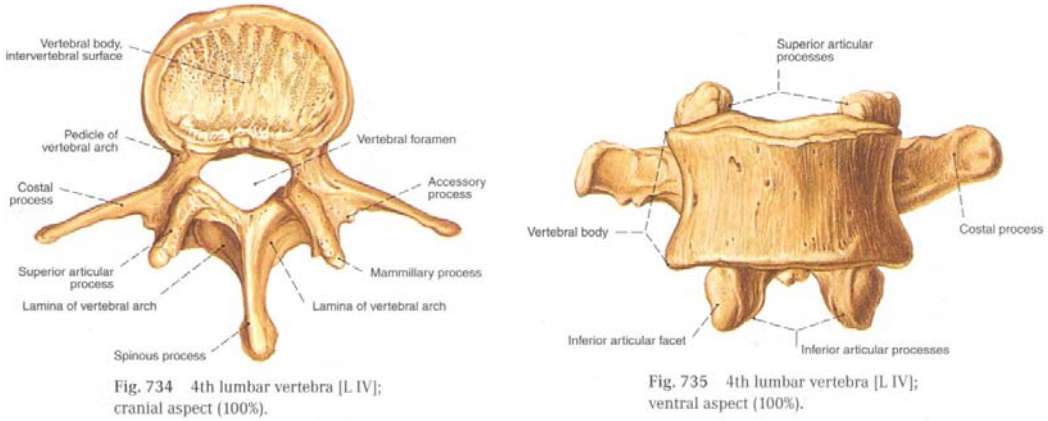


Fig. 4: Lumbar vertebra

arches four articular processes project, of which two are directed upwards and two downwards to articulate with similar processes of the adjacent vertebrae.

The superior articular processes spring from the junctions of pedicles and laminae. They project upwards behind the pedicles and come to lie just above the level of transverse processes and the articular facets on their posterior surface facing backwards and medially. The inferior articular processes extend downwards from the hiferolateral aspects of the laminae. They lie well below the level of the transverse processes and the articular facets on their anterior surface are placed laterally and forwards. So that they articulate with the facets on the superior articular processes of the vertebra below.

The pedicles arise from the upper part of the posterolateral surface of the body. So that there are two notches formed between the body and the pedicle viz, superior and inferior of which inferior is much deeper. So when two adjacent vertebrae articulate they enclose an intervertebral foramen on either side through which the mixed spinal nerve of that particular segment issues. The boundaries of the intervertebral foramen are bounded superiorly and inferiorly by the pedicles of adjoining vertebrae, posteriorly the capsules surrounding the articular processes of adjoint vertebrae and anteriorly by the intervertebral disc and the lower part of the body above it.

The posterior surface of the body and the vertebral arch together form the boundaries of the vertebral foramen. These foramina collectively form the vertebral canal, which contain the spinal cord and its covering membranes. The anterior boundary of the vertebral canal presents as a continuous solid surface

formed by the posterior aspects of the bodies and intervertebral discs, covered by the posterior longitudinal ligament. The lateral and posterior wall formed by the vertebral arches is however incomplete in the articulated skeleton they form.

a) **Laterally:** intervertebral foramina, through which the segmental spinal nerve passes.

b) **Posteriorly:** The interlaminar foramina, through which approach to the subarachnoid and epidural space is possible. In the lumbar regions the inter laminar foramina is small and triangular when the spine is extended. The base of the triangle is formed by the upper border of the lamina of the vertebra below and the sides by the medial aspects of the inferior articular processes of the vertebra above. When the spine is flexed the inferior articular processes slide upwards, and the intcrlaminar foramina are enlarged and become diamond shaped. The medial borders of the superior articular processes of the vertebra below form the lower lateral boundaries of the aperture.

Lumbar intervertebral joints :

These joints are fibiocarlilagenous between the bodies and ligaments, between the arches.

Intervertebral discs :

This forms at least $l/4^{th}$ of the total length of the vertebral column. In the cervical and lumbar regions they are somewhat wedge shaped and thus contribute to the characteristic curves of the column. They are thickest in the

lumbar region. Each disc consists of a fibrous outer cover, the annuius fibrosus that is attached to the hyaline cartilage covering the articular surfaces of the vertebral bodies it connects.

The annuius encloses a core of gelatinous material, the nucleus pulposus that accommodates itself to the change in the shape of its coverings during the movement between the vertebrae. Intervertebral discs thus acts as shock absorber and give tiexibility to the vertebral column. As a result of weakness or strain the annuius may rupture usually posteriorly where it is thinnest. The nucleus pulposus may then herniate through the deficiency, causing signs and symptoms depending on the nerve roots involved. This condition is described as "prolapsed disc" and may occur as a complication of lumbar puncture when the technique is faulty or when the spine exerts exaggerated flexion.

Intervertebral foramina:

The lateral aspects of the vertebral column presents a series of intervertebral foramina through which pass the spinal nerves and accompanying vessels. The aerolar tissue around these foramina is soft and loose in the young individual and the anaesthetic solution injected into the epidural space easily passes through these foramina. An epidural catheter may also passes through one of the foramina, in elderly patient the dense, firm areolar tissue seals this foramina. For this reason lesser amount of local anaesthetic solution is required to produce an epidural block in the elderly as compared to young individuals.

Spinal meninges:

Spinal cord is protected by both the bony vertebral column and three connective tissue covering the meninges.

Dura mater :

The outer most membrane is a tough fibro elastic tube whose fibers run longitudinally. It can be described in two parts, the cranial and the spinal. Superiorly, it is firmly attached to the circumference of the foramina magnum of the occipital bone. Here it becomes continuous with the inner meningeal layer of the cranial duramater. The outer endosteal layer of the cranial duramater becomes continuous with the periosteal linings of the vertebral canal. Interiorly or caudally the dural sac ends at the lower border of S2 vertebra where it is pierced by the filum tcrminale.

The filum terminate is the terminal thread of the pia mater which extends from the tip of the spinal cord to blend with the periostium on the back of the coccyx and helps in anchoring the spinal cord. The spinal dura also provide a thin cover for the spinal nerve. Here it is thinner than that of the dural sac. The separate dural sleeves for the two roots fuse just beyond the spinal ganglion, when the roots unite to form the mixed spinal nerve. The dura here comes to an end and is replaced by the perineurium. Throughout its entire length the spinal dura is thicker in the posterior aspects than the anterior aspect and this is especially true in the mid line. For this reason a dural puncture, with a Tliohy's needle is rarely made.

Arachnoid mater:

It is the middle of the three coverings of the brain and spinal cord. It is a delicate non-vascular membrane closely attached to the dura, which ends at the lower border of S2- There is a capillary internal called subdural space between the dura and arachnoids space. It contains a minute quantity of serous fluid.

The arachnoid villi and granulations penetrate the dura and lie in contact with the epidural veins. These help in transferring the CSF from the subarachnoid space, back into blood stream.

Pia mater:

It is delicate and highly vascular membrane, closely investing the spinal cord and the brain. The space between the arachnoid and the pia is thus called the subarachnoid space. It contains CSF, spinal rootlet, and a large number of trabeculae running between these two membranes. Lateral projections of the pia and the denticulate ligaments are attached to the dura and aid in supporting the spinal cord.

Nerve supply of the meninges:

The posterior aspect of the spinal dura is not supplied by nerve fibers so that pain is not felt when the dura is pierced by a spinal needle.

The anterior aspect is supplied by twigs from the spinal nerves, each of which enters the intervertebral foramen, divides and passes upwards for one segment and downwards for two segments. (Edger and Hundy 1966).

Spinal nerves:

There are 31 pairs of symmetrically arranged spinal nerves, which arc attached to the spinal cord by two roots. Both the anterior and posterior roots arise from the cord as several filaments of rootlets. The lumbar and sacral roots are the longest and largest and extend over several vertebrae. This greater surface area together with the fact that, only a thin layer of pia covers the

rootlets and roots allows for a rapid onset of anaesthesia, when local anaesthetics are injected into the subarachnoid space.

Subarachnoid space:

The subarachnoid space is lined externally by the arachnoid, internally by the pia mater and innumerable cobweb-like trabeculae, between the two membranes. It is traversed by the cranial and spinal nerves. It houses the main blood vessels of the central nervous system and extends along the smaller arteries and capillaries into the nervous tissue of the brain and spinal cord. Here the cerebrospinal fluid takes the place of the tissue fluid (lymph) found in other regions of the body. In the cervical and thoracic regions the space is annular and the distance between the arachnoid and the pia covering the cord, even in an adult is only about 3mm. The cord commonly ends at the lower border of the first lumbar vertebra, so that below this level the subarachnoid space is no longer annular but is practically circular in cross section and has a diameter of about 15mm.

Blood supply of spinal cord:

The arterial supply is from the anterior and posterior spinal arteries.

The anterior spinal artery is a single vessel lying in the pia mater in front of the anterior median Fissure. It arises from the junction of two small arteries one given off from each vertebral artery at the level of foramen magnum. It divides along the whole length of the cord receiving small communications from the intercostals and lumbar arteries to provide the extra blood supply needed in the cervical, thoracic and lumbar enlargements. The

communicating branches at the level of T_1 and T_2 are larger than the others (the arteries of adamkiewicz).

The posterior spinal arteries are two or three in number on each side and originate from posterior inferior cerebellar arteries at the base of the brain. They supply the posterior columns of the cord. There are no anastamoses between the anterior and posterior spinal arteries.

The extradural veins receive blood from the cord and from the vertebral canal and its contents and they communicate above with intracranial sinuses and below with tributaries of the inferior vena cava. They also communicate with the azygous vein. They form a plexus, which is most dense in the anterolateral compartment of the extradural space. The venous plexus surrounds each nerve root and the main longitudinal channels are without valves.

Thus there is a continuous vascular connection between the pelvis and the cranium by passing the venacava.

Cerebro spinal fluid :

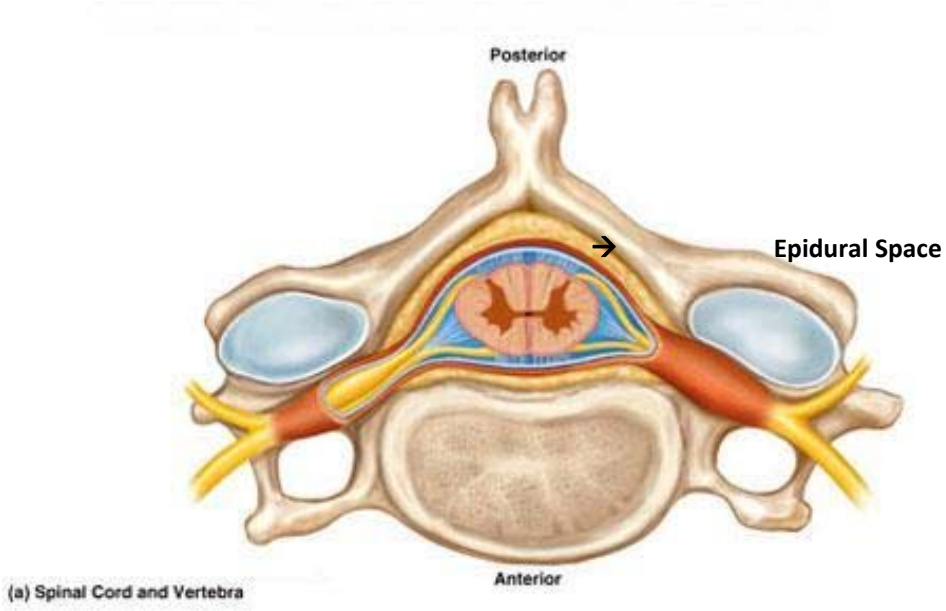
It is formed by the ultrafiltration of plasma by choroid plexus of the ventricles.

Characteristic features:

1. Colourless, odourless clear fluid does not clot
2. P.H. 7.4

   Specific gravity 1.004-1.007

   Protein 15 to 45 mg%



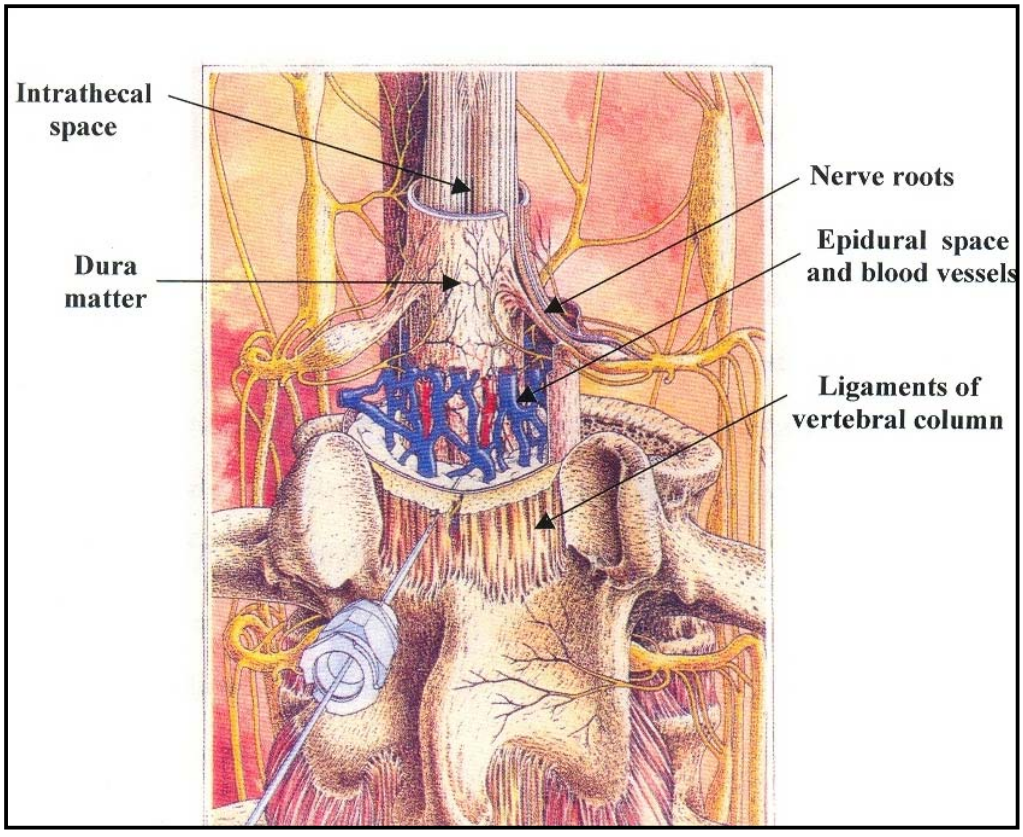


Fig. 5: Epidural space

Glucose	1.5 to 4 mmol/L
Chloride	120 to l30meq/L
Volume	135-150cc in total

Epidural space:

It is the potential space within the bony cavity of the spinal canal and outside the dural sac. The bodies of the vertebrae, the intervertebral discs and the posterior longitudinal ligament covering them bound it anteriorly. Posteriorly it is bounded by the anterior surface of the vertebral laminae and the ligarnentum flava. Superiorly it is closed by the fusion of the dura and periostium at the foramen magnum. Inferiorly fay the sacro coccygeal ligament at the sacral hiatus, laterally by the pedicles of the vertebrae and the intervertebral foramina.[14]

The shape of the epidural space in cross section is nearly circular in the cervical and thoracic regions, but becomes triangular in the lumbar regions. The depth of the epidural space is greatest in the midline in the lumbar region where it is said to be 5-6 mm in adult male. [Change 1963, Bromage 1978) for this reason midline approach is advocated for entering the lumbar epidural space.

Epidural space communicates via the intervertebral foramina, with the paravertebral spaces. The paravertebral spaces in the thoracic region lie between the heads of the ribs and are in direct contact with pleura. The negative intrathoracic pressure is thus conducted via the paravertebral spaces to the thoracic epidural space and to a diminishing extent to the cervical and lumbar regions.[14]

Contents of the epidural space:

Dural space: It extends from the foramen magnum to the lower border of second sacral vertebra. The dural tube tends to hug the anterior wall of the spinal canal, so that the epidural space is narrow anteriorly and wider posteriorly.

Spinal nerve roots: Along with their dural cuffs, they traverse the epidural space on their way to their respective intervertebral foramina. In the cervical region these travel almost horizontally but lower down they become more inclined owing to the discrepancy between the length of the spinal cord and the spinal canal, until the lower lumbar and sacral roots are almost vertical.

The roots vary greatly in size and thickness, the thoracic roots are thin white the cervical and lumbosacral roots subserving the limbs are thick. The great differences in size and neural population with in the roots are interrelated. The very large diameter and high neural populations of the dorsal and ventral roots of the first sacral segment are associated with great resistance to epidural blockade. Prolonged latency and poor analgesia of spinal segments are due to poor penetration of local anaesthetics to the deeply placed fibers at the core of the root.

The root sleeves deserve a special mention, as they have an important role in the mechanism of the action of epidural anaesthesia. In the region of the "dural cuff the arachnoid villi and granulations invaginate the epidural veins and drain the CSF from the subarachnoid space, into the blood stream. Those villi, which are not in contact with the vessels, drain the CSF into the epidural fat, from where it is drained by lymphatics.

Epidural vessels: The branches of the subclavian, aortic and iliac arteries cross the epidural space and enter the subarachnoid space in the region of the dural cuffs. These branches provide blood supply for the spinal roots. Apart from the cervical region, the entire blood supply to the spinal cord passes through the epidural space. The epidural veins are arranged in the mid line. They drain the spinal cord, vertebral canal and CSF from the subarachnoid space. They do not posses valves. These veins although divided into anatomical groups, ail are interconnected and form a series of horizontal segmental anastamosis. They connect with the intervertebrai foramina and communicate with the vertebral, ascending cervical, deep vertical, intercostal, iliolumbar and lateral sacral veins. As the epidural veins have no valves, they afford a connection between the pelvic veins below and the intercostal veins above.

The epidural veins become distended during coughing and straining and also when the inferior vena cava is obstructed by large abdominal tumors or in late pregnancy. This distension of epidural veins diminishes the effective volume of the epidural space. Under these circumstances, the requirement of the local anaesthetic is markedly decreased, as a small volume of drug tend to spread over a wide area of epidural space.

Fat: The content of the spinal canal lie cushioned in a packing of semifluid, lobulated fat. Solutions injected into the epidural space, track up and down between the fatty tissues. The epidural fat consists an important pharmacological space and depot for injected local anaesthetics and other drugs and it is one of the three compitators for its share of the drug. The other two compitators being nervous tissue of spinal roots and cord and blood vessels with in the spinal canal. Drug with high lipid solubility and Hpoprotein binding characteristics will tend to enter the fat phase and remain there for a period of time depending on their pharmacodynamics and on the briskness of the

local blood flow competing for uptake. The compliance of the epidural fat varies from person to person and with age, in children and young adults it offers very little resistance.

Lymphatics : Lymphatics draining the dural sac and its surroundings run anteriorly from each intervertebral foramen and empty into the longitudinal channels mfront of the vertebral column.

Applied aspects of anatomy of epidural blockade: The epidural space is not as voluminous as the subarachnoid space; nevertheless, it extends from the base of the skull to the sacrococcygeal membrane and has direct communications with the parave/tebral space and indirect communications with the cerebrospinal fluid. It also leads directly to the vascular system by way of its large epidural veins; this is a potential direct route to the brain for drugs, air or other material inadvertently injected into an epidural vein. Within the cranium there is no epidural space, as meningeal dura and endosteal dura are closely adherent, except where they separate to form venous sinuses. At the foramen magnum these two layer separate, the former becoming the spinal dura and later the periosteum of the spinal canal Thus although - local anaesthetics cannot enter between the endosteal and meningeal layer of the cerebral dura. They can diffuse across the spinal dura at the base of the brain into the cerebrospinal fluid and then to the brain. Between the spinal dura and the spinal periosteum lies the epidural space.

The ligamentum flavum completes the posterior wall in direct continuity with the periosteum of the spinal canal. Since the spinal canal is approximately triangular in cross section and articular processes indent the triangle. The epidural space narrows

posteriorly and then widens again laterally towards the intervertefaral foramina. Thus the safest point of entry into the epidural space is the mid line.

Size of the epidural space:

The distance across the circular peridural space is variable. It is negligible or almost non-existent in the anterior region. It is more and readily measurable in the posterior region, especially in the midline.

	Epidural Space (mm)	**Thickness of Dura (mm)**
Cervical	1.0 – 1.5	2.0 – 1.5
Upper Thoracic	2.5 – 3.0	1.0
Lower Thoracic	4.0 – 5.0	1.0
Lumbar	5.0 – 6.0	0.66 – 0.33

Phsiological Aspects :

Haldt and Moloney were the first to describe negative pressure in the epidural space in 1928. This negative pressure is maximum at points of firm attachments. It is maximum in the thoracic region, less in the lumbar regions and least or absent in the sacral region

Lower Lumbar - 0.5 cm H_2O

Upper Lumbar - 1.0 cm H_2O

Thoracic - 1.0 to 3.0 cm H_2O

(Average 20 cm of H2O)

Identification of epidural space : There are several methods to identify the epidural space. These methods take advantage of the potential negative pressure or use **of the** sudden disappearance of resistance when the ligamentum flavum is penetrated.

Negative pressure technique:

1) Hanging drop sign (Gutiriez sign)

2) Capillary tube method

3) Manometer technique

Loss of resistance technique:

1) Syringe technique

2) Spring loaded syringe

3) Balloon technique (Macintosh's extradural space indicator)

4) Brooks device

5) Vertical tube of Dowkins

Others:

- Ultrasonic localization
- Oxford epidural space indicator

Negative pressure in the epidural space:

Originally, Heldt and Maloney described a negative extradural pressure in 1828. It is greatest in the thoracic region since the intra thoracic pressure is transmitted via paravertebral space less in the lumbar and least or absent in the sacral region. In the lower lumbar region it amounts to about 0.5cm of water. In the upper

lumbar region it about -1.0 cm of water and in the thoracic region it varies from -1.0 to -3.9 cm of water.

There are three theories explaining this negative pressure.[15]

1) The transmission theory:

Negative pressure in the epidura! space is caused by the transmission of the intrapleural negative pressure through the intervertebral foramina (mountos in Beyer Smith 1947). It varies with the depth of respiration i.e. clinically this negative pressure will be diminished or absent if the patient is not relaxed and if there is straining. Marked flexion at the spinal column increases the negative pressure and is more in young people. A rise in negative pressure may favour the spread of local anaesthetic solution in the epidural space.[15]

2) Cone theory:

This says that the epidural needle introduced depresses the dura, thus creating a large space, it is thus an artefact caused by the indentation of dura by the advancing needle (Janzen 1926).

3) Flexion theory:

This theory says that, more the flexion of spine, the greater the negative pressure (Oden 1938).

Thus a good knowledge of the anatomy of the epidural space, the nearby structures, factors influencing the negative pressure, along with precision adequate timing, proper positioning of the patient and careful identification of the epidural space goes a long way in proper performance of the technique of epidural block.

In pregnant women, identification of epidural space is difficult due to lordosis and sacral edema. The capacity of the space is reduced due to increased abdominal pressure, exaggerated lumbar lordosis engorged epidural veins and results in greater spread of local anaesthetics.

Local anaesthetic injectd into the epidural space acts on the dorsal root ganglia and spinal roots with their dural cuiffs in the extradural space.

- Leakage by vascular absorption
- Leakage through intertebral foramina
- Diffusion through dural root sleeves.
- Diffusion through dura mater.

COMBINED SPINAL EPIDURAL TECHNIQUE

Concept of CSE :

The concept of CSE has come into existence with the aims to provide the benefits of spinal block along with flexibility of an epidural catheter so as to modify and prolong the block for a longer period.

CSE can be used to reduce or eliminate the disadvantages of spinal and epidural anaesthesia, while preserving their advantages.

Application of CSE:

The CSE technique has been used for orthopaedic and trauma surgery of lower limb, general surgery, urologic surgery, gynaecologic surgery, caesarcan section, management of labour pain and postoperative pain. CSE blocks have also been used as research tools for controlled comparison between different epidural and subarachnoid techniques. Furthermore the technique has been used successfully in all age groups, including preterm neonates and infants, the very old, and other high-risk patients.[16]

Advantages of the combined spinal epidural :[11]

The advantages of the combined spinal epidural over an epidural alone are related to the additional use of the spinal component.

Spinal anaesthesia has the advantage of being:

1. Rapid in onset
2. More reliable than epidural anaesthesia, producing excellent analgesia
3. Having lower dose requirements thus preventing toxic systemic effects

4. Modifiable to ensure minimal motor and sympathetic block and cardiovascular stability

5. Definite end point for needle placement

The disadvantages are:

1. No top up facilities to prolong or optimise the block unless a subarachnoid catheter is inserted and these microcatheters are vulnerable, difficult to insert, expensive as well as being implicated in cauda equina damage.

2. Deliberate breaching of the dura with the potential of post dural puncture headache (PDPH)

Epidural Anaesthesia has the advantage of:

1. Widespread use
2. Familiar technique
3. Indwelling catheter allows top up doses, modification and extension of block

4. Less dramatic and slower onset of hypotension compared to subarachnoid anaesthesia

5. Absence of PDPH unless accidental dural tap.

The Disadvantages are of epidural Anaesthesia are:

1 Slow establishment of block

2 May be patchy or asymmetrical

3 Large volumes of local anaesthetic required

4 Some nerve roots are difficult to block

Combined spinal and epidural anaesthesia (CSEA) thus offers:

1 Speed of onset

2 Better quality of analgesia

3 Lower total dose of local anaesthetic

4 Presence of an epidural catheter to add locals anaesthetic and/or other drugs for optimisation and prolongation of spinal block.[11]

Characteristics of CSE block :

CSE is a multicompartment block. It CSE involves intentional dural puncture followed by epidural drug administration. This introduces the possibility of drug flux from the epidural to the subarachnoid space which may alter the characteristics of the block.[16]

Subarachnoid pressure is normally regarded as greater than epidural pressure by 5-15cm H_20. This pressure gradient is an obstacle to drug flux into the subarachnoid space. The epidural pressure rises transiently but dramatically, after drug administration. The similar rise in subarachnoid pressure occurs there is a brief period during which epidural pressure may exced subarachnoid pressure this produces conditions that would allow drug flux into the subarachnoid space.[16]

Drug flux increases with the size of the needle used and is determined more by size of the hole than by the physiochemical properties of the drug. The drug transfer depends in part on how well the drug crosses intact meninges.

In conclusion, epidural drugs should be administered with particular care following CSE. Some drug will transfer through the dural puncture site;

the clinical importance of this will depend on the spinal needle used, the drug, its volume and concentration. The most marked clinical effects are likely when dural puncture with a large spinal needle is followed by administration of a high concentration drug of low dural penetrance near to the dural puncture site.

Similar clinical effects arise from unrecognized accidental dural puncture with the epidural needle and epidural catheter misplacement or migration. Infusions of low concentration of local anaesthetic are therefore safer than high concentration boluses.

CSE is a more complicated technique than either block alone and produces a multicompartment block. This introduces the potential for new complications and the modifications of existing complications. CSE cannot be considered as simply a spinal block followed by an epidural block. Epidural injection may modify the spinal block and epidural drugs may not behave, as they would without prior dural puncture.

Techniques of needle insertion and variation :

Several CSE techniques are described, probably in part because no single technique is entirely satisfactory. Techniques have been varied and modified in order to increase success and avoid potential or actual complications.

Classification of CSE techniques[19] :

The various options in CSE can be classified broadly according to the number of interspaces used for performing the procedure. They can be further classified according to the type of needle used and the approach.

- **Single segment**
  - **Needle-Through-Needle**
    - Without spinal needle Support
      - Median approach
      - Paramedian approach
    - With spinal needle support
      - Spinal needle sleeve
        - Median approach
        - Paramedian approach
      - Spinal Needle Lock
        - Median Approach
        - Paramedian Approach
- **Needle-Beside-Needle (Double Barrel/Lumen)**
  - Without Attached Spinal Needle Guide/Separate Needle
    - Median Approach
    - Paramedian Approach
    - Combined median/paramedian approach
  - With attached spinal needle guide
    - Guide alongside epidural needle
      - Median approach
      - Paramedian approach
    - Guide incorporated within Epidural needle
      - Median approach
      - Paramedian approach
- **Double segment**
  - Median approach
  - Paramedian approach

1) Single pass:

Soresi described performing epidural injection and then advancing the same needle into the subarachnoid space. Sprotte described a similar technique.

No catheter was used and it is difficult to imagine what advantages these techniques have over pure subarachnoid blockade.[11]

2) Needle through needle :

The epidural space is identified by a conventional epidural needle and technique. A spinal needle is then passed through the epidural needle and beyond its tip, until the dura is punctured. Subarachnoid drug is administered and, after removing the spinal needle an epidural catheter is placed. Needle through needle CSE requires that subarachnoid blockade is initiated before epidural catheter placement,[16] the technique may be performed with a normal epidural needle and a long spinal needle. A large number of commercial kits are available, designed specifically for needle through needle CSE. It is the most widely reported CSE technique in the literature and is likely to be the most frequently used.

Separate needle :

With this technique, subarachnoid injection and epidural catheter placement are performed through different needles.

Separate interspaces :

Brownridge's first report of CSE in obstetric anaesthesia described epidural catheter placement at L_{1-2} followed by subarachnoid block at L_{3-4}.[18] This allowed the epidural catheter to be placed and tested before subarachnoid block was initiated, which is not possible with needle-through needle CSE.

Single interspace :

Using two interspace implies two separate local anaesthetic injections and this can be avoided by using a single interspace. Turner and Reifenberg reported the 'single space double barrel technique'. An epidural needle is sited followed by a spinal needle introducer at the same interspace. The epidural catheter is then inserted and spinal needle insertion follows. This was criticised as being 'inherently unsafe' because the spinal needle was introduced after the epidural catheter.[16]

Cook recently proposed a technique to avoid many of the practical problems of CSE. A spinal needle is placed as low as possible in the selected interspace and the CSF identified. The spinal needle stylet is replaced and an epidural needle is placed cephalad in the same interspace. The epidural catheter is then placed. The spinal needle stylet is removed and subarachnoid blockade performed. The technique allows placement of the epidural catheter before subarachnoid injection but does not require placing the spinal needle with an epidural catheter in situ. The technique has not yet been evaluated critically.[16]

TYPES OF NEEDLES :

Combined Spinal- Epidural needles :

- Tuohy; 1944 - modified the tip of the needle called Huber point also added a stylet to the risk of skin plugging in 1945.
- Huber needle; invented by Huber in 1953.
- Weiss; 1954- Introduced metal wings to the hub of the needle.

- Huberand Hanacka needle.
- Eldor needle.
- Eldor, Coombs and Torrieri needle.

Epidural needle[17] :

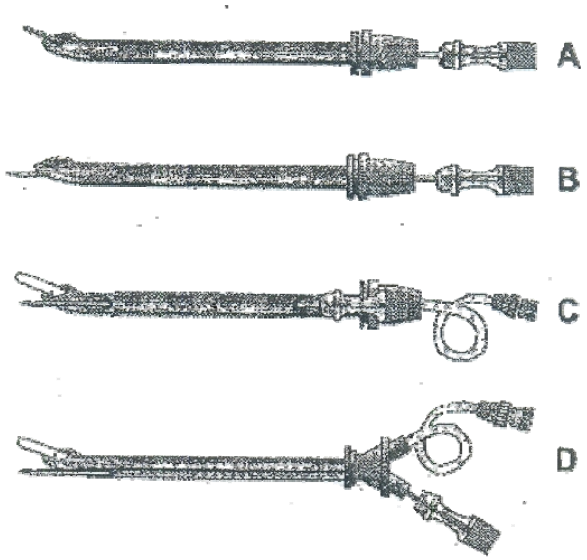


Fig. 5 : Different types of needles used for CSE techniques

A : Needle through needle, B : Huber and Hanaoka needle C : Eldor needle, D : Eldor, Coombs and Torrieri needle

Single Lumen Epidural Needles (Needle- Through-Needle technique) :

1. Epidural needle with single aperture : This most commonly used set consists of a conventional 16-18 gauge epidural needle to locate the epidural space. A spinal needle is then introduced through the epidural needle to exit it through the Huber eye and proceed to puncture the dural wall and enter the subarachnoid space.

2. CSE Needle with Additional Aperture (Huber and Hanaoka Needles): This is basically an epidural needle with an additional aperture ('back eye') situated at the end of the longitudinal axis.

Spinal needles[17] :

The gauge and type of the spinal needle influences the success of the procedure and incidence of outcome. The use of smaller gauge needles and ones with a conical tip reduces the incidence of post dural puncture headache. Lipov et a! demonstrated that the axial force needed to deform the sprotte needle tip was less than that needed for the Whitacre and Quincke needles of similar size. The larger distal side port of the Sprotte needle, as well as its greater distance from the tip, may account for the slightly greater deflection observed with Sprotte needles compared to Whitacre needles.

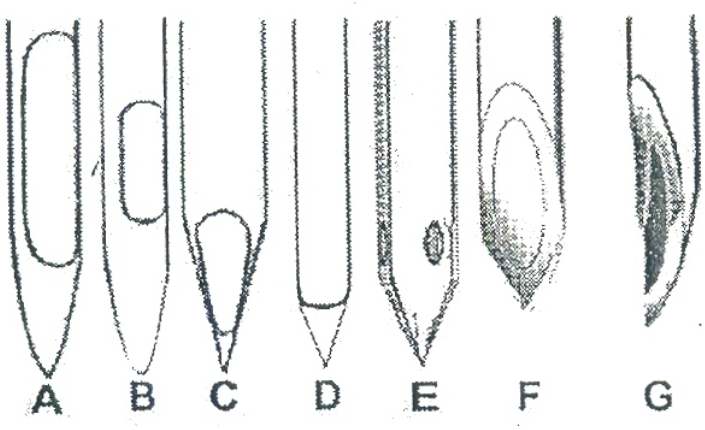


Fig. 6 : Tip of different types of spinal needles A : Sprotte, B : Whitacre, C : Modified pencil-point, D : Ball pen, E : Double-hole pencil-point, F : Quincke, G : Atraucan

Newer needles have been designed to improve efficacy. The atraucan needle has a double bevel with the distal cutting bevel making a cutting incision in the dura. The second part of this needle dilates this incision rather than cutting through the fibres leaving only a small hole in the dura. The sharp tip however is prone to damage. A new pencil-point spinal needle described is the double-hole pencil-point (DHPP) spinal needle, and is composed of a blunt

ogival tip and two circular holes opposing each other just proximal to the tip. The area of the two windows is almost the same as the single holed Sprotte needle's area, which enables more rapid CSF reflux. The anesthetic solution injection spreads through both windows. This allows a more diffuse anesthetic distribution and less anesthetic solution dosage. The DHPP spinal needle allows anesthetic solution injection even when a tissue fragment obstructs one of the holes. More recently, the ball pen needles have been marked in France. The tip of this needle is actually the tip of the stylet leaving a hollow cannula when the stylet is removed. The proposed advantages of this needle are that the tip of the needle is always completely in the subarachnoid space and on removal of the stylet there is no needle tip projecting beyond the orifice causing damage to the neurological tissue. There is no mechanical weakening at the tip caused by the presence of a lateral orifice. The open end of the needle allows laminar flow of cerebrospinal fluid, which results in faster identification of the subarachnoid space.

Optimal Length of the Spinal Needle Beyond the Epidural Needle Tips[17] :

The distance from the tip of the epidural needle to the posterior wall of the dural sac in the midline varies considerably among patients (0.3-1.03 cm). Further, the anteroposterior diameter of the dural sac varies considerably during flexion and extension of the spinal column. At $L_{3\text{-}L4}$, the diameter increases from a range of 9-20 mm in extension to a range of 11-25 mm in flexion. Additionally, these findings are only valid when the epidural puncture is performed in midline. This is because the dural sac is triangular with its base resting on the vertebral body and the triangle top points posterioroly to

ligamentum flavum. A minimum of 13 mm length of the spinal needle protrusion beyond the epidural needle tip is recommended for the CSE sets for a reasonably high success rate. Also, because of needle design, the length of protrusion for needles with side orifices should be greater than that for end orifice needles.

Needle through needle versus separate needle technique :

There are little data on the relative use of needle through needle and separate needle CSE.

In 1993, in Sweden, Holmstrom et al[16] found that 64% of the departments preferred separate needle CSE.

Lyons et al[19] compared needle-through-needle and separate needle CSE in a randomized study of 100 patients undergoing caesarean section. Separate needle CSE had a lower spinal failure rate (4% vs. 16%), was associated with less hypotension and took no longer than needle through needle CSE.

Casati et al[20] studied 120 non-obstetric patients randomly allocated to needle through needle or separate needle CSE performed in the sitting position. Failure of spinal anaesthesia (5%) and hypotension (23%) was higher in the needle through needle group than in the separate needle group (1.6% and 13%).[16]

The specialized equipment for needle-through-needle CSE is estimated to cost approximately 40% more than a standard epidural set and 27G pencil point needle needed for separate needle CSE.

Single segment technique (SST) vs. double segment technique (DST) :

The reduced number of skin punctures in SST may theoretically decrease the incidence of pain, infection at puncture sites, and formation of haematomas. The use of a tuohy needle as an introducer lessens the likelihood of contamination of the spinal needle with skin borne microorganisms.

In single space technique (SST), the Tuohy needle may function as a more suitable introducer than a normal introducer, as the tip of spinal needle can be directed more accurately. Dural identification may be complicated. One cannot be sure of dural penetration after a successful localization of the epidural space, as any deviation of the Touhy needle from the sagittal plane may cause the spinal needle to enter the epidural space and pass the dural sac laterally. There is 16% failure of spinal anaesthesia in single space technique compared to only 4% when using double space technique (DST).[21]

These problems associated with single space technique may be attributable to the lack of tissue support for the spinal needle. In this technique, the spinal needle is in contact with only dura and the Tuohy needle, which may increase the risk of displacement of the spinal needle.[22]

An epidural catheter may enter the subarachnoid space during CSE by passing through the known hole made by the spinal needle or through an unrecognized hole made by the epidural needle. Separate needle CSE where the epidural catheter is placed first or distant from dural puncture can avoid this problem but needle through needle CSE cannot. 'Backholes' and combined needles also aim to ensure that the epidural catheter reaches the dura at a point away from the dural puncture. Rotation of the tuohy needle through 180 degree

after spinal placement has been advocated to redirect the epidural catheter away from the dural hole but rotation increases the ease of dural puncture. Rotation of the epidural needle within the epidural space cannot be recommended.[16]

When subarachnoid block is established before placing an epidural catheter a conventional epidural 'test dose' cannot be interpreted. The problem cannot be avoided using needle through needle CSE but may be if separate needle CSE is used and the epidural catheter is placed before subarachnoid block or if a modified needle through needle technique is used.

When subarachnoid block is initiated before placing and securing the epidural catheter, there is inevitably some delay in attending to the developing neural blockade. Holmstrom et al found a higher level of blockade when comparing spinal and CSE in the sitting position using plain bupivacaine. They attributed this finding to the extra time the CSE group sat in this position, "three to four minutes longer to allow introduction of epidural catheter". Unexpected delay may occur due to blood entering the catheter, difficulty in advancing the catheter or paraesthesia during advancement Delay between subarachnoid injection and completing the CSE will then be considerable. This exposes the patient to the problems of the developing subarachnoid blockade while the anaesthesiologist's attention is on the procedure and risks of cardiovascular destabilization or block failure.

Hyperbaric local anaesthetic solution arc frequently used and any delay in positioning the patient can lead to unilateral or saddle block depending on the patient's position.[16]

TECHNICAL COMPLICATIONS :

Failure of spinal component:

There are many reasons that may explain failure of the needle through needle technique.

1. Too short a spinal needle will not extend for enough beyond the tip of the epidural needle to reach the dura.

2. Excessively long spinal needles pose problems of handling and depth of placement. During needle through needle CSE, the spinal needle is only anchored by the dura and needle movement is difficult to prevent. Long fine needles have high internal resistance leading to delay in reflux of CSF,[23] difficulty in stabilizing during injection,[24] and increasing the risk of loss of drug during injection. A long spinal needle may enter the subarachnoid space then pass through it and out through the anterior dura. Anterior dural puncture may also occur if a blunt spinal needle causes apposition of posterior and anterior dura,[21] the needle passing through the dural sac without encountering CSF.

3. Deviation from the midline will lengthen the epidural dural distance and a Huber tip may exacerbate this.[25] The dural sac is triangular in the lumbar region with the apex pointing posteriorly. Small degrees of lateral deflection allow the spinal needle to pass by.

4. Very fine pencil point needles (28G and smaller) may lack the rigidity and sharpness to puncture the dura.

5. Lack of appreciation of dural puncture (poor feel) is common with needle through needle CSF.

Complications of needle-through-needle :

The possibility of spinal needle damage during contact with the epidural needle has been suggested in needle through needle CSE. Friction between spinal and epidural needles generates metallic fragments, which are then introduced into the subarchnoid space. This is a probable cause of aseptic meningitis following CSE.[26]

Delay in completing epidural catheter placement

Postdural puncture headache :

Is a theoretical risk, use of small gauge atraumatic (pencil-point) spinal needles such as Sprotte, Whitacre, or Gctie Marx, will greatly reduce the incidence of PDPH.

Midline approach to the subarachnoid space results in greater leakage of CSE than paramedian approach. A paramedian approach, by creating a dural flap may also reduce the incidence of post dural puncture headache.

High spinal or inadvertent total spinal :

High spinal anaesthesia with respiratory and vascular embarrassment can occur in any patient. However, the patients more susceptible to high spinal are parturients. This may result from a number of factors;

1. Decreased CSF volume.

2. Use of hyperbaric solution in the presence of an exaggerated lumbar lordosis.

3. Position of the patient as in steep head-low tilt.
4. Increased neuronal sensitivity to local anaesthetics.
5. Ventilatory insufficiency due to enlarged uterus.

High spinal anaesthesia is most likely to occur shortly after the induction of spinal anaesthesia. It is important to closely monitor the patient during and following the induction of spinal anaesthesia until the level of spinal anaesthesia has significantly regressed.[27]

Most patients will become agitated with high spinal. Nausea and hypotension are common. These symptoms should alert the anaesthcsiologist to the possibility that the spinal higher than desired level, the patient should be given oxygen. Ask the patient to take deep breaths and observe for the movements of the intercostals muscles and the movement of the reservoir bag. Inadequate ventilation may require induction of general anaesthesia and assisted ventilation of the lungs following endotracheal intubation.

Nerve injury :

During placement of a needle in the subarachnoid space, needle can come in direct contact with section of the cauda equina or the nerve roots themselves. The incidence of postoperative nerve injury is thought to be less than 1:10,000 spinal anaesthetics. The nerve injuries that do occur tend to consist of persistent paresthesia that resolves without treatment within weeks or months.

Transient neurological symptoms :

Transient neurological symptoms (TNS) were first formally described by Schneider et al[28] and reported four cases of severe radicular back pain occurring after resolution of hyperbaric lidocaine spinal anaesthesia. All four patients had undergone surgery in the lithotomy position. No sensory or motor deficits were detected on examination, and the symptoms resolved spontaneously within several days.

The incidence of TNS has ranged between 0% and 37%. A 16% incidence of TNS in patients receiving either hyperbaric 5% lidocaine with epinephrine or 2% isobaric lidocaine has been reported. However, no patient receiving 0.75% hyperbaric bupivacaine developed TNS. The incidence was higher among patients positioned with knees or hips flexed (genitourinary arthroscopy) than patients positioned supine (mesh repair), presumably because of flexion results in additional stretch on the nerve roots. The incidence of TNS with lidocaine (12%) was significantly higher than that with trocaine (1.6%) or bupivacaine (1.3%). This suggests that the risk of TNS is high among patients in the lithotomy position (24.3%) and low for patients undergoing surgery in positions other than lithotomy (3.1%).

Catheter complications :

Insertion of plastic catheter into the epidural space is usually a simple and safe procedure. Complications related to catheter insertion may occur although the incidence is very low.

1. Catheters may be placed into the subarachniod space.

2. Spinal migration of epidural catheter : This may occur in the needle through needle technique but is rare. Holmstrom and colleagues, reported that even under direct visualization using cpiduroscopy it was impossible to force an epidural catheter into the subarachnoid space after a single perforation of the dura and a small gauge spinal needle.[21] However, with multiple dural punctures and the spinal needle the incidence of catheter migration is increased by 5%.

3. Shearing off of a segment of the catheter in the epidural space. This usually occurs if an attempt is made to withdraw the catheter backward from the epidural needle. One must never withdraw a catheter backward through the epidural needle. If it is not possible to advance the catheter into epidural space, then the needle and the catheter should be withdrawn together, and the procedure repeated at another interspace.[29]

4. Epidural catheters can curl and turn on themselves and form a knot in the epidural space.[21]

Epidural haematoma :

Epidural haematomas may present as neurological deficits in the postoperative period due to cord compression. On insertion of a catheter in an area at high risk for arterial contamination such as the sacral hiatus may also increase the risk of abscess formation, emphasizing the importance of meticulous aseptic technique.

Anterior Spinal Artery Syndrome :

Anterior spinal artery thrombosis causes a syndrome consisting primarily of lower extremity paresis with a variable sensory deficit, usually diagnosed in the postoperative period as the neural blockade resolves. The etiology of this problem is uncertain. Though direct trauma to the anterior spinal artery and ischemia secondary to hypotension or vasoconstrictors may be causative factors.

Vascular injury :

Injury to blood vessels in the performance of spinal anesthesia can be associated with serious complications, including epidural haematoma due to continued bleeding from the epidural venous plexus. This reported most commonly in patients with coagulopathy or those who have been taking anticoagulants, though it is possible in patients with no apparent risk factors.

Mechanism and pathway of parturition pain :

Pain is transmitted from the periphery by small 'A' delta and C fibers, the cell bodies of which lie in the dorsal root ganglia[30]. From the dorsal horn central projections enter the gray matter. Except for a few A delta fibers that terminate in the marginal layer (Lamina I) the remainder synapse in the substantia gelatinosa (Lamina II), communicating by a series of interneurons with cells whose bodies reside in lamina V. These wide dynamic range neurons respond to both high intensity stimuli provoked by pain and also to low intensity light touch. Increased activity in these neurons results in impulse transmission in the anterolateral ascending columns.

Substance-P together with other peptides acts as neurotransmitter in the pain pathway. It is found in the cell bodies of dorsal root ganglia and released in the substantia gelatinosa in response to painful stimuli. The activity of a series of interneurons in the Lamina II inhibits substance-P release. These interneurons are activated by collaterals from the large sensory fibers and also by descending inhibitory fibers in the dorsolateral funiculus.

Stimulation of inhibitory neurons or opioid receptors in the substantia gelatinosa acts principally by reducing cyclic AMP levels in the opioid sensitive cells, resulting in presynaptic inhibition of release of substance-P and also by postsynaptic hyperpolarization of the dorsal horn neurons. Thus the increase in activity in Lamina V neurons and in the anterolateral ascending columns is prevented. Opioids are more effective in blocking activity produced by C than A delta fibers.

Sensory pathway:

Sensory stimuli from the uterine body are transmitted by visceral afferents that accompany visceral sympathetic nerve fiber through the uterine plexus, pelvic inferior hypogastric and Frankenhause's plexus. Then the impulses travel through the middle and superior hypogastric plexus and aortico- renal plexus to enter lumbar and lower thoracic sympathetic chains to communicate with $T_{10,11,12}$ L_1 spinal nerves. They finally pass through the posterior roots of these nerves to make synaptic contact in dorsal horn through lamina V.

Sensory stimuli from the cervix pass through pelvic plexus along the pelvic parasympathetic nerves to sacral segments $S_{2,3,4}$ of the spinal cord.

Sensory stimuli from the upper vagina pass to $S_{2,3,4}$ sacral parasympathetic segments and from lower vagina pass through pudendal nerve.

Perineum receives motor and sensory innervation from $S_{2,3,4}$ through pudendal nerve. Branches of ilioinguinal (L_1), genital branch of genitofemoral nerve ($L_{2,3}$) and posterior cutaneous nerve of thigh ($S_{2,3}$) carry impulses from the perineum and labia majora. Ovarian plexus which runs along the ovarian vessels also carries afferent impulses from the uterus.[31]

Motor nerve supply:

Uterus receives both sympathetic and parasympathetic nerve fibers. Sympathetic nerve fibers arise from lower thoracic and upper lumbar segments of spinal cord and pass through the ganglia of the sympathetic trunk to aortico-renal plexus, superior hypogastric plexus, inferior hypogastric plexus and

pelvic plexus to continue as paracervical plexus on each side of the cervix. Parasympathetic nerves (Nervi erigentes) arise from $S_{2,3,4}$ spinal segments and join the pelvic plexus.

Uterine activity is predominantly under hormonal and humoral influence. Role of motor nerve supply in regulation of uterine activity in labour is doubtful. Severe hypotension caused by widespread sympathetic block may affect uterine activity by impeding hormonal delivery to uterus.[31]

Labour – Anatomical and Physiological Aspects

Labour is divided into 3 stages

The first stage: Begins with the onset of regular painful uterine contractions and ends at full dilatation of the cervix. Average duration in the primigravida is 8-10 hours and in multigravida 6-8 hours.

The first stage of labour is divided into latent phase when the cervix slowly dilates upto 3 cm and an active phase when the cervix dilates from 3 cm to 10 cm. The active phase is further divided into early active phase when the dilatation is upto 7 cm and the late active phase to full dilatation. During the active phase, the cervix dilates at the rate of about 1 cm per hour.

The second stage: Extends from full dilatation of the cervix to the birth of the foetus and varies from a few minutes to about 2 hours. It is divided into the perineal stage and the expulsive stage.

The third stage: Is the period after the birth of the foetus to the expulsion of the placenta and membranes. The average duration is about 5 minutes[32]

Prelabour:

Prelabour is defined as the development phase of preparation for parturition. It occurs several weeks before the onset of true labour. The cervix begins to soften and dilate. Progesterone, oxytocin, prostaglandins (PGE_2 and $PGF_{2\alpha}$), cortisol, prostacyclin, interleukin 8 and monocyte chemotactic peptide interact with each other producing the prelobour softening of the cervix.

The mother, the foetus and the placenta all contribute to the maintenance of pregnancy, the initiation of labour and finally the birth of the foetus. The key component in the initiation of labour is the foetal brain which influences the foetoplacental unit via the hypothalamopituitary – adrenoplacental axis.

The increase in the oestradiol levels at term causes a shift in the oestrogen to progesterone ratio in favour of estrogen, which leads to increase in the oxytocin sensitive receptors in the myometrium and decidua and activates amnion to produce prostaglandin. $PGF_{2\alpha}$ is responsible for myometrial contractility. PGE_2 is essential for cervical ripening. The main sources of these are the decidua for $PGF_{2\alpha}$ and amnion for PGE_2.

Uterine changes:

The uterus is composed of smooth muscle (myometrium). During the transition from pregnancy to labour, the myometrial oxytocin sensitive receptors increase. There is increased frequency and amplitude of contraction of the myometrium and also increase in the concentration of myometrial gap junctions between these smooth muscle cells thereby facilitating synchronization.

Cervical changes:

The cervix is mainly made of connective tissue collagen. Gradual softening of the cervix occurs 4 weeks prior to onset of labour (prelabour). This involves degradation of stromal collagen by changes in its proteoglycan complexes and increase in the water content of the ground substance leading to reduced tensile strength which eventually results in effacement and dilatation.[33]

Foetal membranes :

The decidua and the amnion are the main sources of arachidonic acid, the main precursor of prostaglandin. The decidua also produces prostaglandin synthetase enzyme.

Mechanisms of labour pain:

The uterus is made up of bundles of smooth muscle cells separated by thin sheets of connective tissue. The proportion of muscle cells to connective tissue varies from the body of the uterus to the cervix[33].

The pain of labour is an intermittent, crescendo pain. The intervals between painful episodes become shorter as labour progresses and the pain increases in frequency, duration and severity. The pain from the reproductive system can be divided into 2 components - Somatic and Visceral

Somatic sensations are evoked by stimulation of skin and underlying tissues and the superficial nerves involved are described by dermatomes. The visceral component originates from the internal organs and the only sensation felt is pain[34].

Labour pains during the first stage of childbirth are due to dilatation of the cervix and the lower uterine segments as well as the associated uterine contractions.[35] The visceral pain is referred to T10,11,12 and L_1 dermatomes as the Lamina V of the dorsal horn synapse with cells which also receive afferents from areas of the skin supplied by the same segments of the spinal cord. These are blocked by opioids. As the presenting part descends, compression of other viscera such as the bladder and the rectum adds to the pain. Once the cervix is fully dilated, the amount of painful stimuli originating in this structure decreases but uterine contractions continue and pressure effects in the pelvis and perineum become significant.

The pain during the second stage of labour results from distention of the vagina, pelvic floor and perineum with the descent of the foetal presenting part. The pain is transmitted by thin myelinated, rapidly conducting A-delta fibers via the pudendal nerve-S2,3,4 pathway. Somatic pain is sharp and well localized to vagina and perineum. Local anaesthetics provide dense somatic analgesia

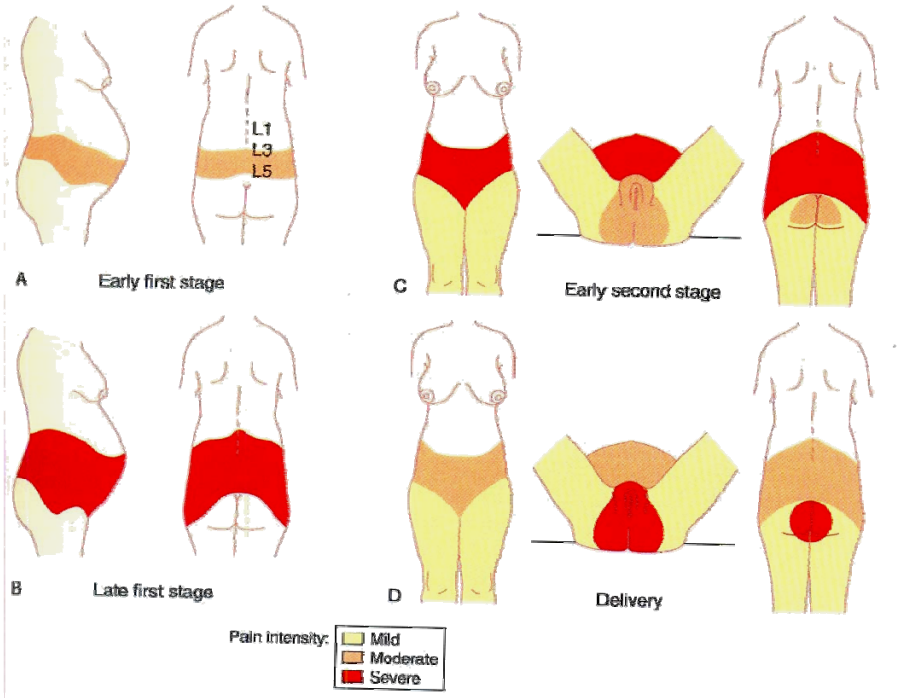


Fig 7 : The pants and stocking distribution of pain during labour

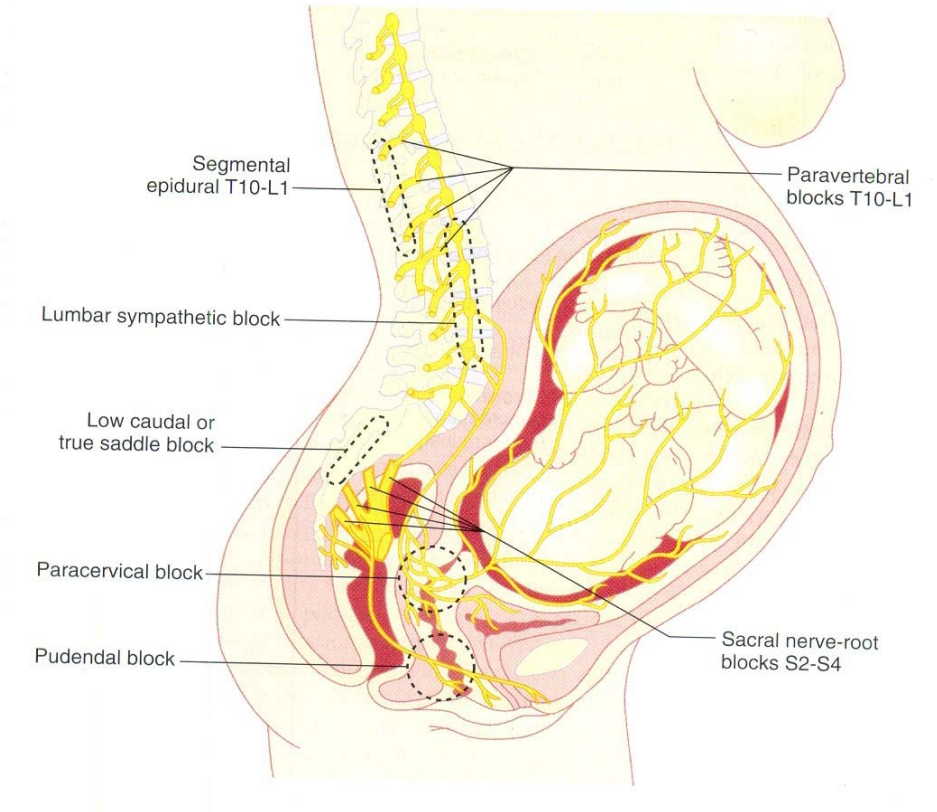


Fig. 8 : The sensory pathways involved in parturition

Summary of labour pain in the first and second stages:

I stage :	**Origin :**	Cervix (visceral)
	Quality :	Dull and aching
	Site :	Poorly localized to the lower abdomen. Backache or lumo-sacral discomfort
	Pathways :	Autonomic via T_{11}
II stage :	**Origin :**	Mixed visceral uterus) and somatic (perineal and pelvic structures, including joints)
	Quality :	Dull and sharp
	Pathways :	Autonomic : Parasympathetic and sympathetic plexuses Lumbar and sacral spinal segments
	Site :	Somatic component localized to the perineum Referred pain felt in any corresponding spinal segment from T_{11} to S_5

Central pathways

The nociceptive information is transmitted through A delta fibers and C-fibers to the dorsal horn. Synapses and fibers cross to relay in the thalamus and cortex. Modulation of this activity occurs both centrally and peripherally. Peripherally in the uterus local release of bradykinin, 5 – hydroxytryptamine and prostaglandins can amplify peripheral nerve activity.

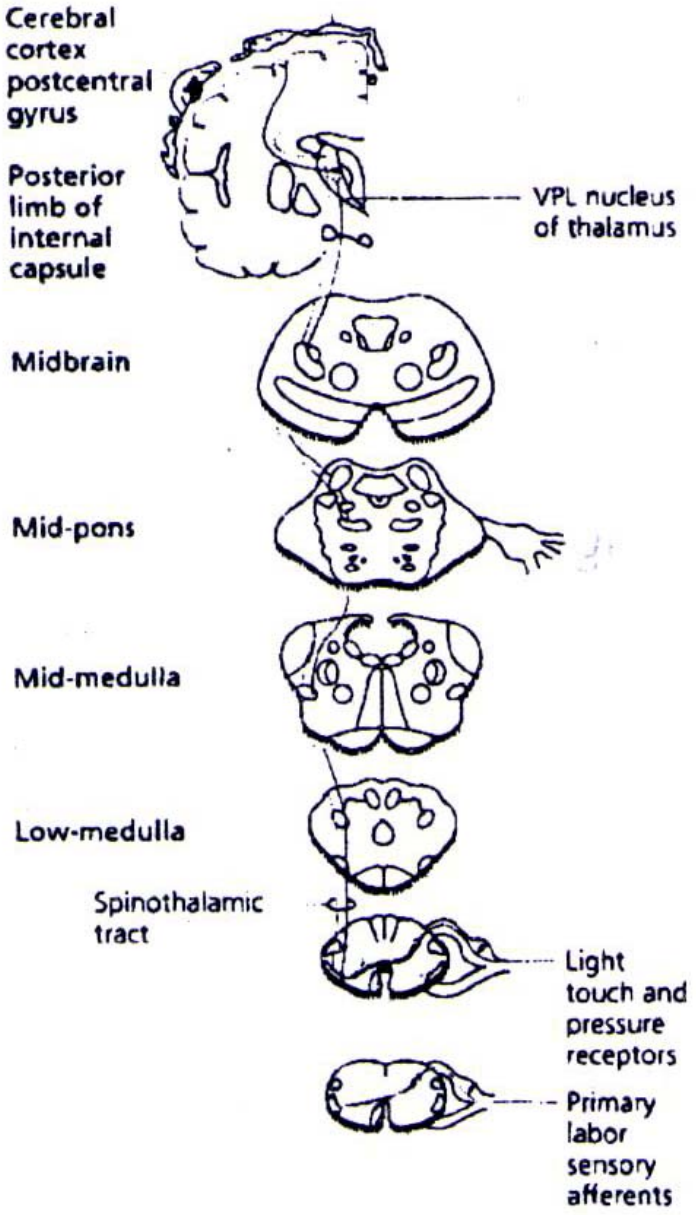


Fig. 9 : Cephalad extension of labour sensory pathways

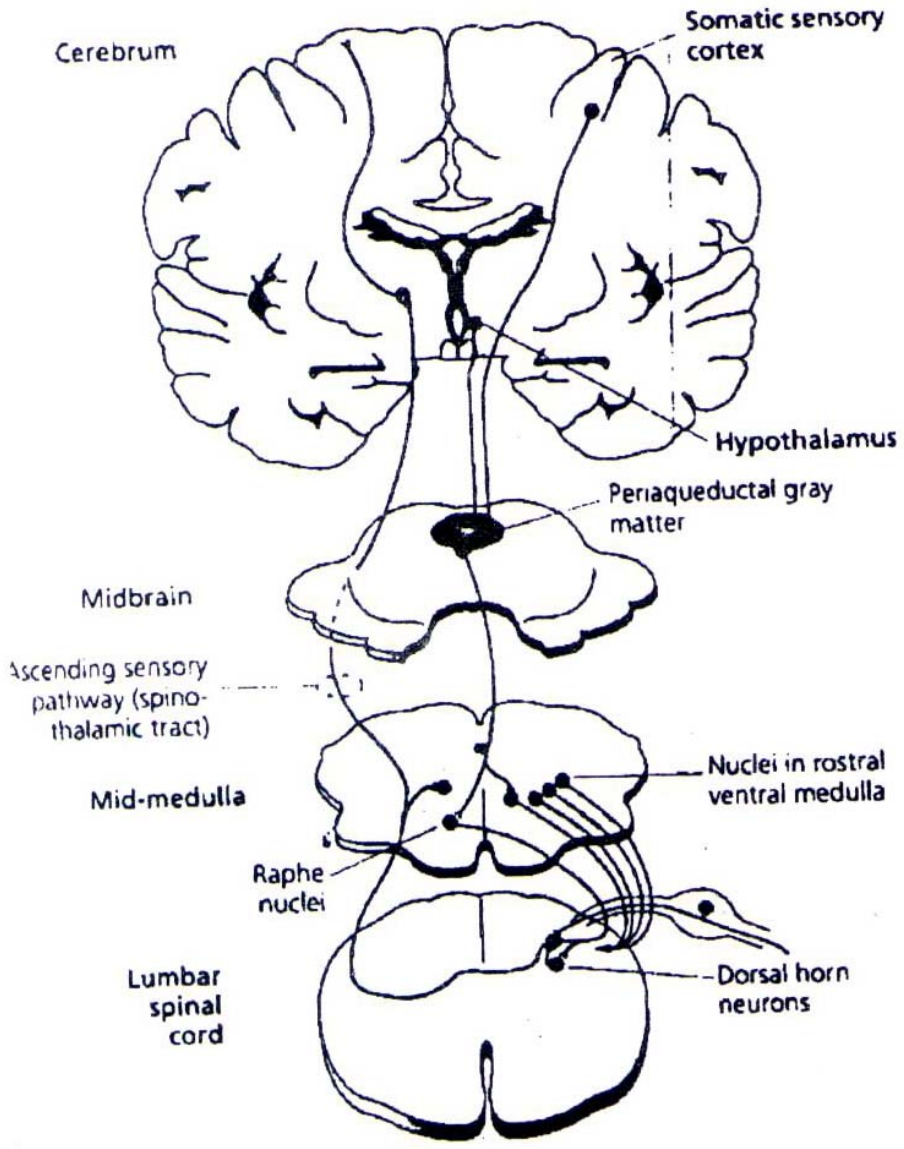


Fig. 10 : Descending inhibitory pathway

In the spinal cord, convergence of information from somatic and visceral structures occurs at the same spinal segment. This is one of the mechanisms for the referred pain from viscera to body wall dermatomes. The laminae activated by visceral nociceptive fibers also receive an input from a number of other segments in the spinal cord, so that the sensation produced is diffuse. Cervical pain is therefore dull, centrally placed in the part of the body wall innervated by T_{11}. Anteriorly, this lies just below the umbilicus. Posteriorly, the T_{11} dermatome innervates skin over the lumbo-sacral junction.

Nociceptive visceral pathways from the dorsal horn relay on anterior horn cells. They also pass cephalad by many interconnecting pathways such as the spinothalamic tract and the spinoreticular tract, the solitary nucleus and the dorsal column. The spinothalamic tract (rapid impulse conduction) and the spinoreticular tract (slower impulse conduction) are responsible for the emotional responses to pain and are partially responsible for the analgesia produced by descending control system activity.

Midbrain structures (periaqueductal grey matter) are sensitive to opioids and project to the dorsal horn of the spinal cord with adrenergic or serotonergic neurons. Pain modulation occurs in the spinal cord by enkephalinergic interneurons directly inhibiting nociceptive transmission. Pregnancy itself induces opioid-mediated analgesia.[2]

The aim of pain relief is to obtund pain. Regional nerve blocks are effective if part of the dorsal column pathway is anaesthetized.

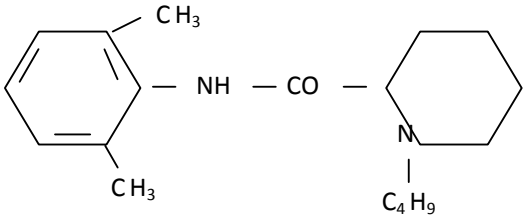
PHARMACOLOGICAL REVIEW

PHARMACOLOGY OF BUPIVACAINE:

Bupivacaine was synthesized by Ekenstam in 1957 and used clinically in 1963**.**

It is a synthetic long acting amide local anaesthetic.

Structure:



It is a 2-piperidine carboxamide 1- Butyl N (2, 6, di methyl, phenyl) mono hydrochloride, monohydrate.

Molecular weight: 288.

pKa – 8.1.[36]

Pharmacokinetic Properties:

Absorption: the absorption is related to

- The site of injection (Intercostal >Epidural > Brachial plexus > Subcutaneous).
- A linear relationship exists between the total dose and the peak blood concentration achieved
- The drug is highly lipid soluble and therefore uptake into fat is rapid and the drug has a direct vasodilator effect.

Distribution: 95% of the drug is bound to alpha- 1- acid- glycoprotein in the plasma The V_D is 4L and the Foeto / maternal ratio is 0.2 to 0.4.

Metabolism: It is metabolized in the liver by N-dealkylation and is conjugated with glucuronic acid to 2, 6, pipecolyloxylidine. N-desbutyl bupivacaine and 4 hydroxy bupivacaine are also formed. Hepatic disease potentiates its toxicity.

Excretion: 5% of the dose is excreted in the urine as pipcolyloxylidine; 16% is excreted unchanged. The clearance is 0.47 L / min.

Presentation:

Vials of 20ml containing a clear colourless solution of 0.25% / 0.5% Bupivacaine hydrochloride.Vials containing 0.75% of Bupivacaine are also available (presently not in India).20 ml vials of 0.25% - 0.5% Bupivacaine without preservative are also available.

Ampoules containing 4ml of 0.5% (heavy) solution with dextrose for spinal anaesthesia.

Mode of action:

Bupivacaine diffuses in its uncharged base form through neural sheaths and the axonal membrane to the internal surface of cell membrane sodium ion channels where it combines with hydrogen ions to form a cationic species which enters the internal opening of the sodium ion channel and combines with a receptor. This produces blockade of the sodium ion channel, thereby decreasing sodium ion conductance and preventing depolarization of the cell membrane. Thus it blocks the generation and conduction of nerve impulses by

increasing the threshold of electrical excitation in the nerve, by slowing the propagation of nerve impulse and by reducing the rate of rise of action potential. At blood concentrations of 1 -2 mcg/ml achieved with therapeutic doses, no systemic side effects are seen.

Routes of administration and dose:

Bupivacaine can be administered topically, by infiltration, intrathecally or epidurally. Toxic dose is 2 mg / kg[37]

It is used in varying concentrations:

For spinal anesthesia it is used in the concentration of 0.5% with a duration of 75 – 150 min.

For epidural it is used in the concentration of 0.5% to 0.375%.Onset of action is 10 – 20 min and duration 180 – 300 min.

For infiltration- - 0.5% - 0.25%. Onset of action is rapid and duration is 200 min.

For peripheral nerve blocks – 0.25% - 0.5%. Onset of action is 10 – 20 min and duration of action is 400 min[37]

In obstetric analgesia it is used in concentrations of as less as 0.0625% to 0.25%.

At concentrations of 0.125% to 0.0625% sensory blockade predominates and at concentrations above 0.25% motor blockade is seen.

It should not be used in IVRA.

Average duration of action of epidural Bupivacaine is 120-180 minutes and 5 to 6 hours for nerve blocks.

Changes during pregnancy :

Increased sensitivity i.e., more rapid onset of block may be seen during pregnancy. Alterations in serum protein binding characteristics of bupivaciane may result in increased concentration of pharmacologically active unbound drug in plasma of parturient. Dose 2mg/kg.

Safe limit : upto 150mg in 4 hours.

CVS: Bupivacaine is markedly cardiotoxic as it binds specifically to myocardial proteins. In toxic concentrations the drug decreases the peripheral vascular resistance and myocardial contractility, producing hypotension and cardiovascular collapse.

CNS: The principal effect of Bupivacaine is reversible neural blockade which leads to a characteristically biphasic effect in the CNS. Initially, excitation (light headedness, dizziness, visual and auditory disturbances and fits) occurs, due to the blockade of inhibitory pathways in the cortex. With increasing doses, depression of both facilitatory and inhibitory pathways occur leading to CNS depression (drowsiness, disorientation and coma) Bupivacaine is more lipid soluble and protein bound. This limits its passage across the placenta to foetus. Bupivacaine is undetectable in neonatal plasma 24 hours after cesarean section using Bupivacaine induced spinal anaesthesia.[38.]

Toxicity / side effects:

Allergic reactions to the amide-type local anesthetic agents are extremely rare. Toxic levels in plasma are 2-4 μgm/ml. The dose required to produce toxicity in the foetus and newborn are much lesser than adults.

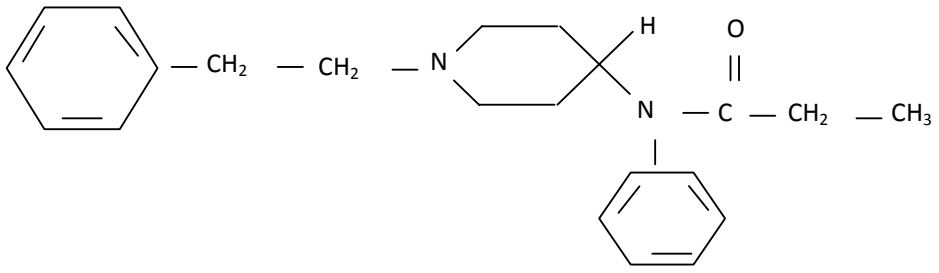
CVS toxicity includes atrio-ventricular block, ventricular arrhythmias and cardiac arrest. CC / CNS dose ratio is 3.7 ± 0.5.[37] Cardiovascular collapse is more difficult to resuscitate and pregnant women are more sensitive to the cardiovascular effects than non-pregnant women.

Bupivacaine is used in obstetric analgesia due to its longer duration of action, limited placental transfer, high degree of sensory block than motor block, less cumulation and no tachyphylaxis[39]

PHARMACOLOGY OF FENTANYL :

Fentanyl was first synthesized in 1960 by Dr. Paul Jannsen, a chemist working for a Belgian pharmaceutical company. It was released into clinical practice in 1963. Fentanyl is a synthetic opioid, a tertiary amine and a phenylpiperidine derivative.

Structure:



Presentation:

1. As a clear, colourless solution for injection containing 50μg /ml of Fentanyl citrate.

2. Transdermal patches which deliver 25/50/75/100 μg / hour over a 72 hour period.

3. Lollipop (Fentanyl citrate on stick) – dissolves slowly in mouth, available in 6 dosages, 200 to 1600 micrograms in 200 microgram increments (excluding 1000 and 1400 microgram).

Potency

Fentanyl is 1000 times more potent than Meperidine

50 –100 times more potent than Morphine

100 microgram of Fentanyl is equal to 10 mg of Morphine

75 mg of Meperidine[40]

Mode of action:

Fentanyl is a highly selective μ receptor agonist, which is mainly responsible for its analgesic properties. It acts by increasing intra-cellular calcium concentration which inturn increases K^+ conductance and hyperpolarization of cell membranes. This decreased membrane conductance decreases both pre and postsynaptic responses. Analgesia is produced principally through interaction with μ receptors at supra spinal sites. Fentanyl also binds to κ receptors causing spinal analgesia, sedation and anaesthesia.

Pharmacokinetics and Pharmacodynamics:

A single dose of Fentanyl administered IV has a rapid onset and shorter duration of action than Morphine. The effect site equilibration time between blood and the brain is 6.4 minutes. The greater potency and more rapid onset of action reflect the greater lipid solubility, which facilitates its passage across blood-brain barrier. Its rapid redistribution to inactive tissue sites accounts for its shorter duration of action. 75% of the initial dose undergoes first pass pulmonary uptake.

Effective analgesic concentrations are between 1 and 3ng/ml, while concentrations of 1.5 to 3ng/ml result in a 50% decrease in ventilatory response to carbon dioxide.

Metabolism:

Fentanyl avidly binds to alpha-1-acid glycoprotein and is also bound to albumin. It is metabolized in the liver to polar inactive metabolites by N-dealkylation, producing norfentanyl with subsequent hydroxylation to hydroxypropionyl derivatives. Cytochrome P–4503A4 plays the predominant role in Fentanyl metabolism[41].

Absorption and distribution:

It is absorbed orally and has a bioavailability by this route of 33%. It is 81 to 94% protein bound in the plasma. The V_D is 0.88 – 4.4 L/Kg.

Excretion:

10% of it is excreted in urine. Clearance is 0.4 – 1.5 L / min and elimination half-life is 1.5 – 6 hrs. Halothane decreases the clearance of Fentanyl by 48%, a similar effect occurs with Enflurane. Clearance is decreased in patients with hepatic involvement.

Analgesic potency:

- Minimal analgesic dose is 0.011 mg/kg. It is 29 times more potent than Morphine.
- Therapeutic index – 323 and pKa – 8.4.
- The onset of action and duration depends on the route of administration.
- IM – onset of action is 7-15 min, peaks at 15 mins and duration of action is1-2 hrs.
- IV- onset of action is 2-5 minutes and duration of action is 30-60 minutes.
- Epidural route – onset of action is 4-6 min, peaks at 5-10 min and duration of action is 2-3 hours.[41]

Effects:

CVS:

- In the dose of 1mcg/kg there is no significant effect on papillary muscle mechanics.

- Doses of 7mcg/kg at induction decreases heart rate but there is no change in mean arterial pressure.
- At 10mcg/kg myocardial contractility is reduced by 50%.
- 20 to 25mcg/kg decreases heart rate, MAP, systemic and pulmonary vascular resistance and PCWP by15% in patients with coronary artery disease
- At 75mcg/kg there is haemodynamic stability. There are clinical reports attesting to hemodynamic stability of high dose Fentanyl for both cardiac and noncardiac surgeries.
- It rarely causes histamine release.
- Fentanyl produces bradycardia of vagal origin.

RS:

- Fentanyl at 1 to 2 mcg/kg decreases respiratory rate and increases tidal volume.
- At doses greater than 3mcg/kg it decreases both the respiratory rate and tidal volume and also the ventilatory response to hypoxia and hypercarbia.
- It has an antitussive property.
- Chest wall rigidity ("Wooden – chest" phenomenon) due to its effect on μ receptors located on GABAnergic interneurons can be controlled by the early use of muscle relaxants.

- The side effect of great concern after epidural or spinal opioid administration is respiratory depression. Use of more lipid soluble opioids like Fentanyl decreases the potential occurrence of the problem.[27]

CNS:

It is a CNS depressant. At low doses (1-2 microgram/kg) it is devoid of hypnotic and sedative activity. Miosis is seen as a result of stimulation of Edinger Westphal nucleus. The central effects are markedly less after epidural injection than after IV administration.

GIT:

It increases the common bile duct pressure by causing spasm of the sphincter of oddi. It causes nausea, vomiting and decreases GI motility.

GU:

It increases the tone of the ureters, bladder detrussor muscle and vesicle sphincter causing retention of urine.

Metabolic / others:

At doses of 50-100mcg/kg it prevents increases in plasma epinephrine, cortisol, glucose, free fatty acids and growth hormone levels during surgery

Relationship between Fentanyl Plasma Concentration and Effect[37]

Plasma Fentanyl concentration (ng/ml)	Pharmacological effect
>1	Slight analgesia, minimal ventilatory depression
1-3	Analgesia; 50% decrease in the ventilatory response to carbon dioxide
4-10	Analgesia for surgery if combined with nitrous oxide
>20	Unconsciousness, satisfactory anesthesia if used as sole agent

Uses:

- To provide analgesic component of balanced anaesthetic technique for short surgical procedures in the dose of 2mcg/kg.
- High dose (50 to 100 μ/kg) Fentanyl anaesthesia with nitrous oxide/ oxygen or oxygen alone has been employed for cardiac surgery and long surgical procedures. Postoperative ventilation should be routinely employed when high doses are administered.
- It is used for postoperative pain relief in the loading dose of 50 to 150mcg and a maintainance infusion of 0.5 to 1.5mcg/kg/hour.
- Used for sedation and analgesia in the dose of 1-4mcg/kg IV.
- As a component of neuroleptanalgesia with Droperidol (Innovar).

Side effects:

- Respiratory depression can occur post-operatively, related to the appearance of secondary peak in the plasma Fentanyl concentration due to elution from muscle.

- Other side effects include nausea, vomiting, pruritus, urinary retention and dependence

CLINICAL REVIEW

In a study, 84 parturients in active labour willing for analgesia were randomly divided into 7 groups to receive either 5, 10, 15, 20, 25, 35 and 45 µg intrathecal fentanyl as a part of combined spinal epidural technique. They concluded that intrathecal fentanyl produced rapid, profound labour analgesia with minimal side effects. The study data indicated that there was little benefit to increasing the dose of fentanyl, beyond 25 µg, when used as a sole agent for intrathecal labor analgesia.[42]

In a study 223 parturients in first stage of labour willing for extradural labour analgesia were divided randomly into 5 groups namely bupuvacaine 0.1%, Bupivacaine 0.07% and fentanyl 1µg/ml, bupivacaine 0.05% and fentanyl 2µg/ml, bupivacaine 0.05% and fentanyl 3µg/ml and bupivacaine 0.04% and fentanyl 4 µg/ml. They concluded that addition of extradural fentanyl to extradural bupivacaine in labour produced a dose dependent reduction in requirement for bupivacaine.[43]

In a study 197 parturients in active phase of labour willing for analgesia were randomly assigned to 2 groups of 99 each. Group 1 received 2.5 mg bupivacine + 25 µg fentanyl (intrathecally and epidural top up of bupivacaine) group II received 25mg bupivacaine cpidurally followed by top up. They concluded that combination of spinal epidural technique was faster in onset with less motor blockade and with feeling of greater self control[44].

In a prospective randomized double blind study for combined spinal epidural analgesia, 50 laboring parturients were divided into 2 groups. Group I

received intrathecal 1.25 mg bupivacaine and fentanyl 25 µg, group II received 2.5 mg bupivacaine + 25 µg fentanyl. They concluded that Bupivacaine 2.5mg when added to fentanyl 25 µg for combined spinal epidural analgesia in 1st stage of labour produced less motor block, sensory block and less hypotension.[9]

In a study, 67 full term parturients requesting extradural analgesia received either bupivacaine 0.25% group A or 0.125% group B or 0.0625% group C. They concluded that of the three concentrations based in labour analgesia, 0.125% was the most suitable concentration of plain bupivacaine to initiate extradural analgesia in labour and that 0.25% bupivacaine increased the incident of motor block, whereas for 0.0625% plain bupivacaine the probability to achieve adequate analgesia was unacceptably low.[45]

In a study, 90 parturients in active labour who requested regional anaesthesia were randomized to receive an intrathecal injection of either fentanyl 25µg; bupivacaine 1.25 mg with fentanyl 25 µg or bupivacaine 2.5 mg with fentanyl 25µg as part of a combined spinal epidural technique. They concluded that duration of analgesia was longer in the group receiving bupivacaine 2.5 mg and fentanyl 25 µg than group receiving plain fentanyl and onset of analgesia was faster in both groups receiving bupivacaine compared with plain fentanyl.[46]

In a study, 40 consenting nulliparous women received low dose combined spinal epidural analgesia for labor intrathecal injection of 2.5mg bupivacaine with 25µg Fentanyl was given followed by epidural top up, after regression of intrathecal block, subsequent analgesia was provided by intermittent epidural boluses of 10-15ml of low dose mixture consisting of

0.1% bupivacaine and 2µg/ml fentanyl. They concluded that small amount of of both drugs were present in mother and the neonate, such quantities were of little clinical significance in contrast to much longer acting neonatal effects of pethedine given to mother intramuscular during labour, use of fentanyl for ambulatory CSE in doses used in the study appeared to be safe for newborn.[47]

In a study, 50 healthy term parturients receive either CSE analgesia or lumbar epidural analgesia in labour epidural group receive bupivaciane 0.0625% fentanyl 0.0002% with 0.05ml in 10ml local anaesthesia CSE group received intrathecal 25µg Fentanyl and 2.5 mg bupivacaine the study concluded that the first sign of analgesia was not different between 2 groups, the onset of complete analgesia was more rapid with CSE technique.[48]

Comparative Obstetric Mobile Epidural Trial Study (COMETS) confirmed part of Nageotte's study that low dose techniques influence the mode of delivery in both CSE and low dose infusion groups there was an increased percentage of spontaneous vaginal deliveries compared to a traditional technique. The spontaneious vaginal delivery rate in COMET study were respectively CSE 42.7%, LDI 42.9% , traditional 35.1%.[11]

In a study, 40 nulliparous patients were divided into two groups. One group receiving intrathecal bupivacaine 1.25 mg with either Fentanyl 25µg or sufentanil 5µg as the intrathecal component in CSE for labour analgesia, study concluded that there was no significant difference in duration of analgesia. (Group F → mean 109±SD 49 min and group S – mean 118±54 min). Fentanyl group had more rapid onset of analgesia and higher cephalad block. (median TUVs T7 in first 30min after the block. No difference in side effects was

detected, Fentanyl 25µg was a good alternative to sufentanil 5 µg when added to bupivacaine 1.25 mg for early labour analgesia. [49]

On a study, 95 nulliparous term partiuents where divided into 3 groups and received 0.25% spinal ropivacaine, levobupivacaine or bupivacaine aim was to compare analgesic efficacies of intrathecal ropivacaine, levobupivacaine and bupivacaine for labour analgesia and determine analgesic potency ratios for these three drugs, they concluded that potency hierarchy of spinal bupivacaine > levobupivacaine > ropivacaine.[50]

In a study, 60 parturents where divided into two groups one group received spinal injection of 12.5µg of fentanyl with 2mg of bupivacaine other along with it received an additional 125 µg of morphine. Median duration of analgesia was similar between groups 89 min then 84 min only 20% of MBF group experienced prolonged analgesia. Intrathecal injections of this small dose of bupivacaine/ fentanyl produced rapid onset of labour analgesia, addition of small dose of morphine did not significantly prolong analgesia but improved, subsequent pain relief.[51]

A retrospective review of FHR tracing from 199 labouring parturients, there was no statistically significant difference in incidence of FHR abnormalities when an intrathecal technique using fentanyl was compared with a conventional epidural technique for labour analgesia, low incidence (6% - 12%) of FHR. Changes was found in both groups none of FHR changes identified resulted in need for cesarean delivery, no difference between groups in ultimate mode of delivery. [49]

In a study 65 labouring parturients were randomly assigned to receive intrathecal F (fentanyl 25 µg), B (bupivacaine 2.5mg) or fentanyl and bupivaciane (F 25 µg and B 2.5 mg) as a part of CSE technique, visual analog scores, sensorylevel, motor strength and prurities were recorded before injection and at interval. Median duration of analgesia in F, B, F+B groups were 62.5, 55.0, 94.5 min respectively. The combination of F+B lead to decreased frequency of pruritis (36.4% Vs 95%). They concluded when intrathecal fentanyl 25 µg is administered with, bupivacaine 2.5mg. The combination also results in rapid onset and prolonged duration of labour analgesia compared with either drug alone. [52]

In a study, 40 women in labour requesting regional analgesia were divided into 2 group one received intrathecal 2.5 mg bupivacaine + 25 µg Fentanyl other received 2.5 mg Ropivacaine + 25 µg Fentanyl, they found similar duration of analgesia in both the group (86% BF, 85% RF group). 40% (8/20) percent of women receiving bupivacaine developed detectable motor block. Compared only with 5% (1/20) in ropivacaine group. Adverse effects did not differ between groups. They concluded that combination of 25 µg Fentanyl as a part of CSE techniques, provides rapid and safe for labour as both are equally effective and ropivaciane and fentnyl group had significantly less motor block. [53]

In a study, 50 parturitions were divided into 2 groups one receiving epidural of 10ml containing bupivacaine 15mg and Fentanyl 20 µg, Group B received CSE intrathecal 1ml bupivacaine 1.25 mg and Fentanyl 20 µg. Onset of analgesia was within 5 min in group B and group A, 5-15 min there was no

significant difference in duration of labour, analgesia requirement, mode of delivery side effects, or condition of neonatal outcome. They concluded that the use of epidural technique in early active labour, reserving CSE labour analgesia for later stages of labour.[10]

In a study, 50 healthy term parturient were randomized, in a prospective, double blind fashion to receive either CSE analgesia or lumbar epidural analgesia in labour, epidural group received bupivacaine 0.0625% - Fentanyl 0.0002% and CSE group received intrathecal 25 µg fentanyl and 2.5mg bupivacaine. Additional analgesia was provided upon maternal request. Although the first sign of analgesia was not different between the two groups, onset of complete analgesia was more rapid with, CSE technique at 5 min.[48]

In a study, 90 full term parturients where divided into 3 groups to receive a single intrathecal injection of either Fentanyl 25 µg, Bupivacanine 1.25mg or fentanyl 25 µg and bupivacaine 2.5 mg as a part of CSE technique. Duration of analgesia was longer in the group receiving bupivacaine 2.5mg and fentanyl 25 µg than the group receiving plain fentanyl (108 Vs 92 min). Onset of analgesia was faster in both groups receiving bupivacaine compared with plain fentanyl. They concluded addition of 2.5 mg isobaric bupivacaine to 25 µg fentanyl for intrathecal labour analgesia modestly increased duration and speed of onset of analgesia compared with plain intrathecal fentanyl.[54]

Methodology

METHODOLOGY

Study pattern:

This clinical study was conducted in Department of Anesthesiology in association with Department of Obstetrics and Gynecology at Chigateri General Hospital and Women Children Hospital and Bapuji Hospital attached to J.J.M. Medical College, Davangere from October 2008 to August 2010. Clearance was obtained from hospital ethics committee for the study, written informed consent was obtained from all the patients.

The study of 60 patients in labour were included

Patients selection:

Inclusion criteria:

a) Healthy primigravida and gravida 2 patients at term.

b) ASA I and ASA II.

c) Maternal request for epidural analgesia.

d) Age group 18-35 years.

e) Women in active labour with cervical dilatation in primi about 4-5cm and gravid 2 with cervical dilatation of 3-4 cm.

Exclusion criteria:

a) Patients unwilling for procedure.

b) Parturient with gravid 3 or more.

c) Parturients with multiple pregnancies.

d) Pregnancy induced hypertension.

e) Severe anemia.

f) Cephalopelvic disproportion.

g) Previous LSCS

h) History of ante partum hemorrhage.

i) History of allergy to local anaesthetic.

j) History of CVS/RS disease.

k) History of bleeding disorders.

l) Diabetes mellitus.

m) History of psychiatric/neurologic disease.

Equipments:

1. IV canula and IV fluids
2. Sterile combined spinal epidural tray.
3. Combined spinal epidural kit
    a. Epidural 18 gauge Tuohy's needle
    b. Spinal 27 gauge Whitacre 15mm extendable pencil point needle.
    c. Epidural catheter 20G polyamide closed end.
    d. Loss of resistance syringe .
4. Drugs
    a. Study agent
        i. Inj. Bupivacaine 0.5% heavy (spinal) amp
        ii. Inj. Bupivacaine 0.5% vials
        iii. Inj. Fentanyl 100 µg amp.

b. Emergency drugs

i. Inj. Adrenaline

ii. Inj. Atropine

iii. Inj. Ephedrine

iv. Inj. Mephenteramine

v. Inj. Dopamine

vi. Inj. Dobutamine

vii. Inj. Thiopentone,

viii. Inj. Scoline.

c. Others

i. Inj. Metoclopramide

ii. Inj. Ranitidine

iii. Inj. Oxytocin

iv. Inj. Methergine

v. Inj. Carboprost

5. Boyle's machine

6. Resuscitation equipment, oxygen cylinder, AMBU bag, laryngoscope, endotracheal tubes of different size, suction apparatus.

   Emergency drug tray.

7. Monitoring equipment ECG, NIBP, Pulse oximeter.

Methodology:

60 parturients with ASA I and ASA II in established labor with cervical dilatation less than 5cm was randomly selected informed written consent was taken from patients.

A detailed history, complete physical examination and routine investigations was done for all patients. IV line was secured with 18G canula. Patients were divided into 2 groups of 30 each.

Group I received intrathecal Inj. Bupivacaine 1.25 mg and Inj.Fentanyl 25 µg.

Group II received intrathecal Inj. Bupivacaine 2.5 mg and Inj. Fentanyl 25 µg for combined spinal epidural.

IV line was secured with 18 G cannula, patient was preloaded with 500ml of Hartmann's solution basal vital parameter like pulse rate, blood pressure, respiration, O_2 saturation were recorded.

The patient was positioned in a left lateral position with the help of an assistant. Under aseptic conditions the back was prepared with 5% providine iodine solution, spirit and area was draped.

L3-L4 interspace was identified, skin was infiltrated with 2ml of 1% xylocaine.

Procedure:

After infiltration of local anaesthetic by using needle through needle technique 18 guage Tuohy needle, epidural space was identified with loss of resistance to air

technique. Then a 15mm (25 G) long 'Whitacre' spinal needle was introduced through the epidural needle and the correct position of the tip in the intrathecal space was confirmed by observation of free flow of CSF.

Patients were allocated randomly to receive intrathecal injection of Bupivacaine 1.25 mg (0.5% Bupivacaine 0.25ml) with Fentanyl 25 µg (Group I n=30) or Bupivacaine 2.5 mg/0.5% Bupivacaine 0.5ml) with Fentanyl 25 µg (Group II, n=30) both made up to total volume of 2ml with saline.

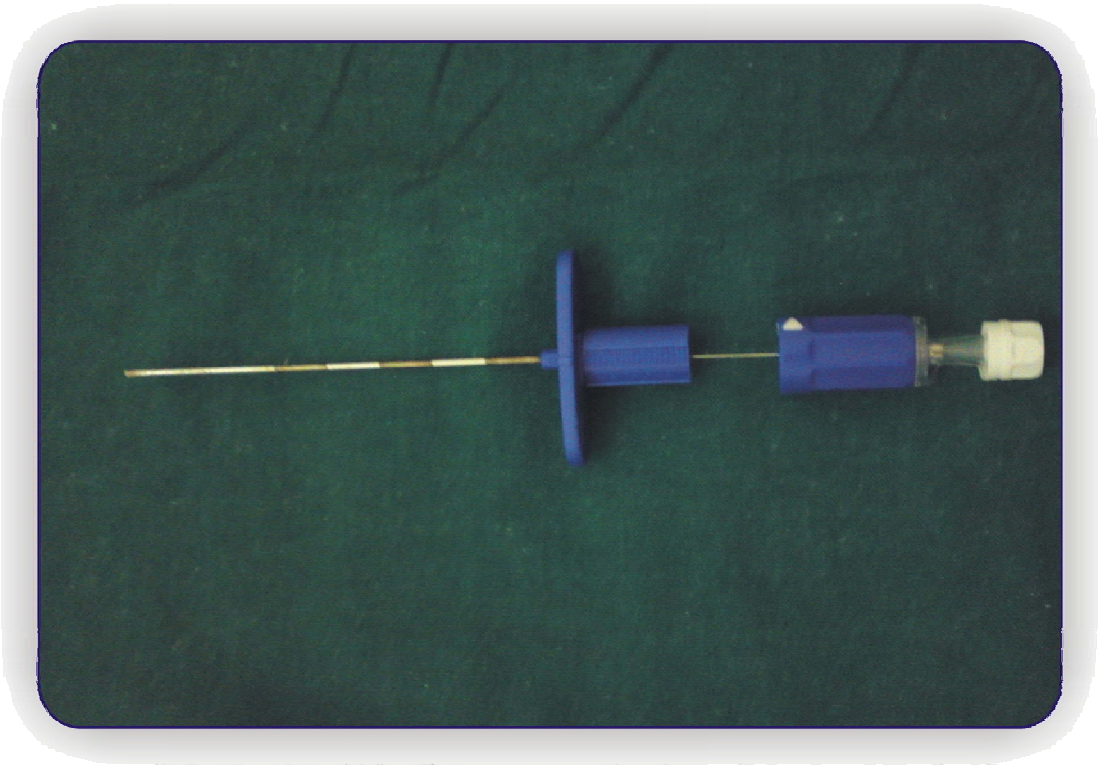
Injection of intrathecal drug was completed in 10 sec then 20G epidural catheter was threaded through the epidural needle into the epidural space in cephalad direction. The epidural needle was slowly pulled out without disturbing the catheter, about 3 to 5 cm of catheter was left in epidural space. The catheter was well secured with plaster .

Patients VAS pain score was recorded every 5, 10, 15, 30, 45, 60, 75, 90, 105, 120 min i.e., (every 5min for 15 min and then every 15 min for 2hrs) until the next request for analgesia.

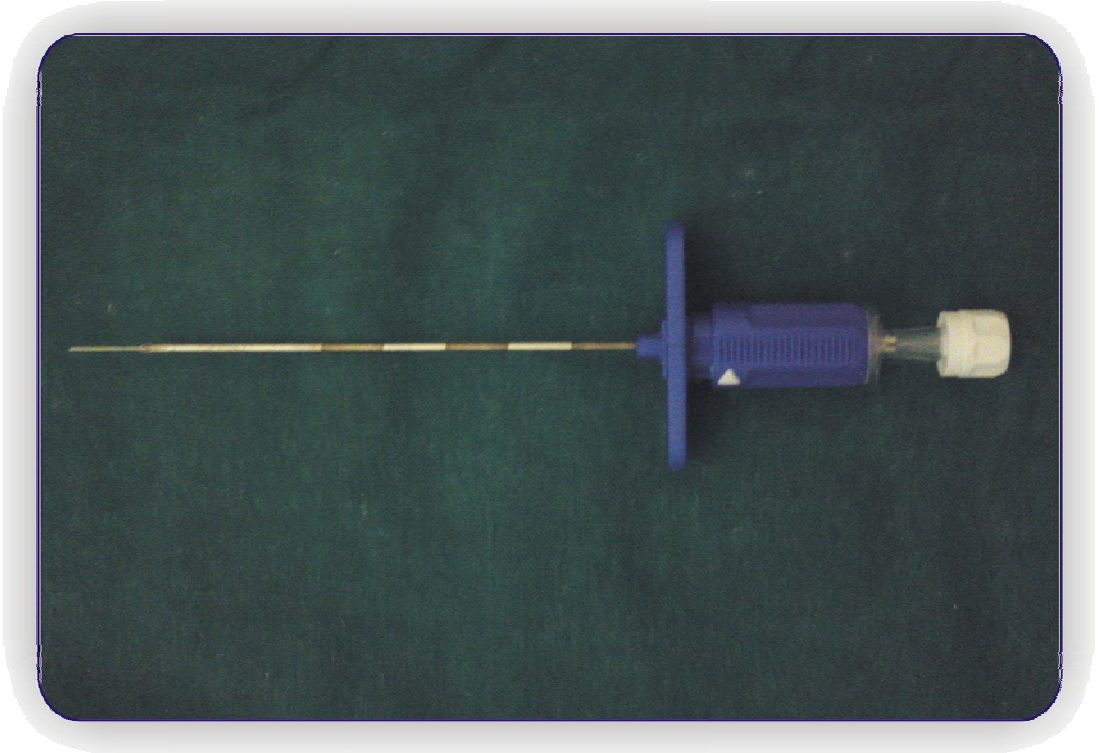
After positioning the patient in supine position, onset of analgesia and dermatomal level were checked by loss of sensation to pin prick, time of onset and degree of motor blockade was checked by Bromage classification.

VAS pain score for all patients at the next request for analgesia was recorded and study was terminated. Continuation of epidural analgesia was done with 0.125% bupivacaine + 2µg fentanyl in 10 ml.

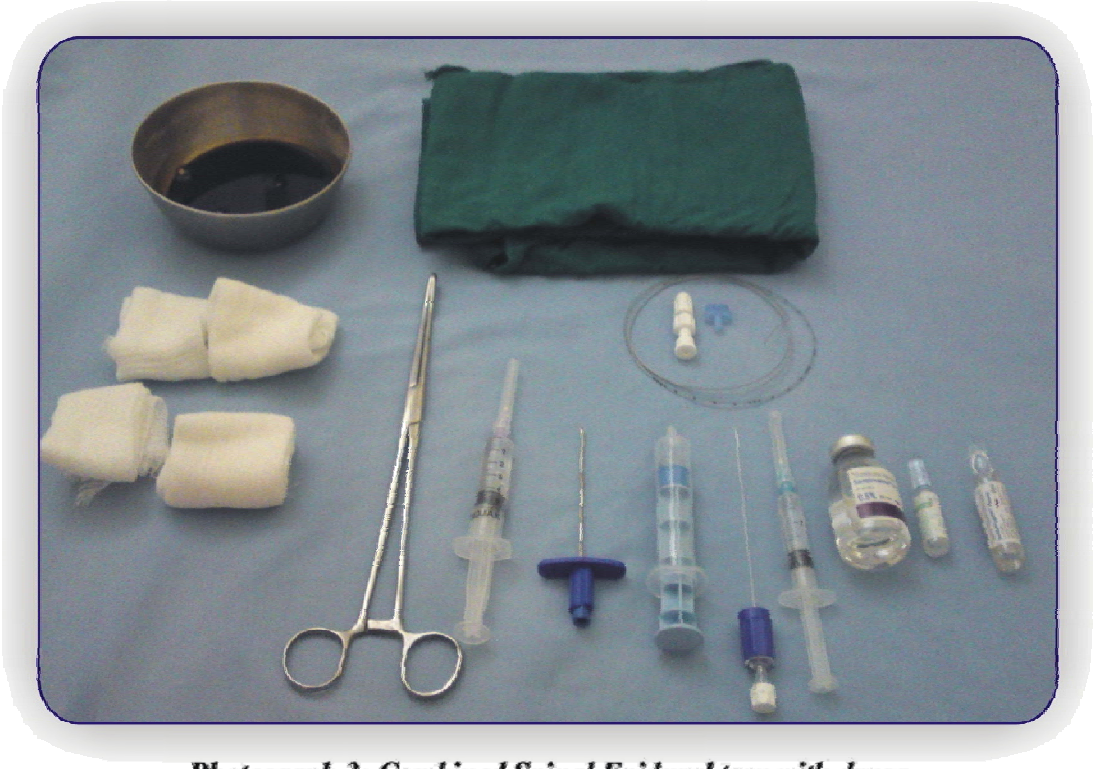
ILLUSTRATING THE LOCKING MECHANISM



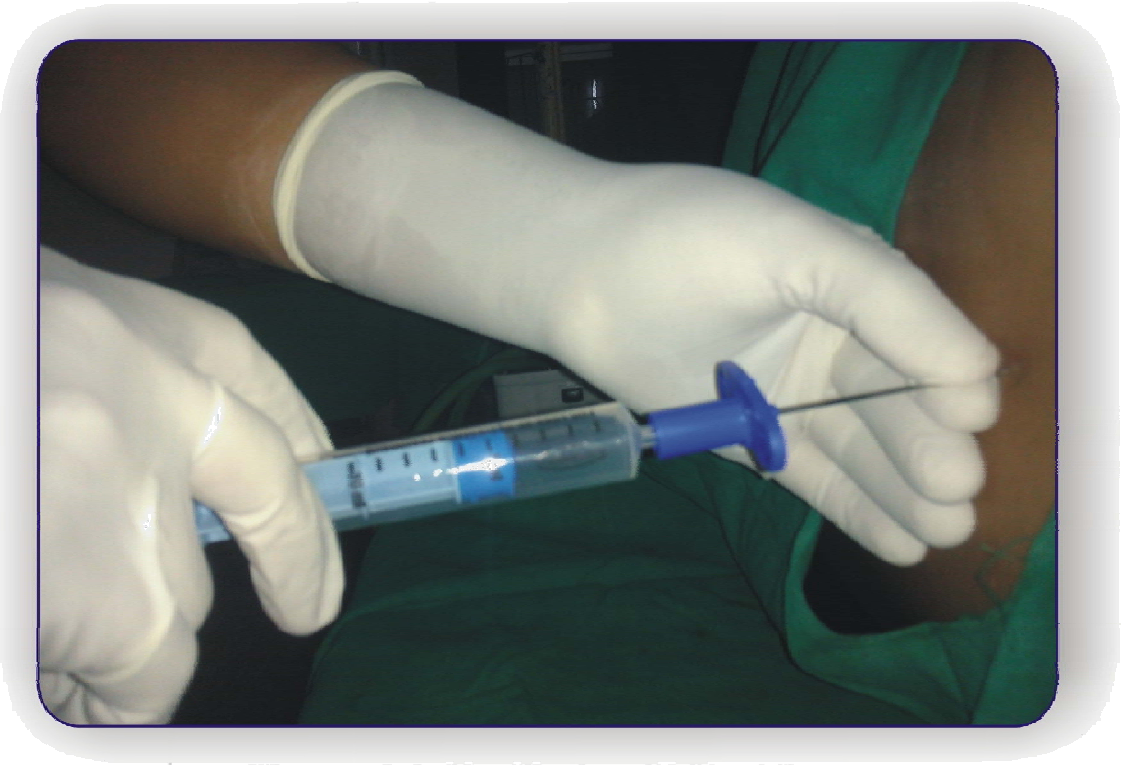
Photograph 1: The Spinal Needle is not extending beyond the tip of Tuoky Needle



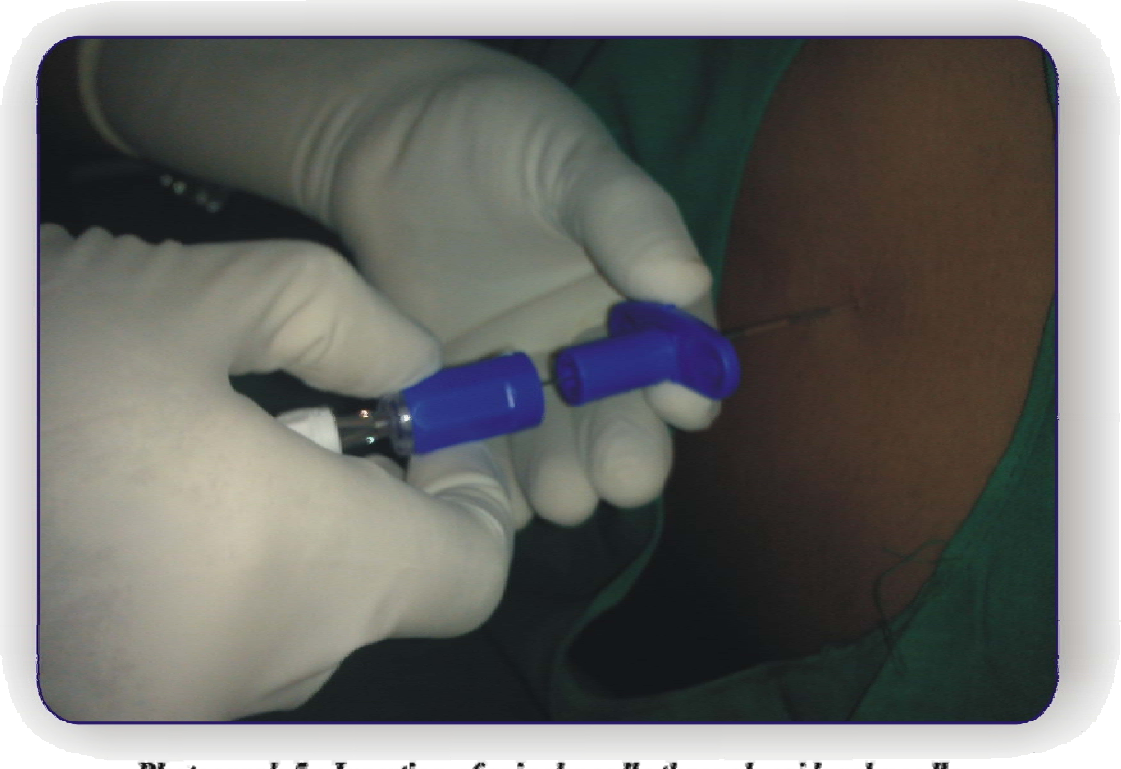
Photograph 2 : The Spinal Needle is maximally extended



Photograph 3: Combined Spinal Epidural tray with drugs

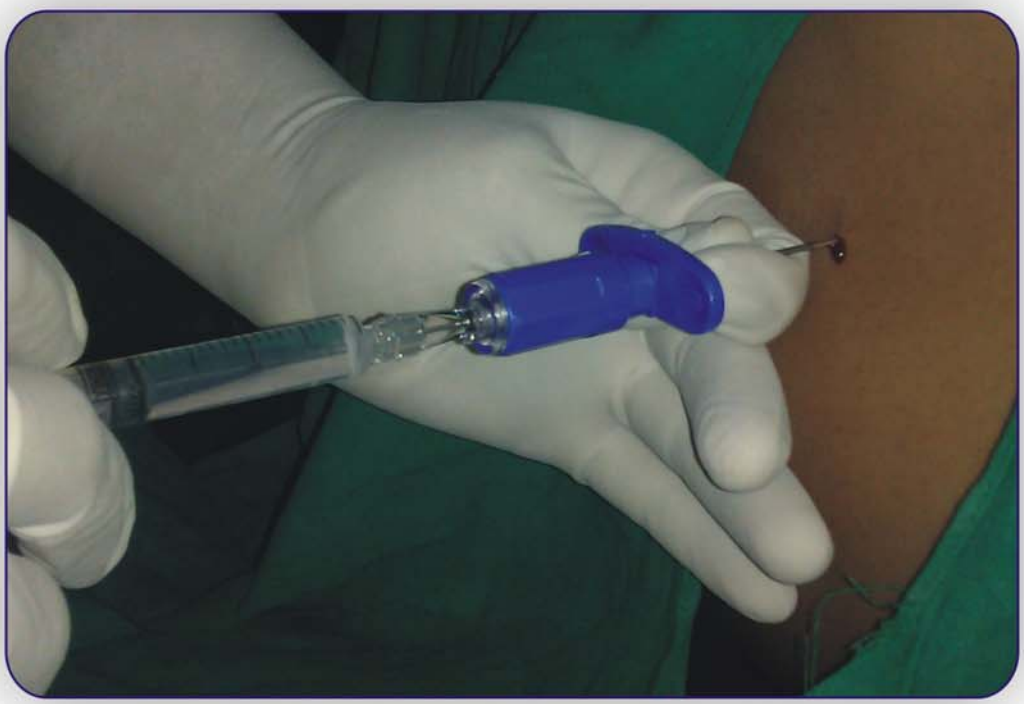


Photograph 4: Identification of Epidural Space

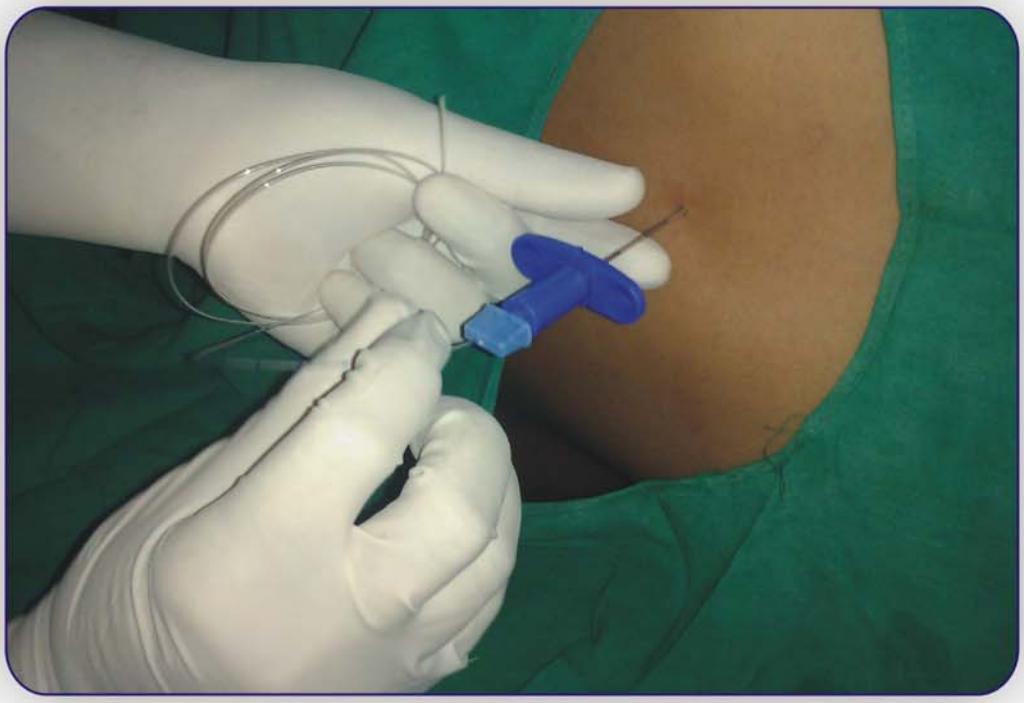


Photograph 5 : Insertion of spinal needle through epidural needle

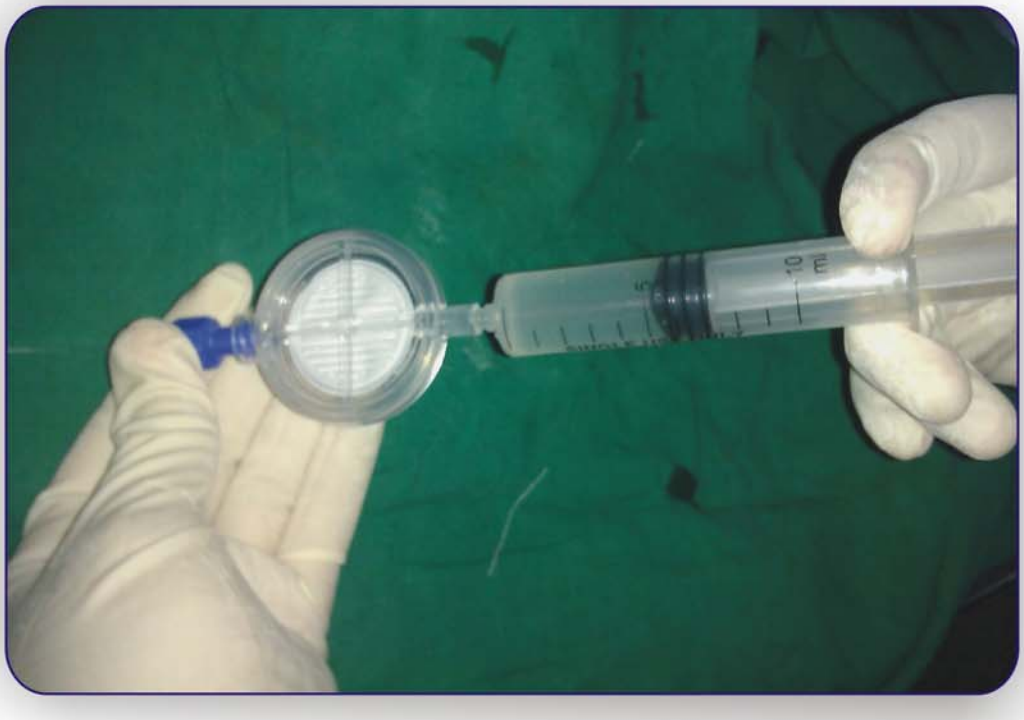
Photograph 6: CSF Tapping



Photograph 7 : Administration of Drug into Intrathecal space



Photograph 8: Insertion of Epidural Catheter



Photograph 9 : Administration of Epidural top-up drug

Monitoring – mother's vital parameters, progress of labour, efficacy of analgesia and fetal welfare were watched in coordination with attending obstetrician. Pulse, NIBP, SPO2, respiratory rate were recorded before and after the start of procedure and every 5 min for first 15 mins and then every 15 mins for 2hrs. If bradycardia occurred at any time (<60 bts/min) Inj. glycopyrolate 0.2mg was given. If hypotension occurred then it was treated appropriately with IV fluids and vasopressor. If pruritis occurred it was treated with pheniramine.

Parameters studied:

The following parameters are studied

1. **Assessment of sensory blockade** – Sensory blockade assessed by pin prick and time noted for block to reach different dermatomal level.

   - Onset of sensory block.
   - Maximum height reached and time required,
   - Duration of analgesia,
   - Quality of analgesia.

2. Assessment of motor block :

- Motor blockade was assessed by Bromage scale.
- Time required for complete recovery.

3. Untoward effects :

The patient were carefully monitored for any untoward effects like inadequate block, hypotension, bradycardia, respiratory distress, nausea, vomiting, restlessness, pruritis, shivering, anaphylactic reaction, fetal bradycardia.

4. Complications :

Following components were looked for and patients were watched for 12 hours for Cardio respiratory catastrophe, and complete spinal, dural puncture, PDPH, backache, transient neurological symptoms, vascular injury, infection and catheter related problems.

Terms and definitions :

Time of onset of analgesia, this was taken as time from deposition of drug to the feeling of tingling sensation in the legs.

Time of onset of paralysis (motor blockade). This was taken as time from onset of paresis to loss of power i.e., patient was not able to lift the legs (Modified Bromage Scale Onset of Motor Block).

Scale	Criteria	Degree of Block
0	full flexion of knees and feet and	No block 0%
1	Just able to flex knees possible, still full flexion of feet possible.	Partial block 33%,
2	unable to flex knees but flexion of feet possible	Almost complete 6%.
3	unable to move legs or feet	Complete 100%.

Duration to reach maximum dermatomal level :

This was taken as the time interval between the deposition of drug and loss of sensation at highest dermatomal level.

Statistical analysis :

In the present study, results are given as mean ± standard deviation and range values for continuous data. Students ‘t’ test was used to compare the two groups, categorical data are expressed as number and percentages and difference between the groups was compared by chi-square test. A p value of 0.05 or less was set for statistical significance.

Results

RESULTS

TABLE 1 : AGE DISTRIBUTION

	Mean+/- SD				
Parameter	Group I	Group II	Mean difference	P* Value	Sig
Age (Years)	23.43+/-2.87	22.63+/-2.94	0.8	0.29	NS

Both the group I and group II were similar with respect to age of the parturients. Mean age in group I was 23.43 and SD of 2.87. In group II mean age was 22.63 with SD of 2.94. p-value of 0.29 and was statistically insignificant.

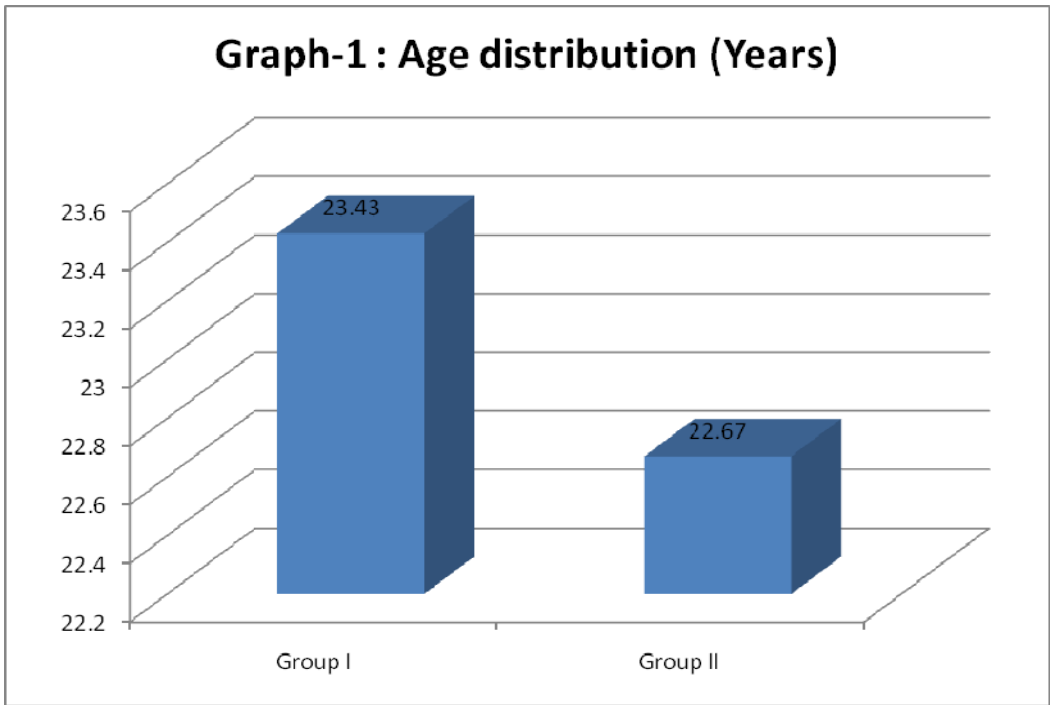


TABLE – 2 : DISTRIBUTION OF PARTURIENTS ACCORDING TO THEIR HEIGHT

Height (cm)	Group I	Group II
<150	4 (13.3%)	2 (6.7%)
150-160	22 (73.3%)	22 (73.3%)
>160	4 (13.3%)	6 (20%)
Total	**30**	**30**
Mean	154.2±4.1	154.9±4.4
Range	147-163	146-162

t=0.64, p=0.52 NS

Height of parturient studied ranged from 145 cm to 165 cm . Shortest height was 147cm and tallest being 163cm in group I . Shortest height 146 cm and tallest being 162 cm in group II. The mean height and standard deviation were 154.2 cm and 4.1 in group I and 154.9 cm and 4.4 in group II respectively. The p-value of 0.52 was statistically not significant.

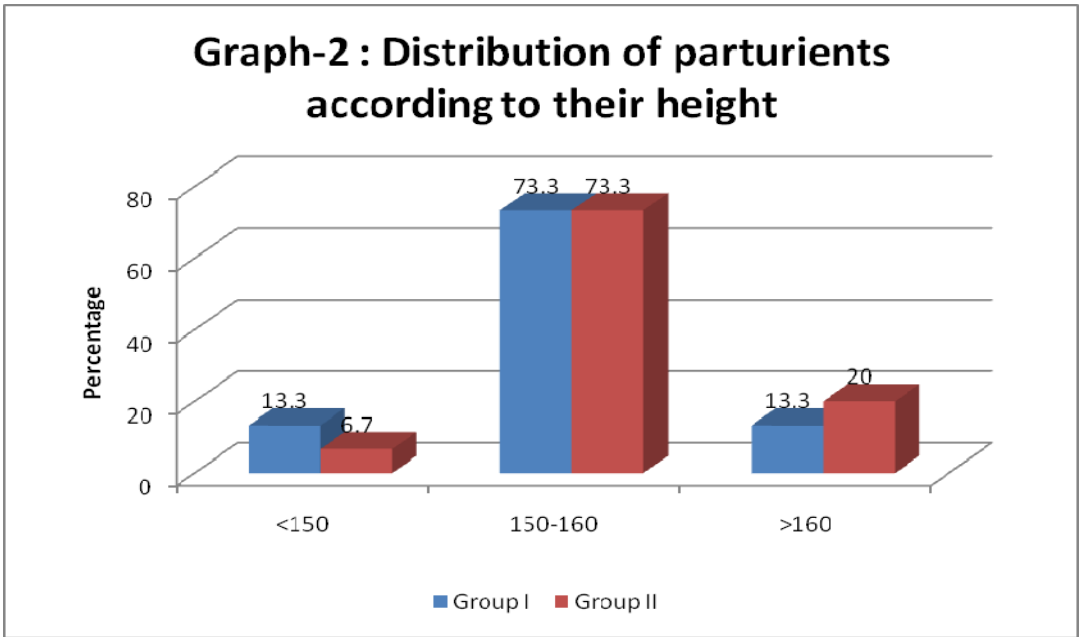


TABLE – 3 : DISTRIBUTION OF PARTURIENTS BASED ON THEIR WEIGHT

Weight (kg)	Group I	Group II
<50	2 (6.6%)	1 (3.3%)
50-60	14 (46.7%)	15 (50%)
>60	14 (46.7%)	14 (46.7%)
Mean ± SD	59.8 ± 6.5	58.7 ± 6.2

t=0.69, p-51 NS

Most of the parturients weighed between 50-60 kgs in both the groups.

In group I the mean weight was 59.8 kg and SD 6.5.

In group II mean weight was 58.7 kg and SD 6.2, p-value of 0.51 was not significant.

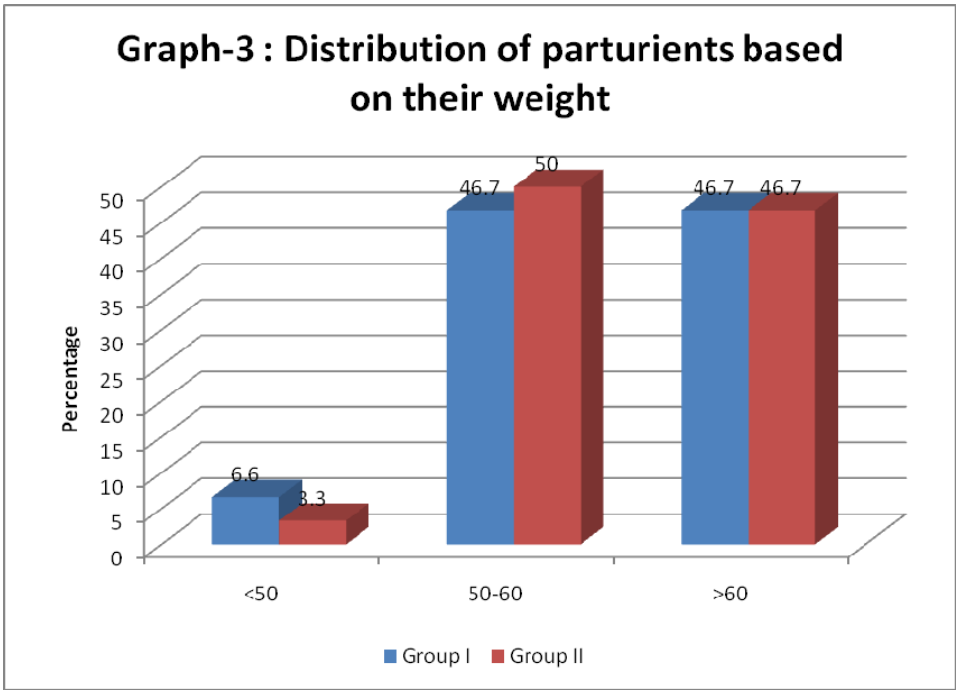


TABLE – 4 : DISTRIBUTION OF PARTURIENTS ACCORDING TO CERVICAL DILATATION

Cervical dilation	Group I	Group II
4	5(17%)	4(14%)
5	15(50%)	13(43%)
6	10(33%)	13(43%)

X^2 = 64 P=0.72 NS

50% of patient in group I and 43% of patient in group II had cervical dilatation of 5 cm 33% of patient in group I and 43% of patient in group II had cervical dilatation of 6cm. p-value of 0.72 not statistically significant.

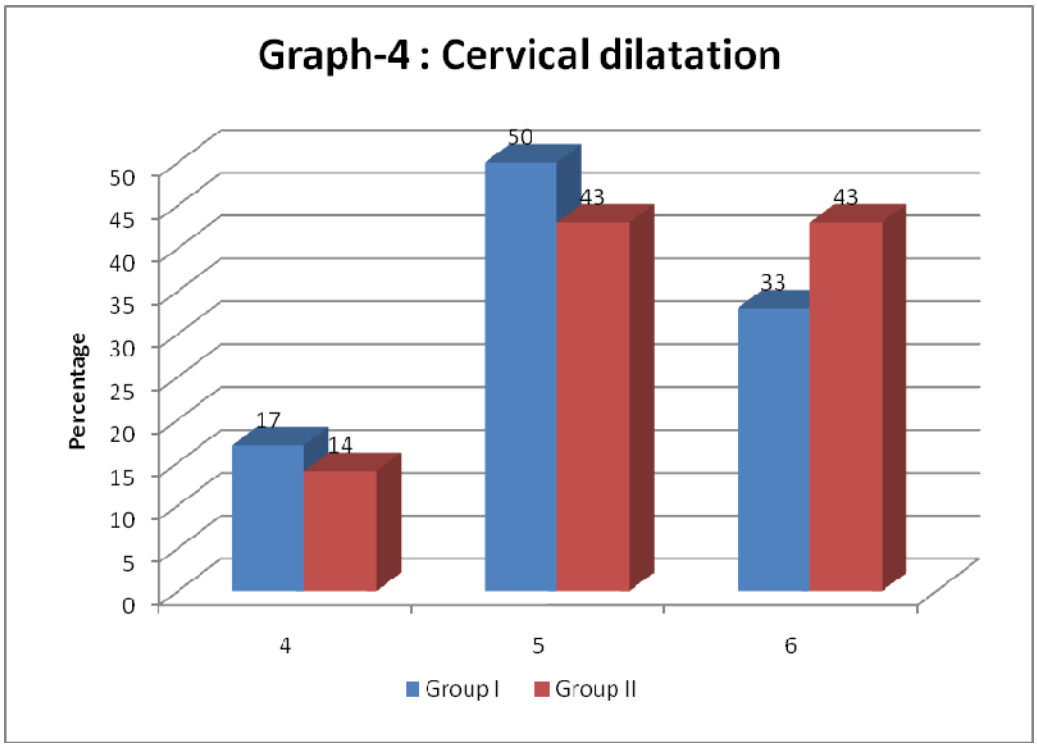


TABLE – 5 : TIME OF ONSET OF SENSORY ANALGESIA AFTER SPINAL COMPONENT OF CSE

Parameter	Mean+/- SD		Mean difference	P* Value	Sig
	Group I	Group II			
Sensory onset of action in secs	204.33+/- 53.06	87+/- 30.61	117	<0.001	HS

Table-5 shows the onset of sensory analgesia in seconds in number of patients of this study. In group I. Mean was 204.33 secs and SD of 53.06 and in group II mean was 87 sec. SD of 30.61 and mean difference between 2 groups 117 secs. p-value of <0.001 highly significant.

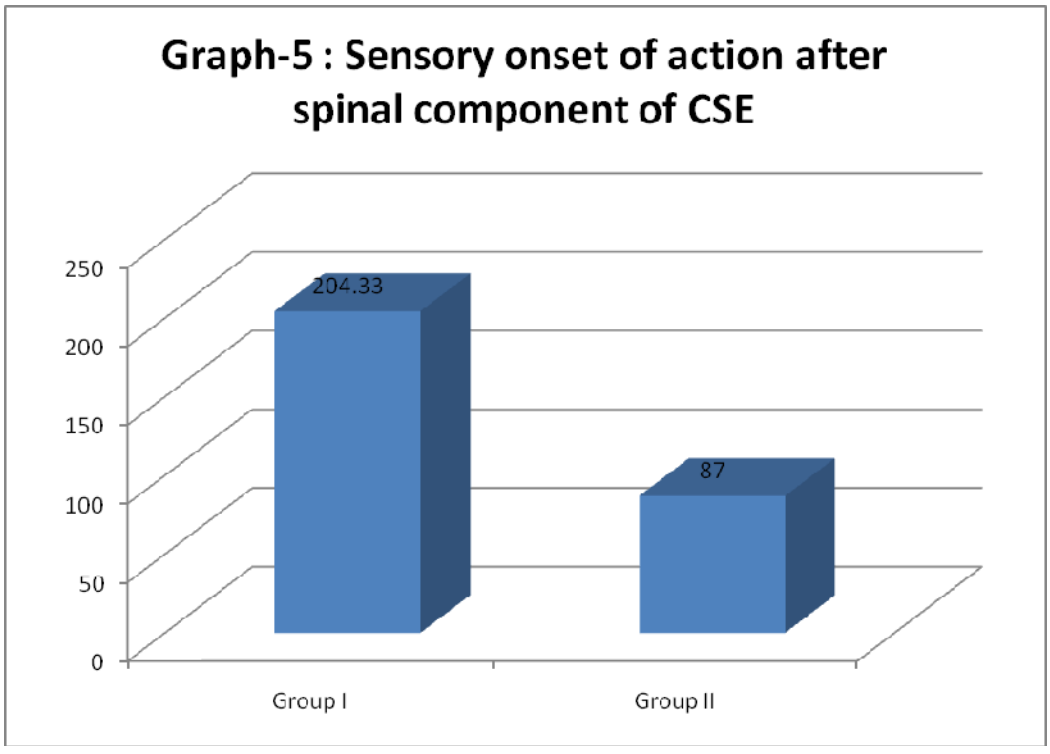


TABLE – 6 : MAXIMAL DERMATOMAL LEVEL OF SENSORY BLOCKADE AFTER SPINAL COMPONENT OF CSE

Dermatomal of level	Group I	Group II
T6	0	2(7%)
T7	0	11(37%)
T8	5(17%)	11(37%)
T9	13(43%)	4(14%
T10	10(33%)	2(7%)
T11	2(7%)	0

X^2 = 27.3 P<0.001 HS

Table-6 shows maximum dermatomal level of sensory blockade achieved after spinal component of CSE. Range being T8 to T11 in group I with average level of T9. Majority of patients 43% of cases achieved an average sensory blockade upto T9 segment. In group II , range being T6 to T10 with average level of T7-T8 , 37% of patients achieved sensory blockade of T8segment. P-value of <0.001 which was highly significant.

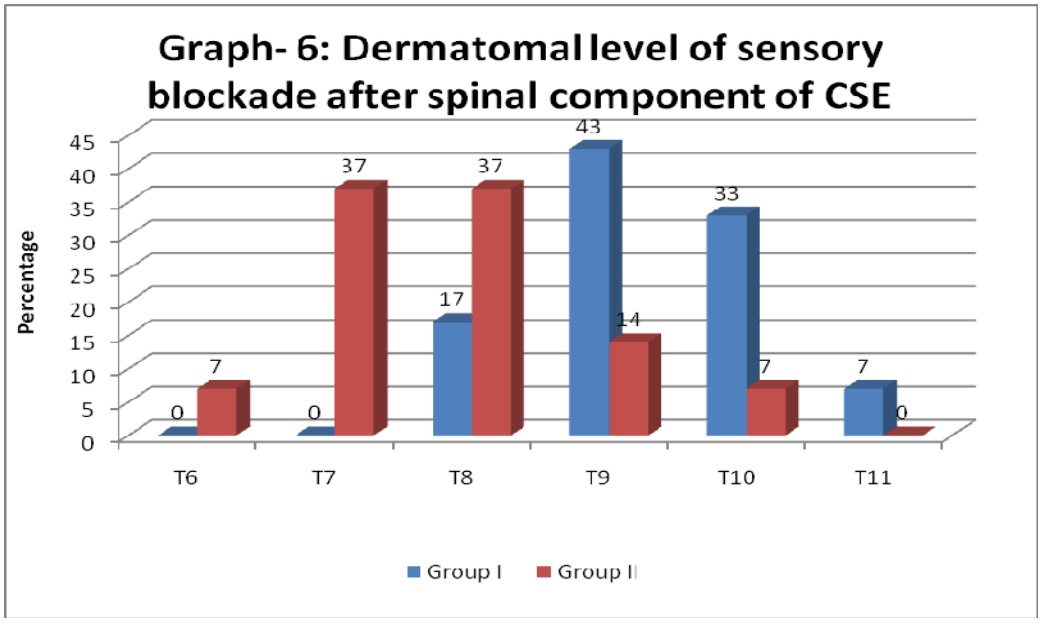


TABLE – 7 : GRADE OF MOTOR BLOCKADE AFTER SPINAL COMPONENT OF CSE

Motor onset of action	Group I	Group II
0	26(87%)	18(60%)
I	4(13%)	9(30%)
II	0	3(10%)

X^2 = 6.3 P=0.04 S

Table 7 shows grade of motor blockade by Bromage scale 87% of patients had grade 0 motor blockade and 60% of patients in group II had grade 0 motor blockade. Grade I motor blockade in 13% of patients of group I and 30% in group II. GradeII motor blockade was not there in group I and in 10% of cases in group II.

p-0.04 statistically significant.

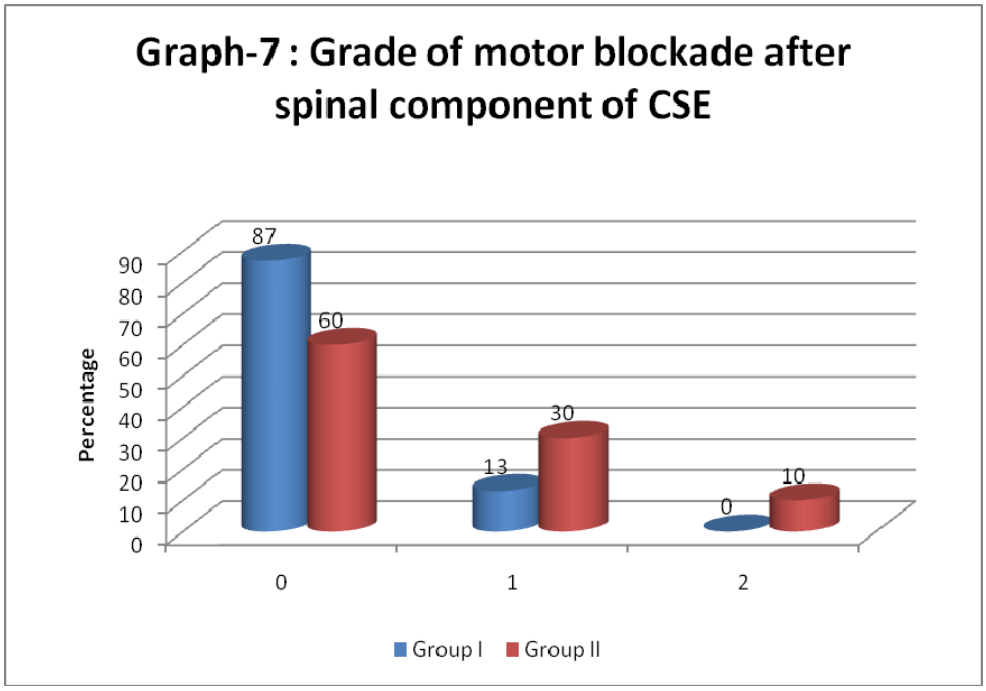


TABLE – 8 : CHANGES IN HEART RATE

Heart Rate	Mean+/- SD		Mean Difference	P* Value	Sig
	Group I	Group II			
0	97+/-11	95+/-8	2.0	0.43	NS
1	80+/-8	81+/-15	-0.8	0.80	NS
5	80+/-9	82+/-6	-2.2	0.29	NS
15	79+/-6	80+/-7	-0.3	0.87	NS
30	79+/-7	80+/-8	-0.4	0.83	NS
45	79+/-7	77+/-8	1.8	0.37	NS
60	78+/-7	75+/-9	2.3	0.26	NS
90	82+/-5	75+/-7	6.6	<0.001	HS
180	81+/-6	75+/-6	5.5	0.001	HS

* Student's unpaired t test

Table 8 shows mean pulse rate ± SD per min. It was statistically significant at end of 90 min and 180 min mean difference being 6.6 and p of <0.001 which was significant and mean difference of 5.5 and p<0.001 which was statistically significant.

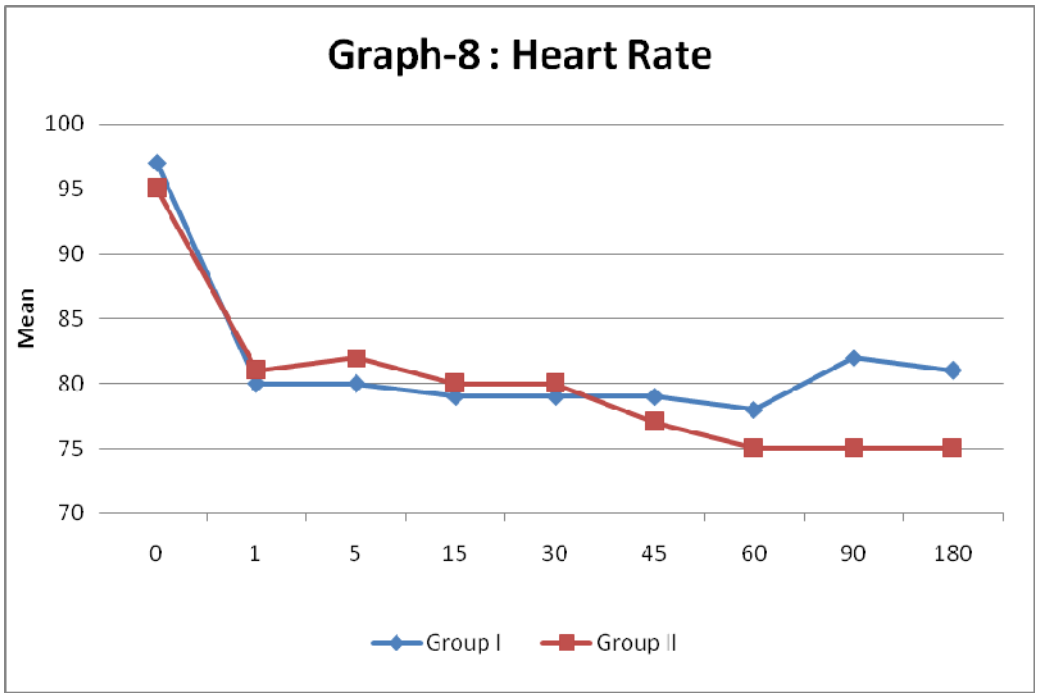


TABLE – 9 : CHANGES IN SYSTOLIC BLOOD PRESSURE

Systolic BP	Mean+/- SD		Mean Difference	P* Value	Sig
	Group I	Group II			
0	120+/-7	121+/-8	-0.4	0.84	NS
1	111+/-10	112+/-9	-1.5	0.52	NS
5	110+/-10	112+/-10	-1.8	0.48	NS
15	107+/-22	106+/-13	0.5	0.92	NS
30	113+/-9	106+/-13	7.0	0.02	S
45	117+/-13	110+/-13	6.3	0.07	NS
60	115+/-9	115+/-8	0.4	0.86	NS
90	117+/-8	115+/-7	2.1	0.29	NS
180	119+/-5	110+/-13	8.3	0.002	S

* Student's unpaired t test

Table – 9 shows the mean systolic pressure changes. Systolic pressure showed a decline to 106±13 mm of Hg in group II at 30 min which was statistically significant.

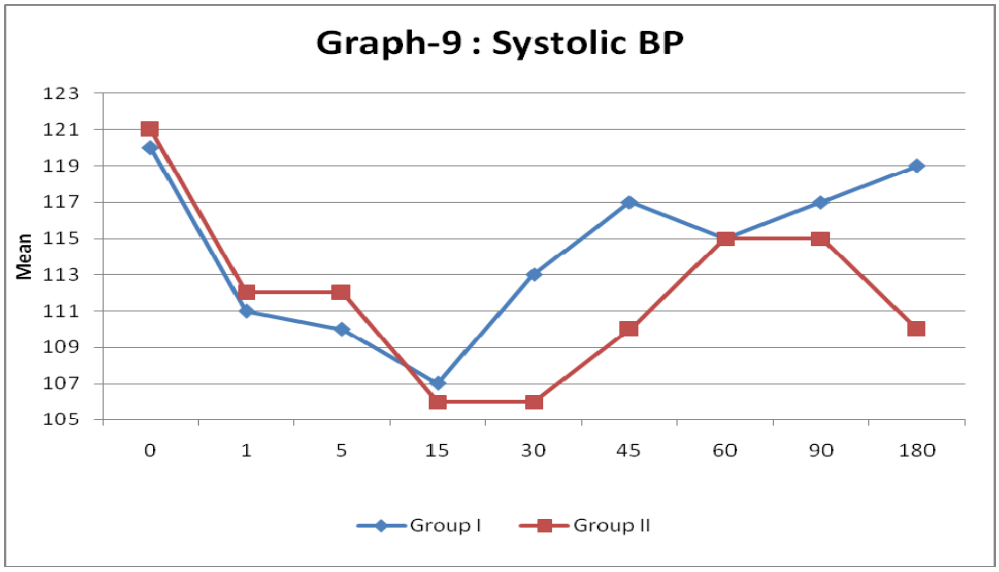


TABLE – 10 : CHANGES IN DIASTOLIC BLOOD PRESSURE

Diastolic BP	Mean+/- SD		Mean Difference	P* Value	Sig
	Group I	Group II			
0	78+/-7	81+/-5	-2.6	0.10	NS
1	75+/-10	78+/-8	-2.9	0.21	NS
5	74+/-10	74+/-9	-0.1	0.98	NS
15	75+/-11	72+/-11	2.6	0.36	NS
30	77+/-9	74+/-11	3.0	0.25	NS
45	75+/-10	75+/-10	-0.6	0.81	NS
60	78+/-10	77+/-8	1.4	0.56	NS
90	74+/-10	74+/-10	0.0	-	-
180	76+/-9	76+/-9	0.0	-	-

* Student's unpaired t test

Table 10 shows mean diastolic pressure changes. There was no significant fall in the diastolic blood pressure in our study.

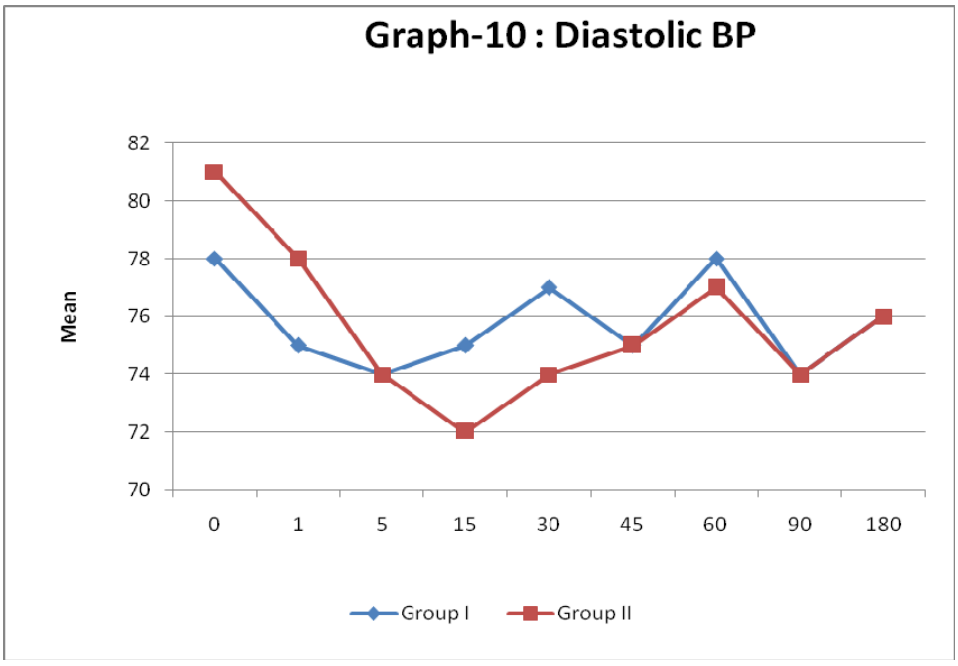


TABLE – 11 : DURATION OF TWO SEGMENT REGRESSION

Parameter	Mean+/- SD		Mean difference	P* Value	Sig
	Group I	Group II			
Time of 2 segment regression in mins	82.67+/- 16.17	104.33+/- 19.37	-21.6	<0.001	HS

Table 11 shows the time taken for two segment regression of spinal component of CSE. Mean time of 82.67 min and standard deviation of 16.17 in group I and mean time of 104.33mins and SD of 19.37 with mean difference of 21.6 and p-value <0.001 which was highly significant.

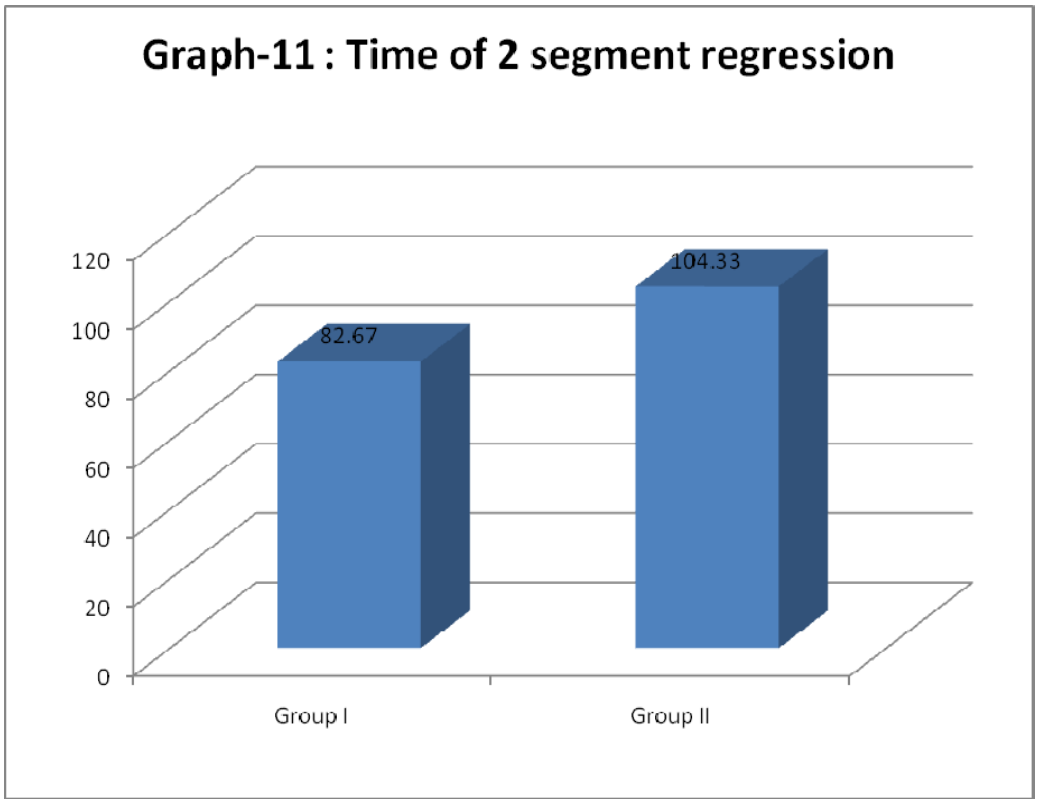


TABLE – 12 : MODE OF DELIVERY (OUTCOME)

Outcome	Group I	Group II
Caeserian	1(3%)	5(17%)
Forceps	4(13%)	6(20%)
Normal	25(84%)	19(63%)

$X^2 = 3.8$ P=0.14 NS

Table-12 shows mode of delivery 84% of case in group I went for normal delivery with 3% for caesarean section and 13% for forceps. In group II 63% went for normal delivery and 17% of caesarean section and 20% for forceps p-value <0.14 which is not significant.

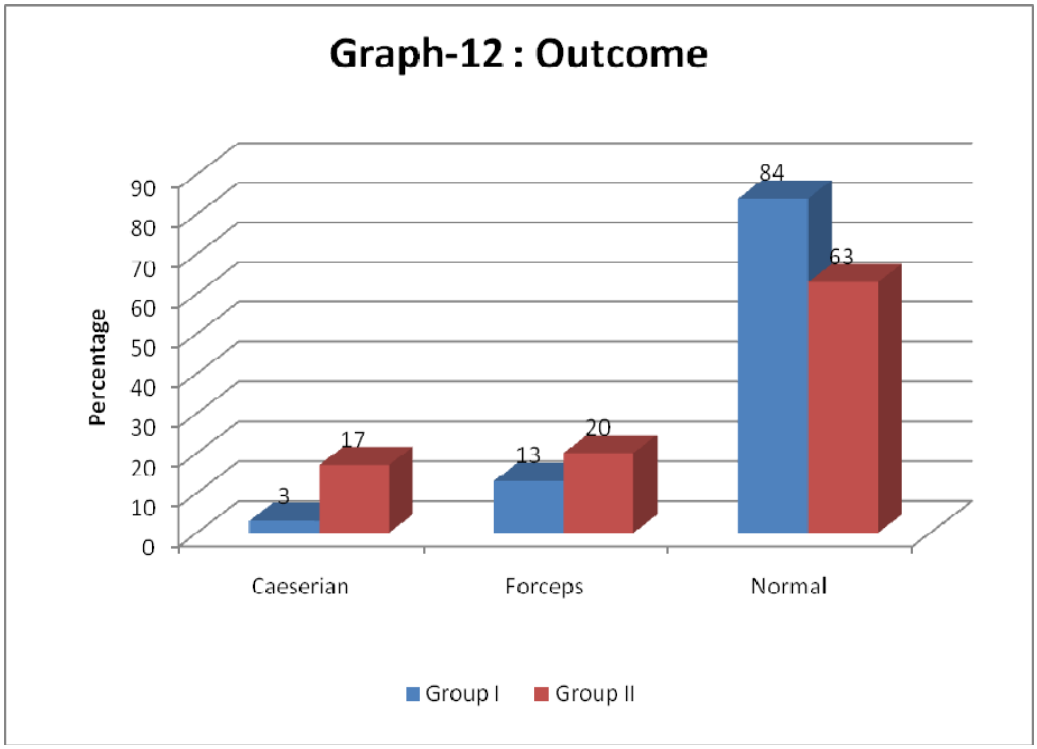


TABLE – 13 : EPIDURAL TOP UP NEEDED OR NOT AFTER SPINAL

Epidural needed	Group I	Group II
Yes	29(97%)	23(77%)
No	1(3%)	7(23%)

X^2 = 5.8 P=0.01 S

Table 13 shows number of patients who needed epidural top up after spinal component were of 97% of patients in group I need epidural top and 77% of patients in group II needed top up . 23% of patients in Group II did not need epidural top up with p-value <0.01 which was significant.

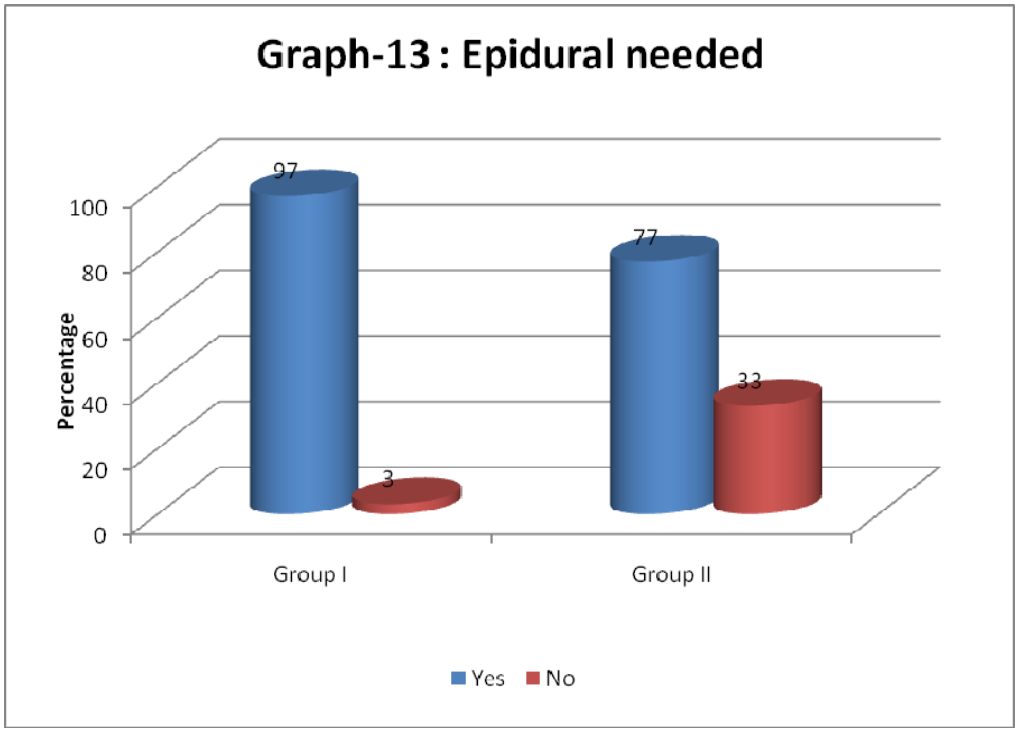


TABLE – 14 : VAS SCORE

VAS after spinal	Group I	Group II
1- 2	20(67%)	28(93%)
3- 4	10(33%)	2(7%)

X^2 = 6.7 P=0.01 S

Table 14 shows pain relief during labour by patient feed back method using visual analogue scale.

In group I 67% percent of patient showed excellent analgesia compared to 93% in group II.

33% showed good analgesia in group I compared to 7% in group II. P-value <0.01 which was statistically significant.

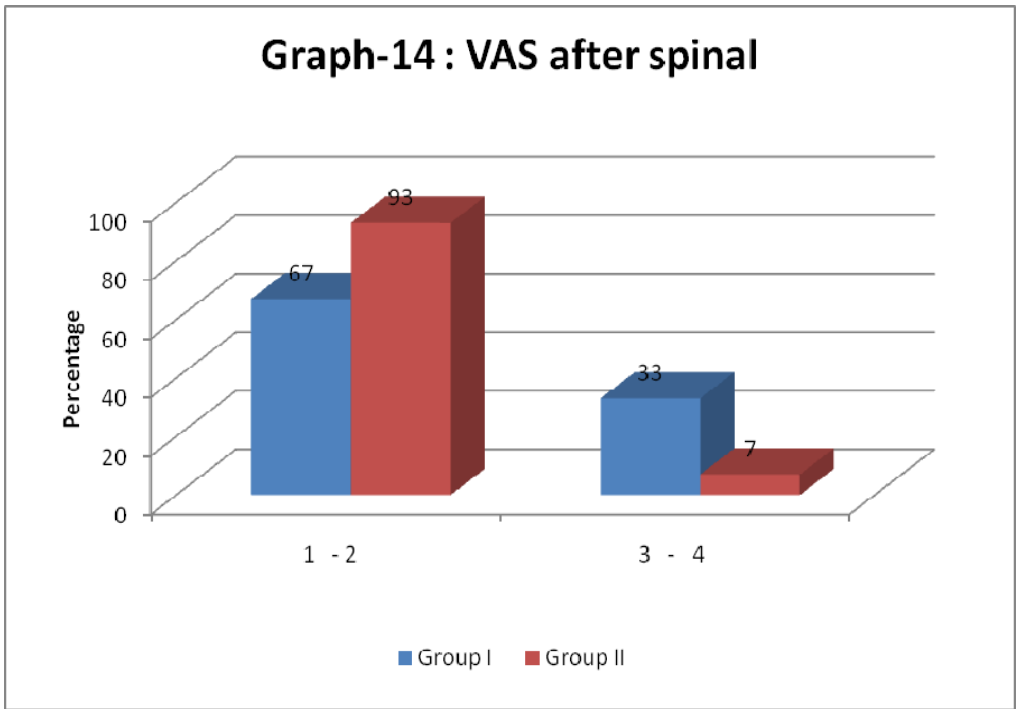
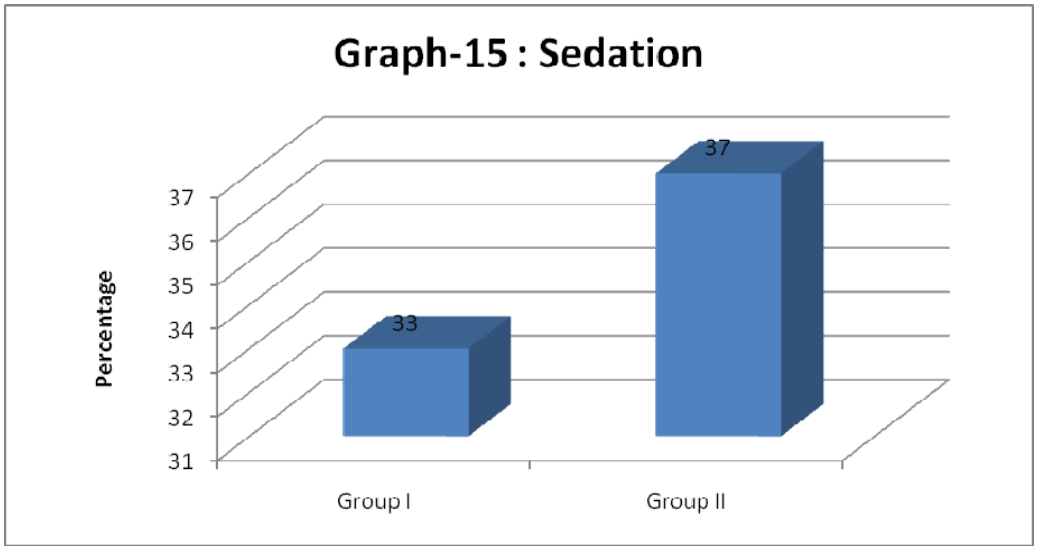


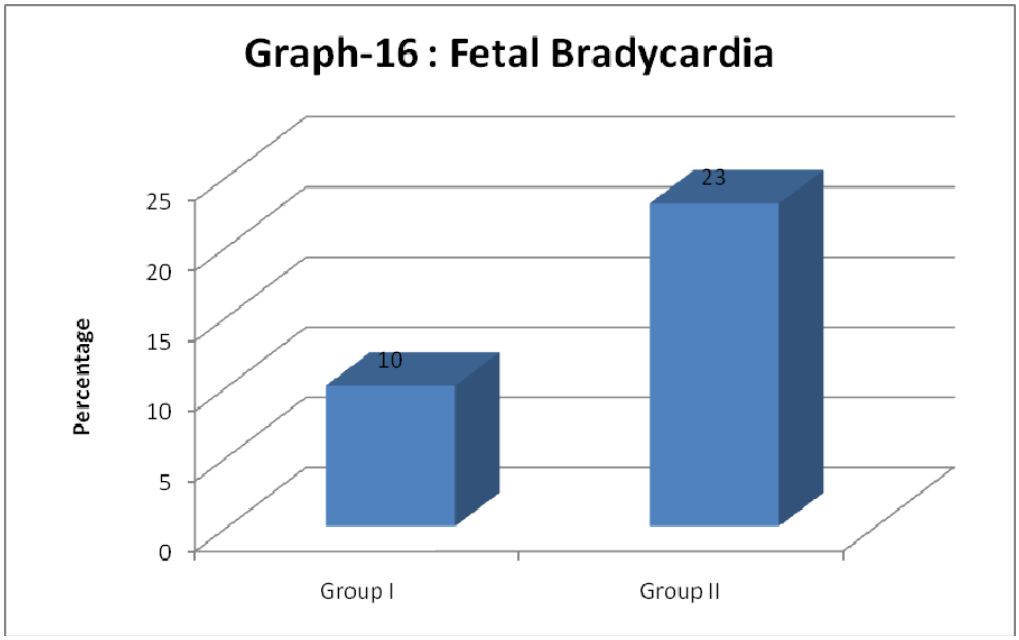
TABLE – 15 : COMPLICATIONS

Parameter	Present cases		P* Value	Sig
	Group I	Group II		
Sedation	10(33%)	11(37%)	0.78	NS
Fetal Bradycardia	3(10%)	7(23%)	0.16	NS
Nausia	8(27%)	8(27%)	-	-
Vomiting	5(17%)	5(17%)	-	-
Pruritis	10(33%)	11(37%)	0.78	NS
Hypotension	3(10%)	13(43%)	0.004	S

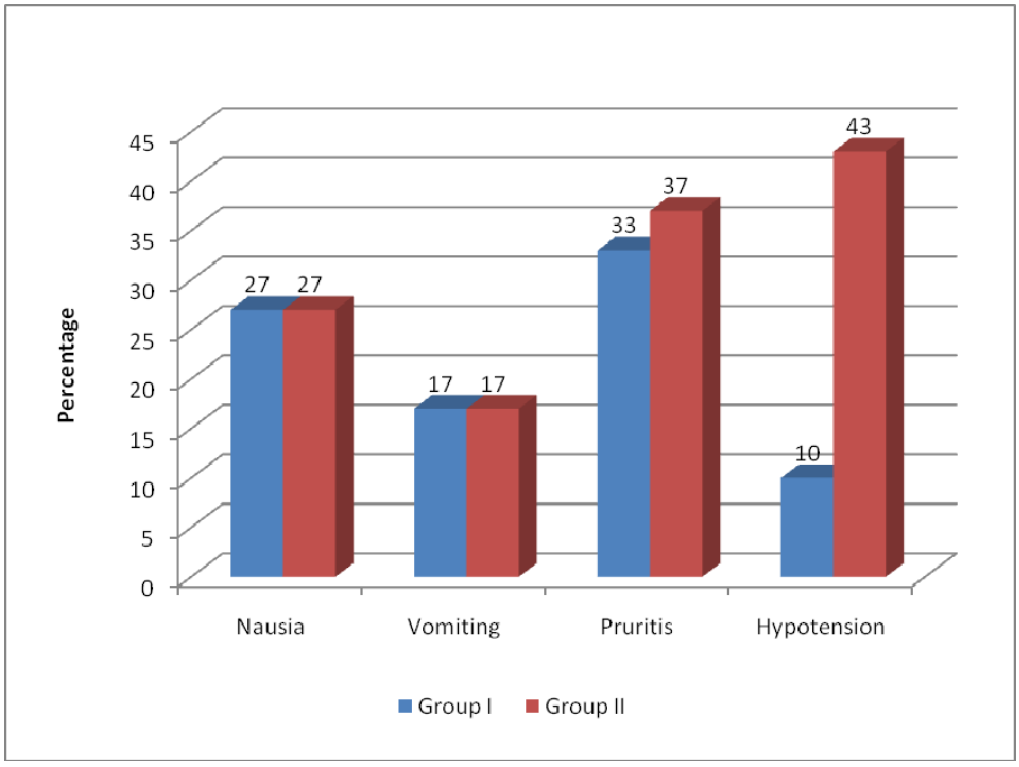
* Chi square test

In the present study group I sedation was present in 33% of cases and group II 37% of cases which was not statistically not significant. Fetal bradycardia 10% in group I and 23% in group II not statistically significant, hypotension was present in 10% in group I and 43% in group II which was statistically significant. Nasuea and vomiting was equal in both the groups, pruritis present in 33% in group I and 37% group II which was not statistically significant.





Graph – 17 : Other Complications



Discussion

DISCUSSION

"Divine is the task of relieve pain" - Hippocrates

Labour is a painful process and childbirth can be the most agonizing event experienced by majority of women. This distress serves no useful purpose and may instead harm the mother and fetus. Painful labour often results in excessive maternal stress and mechanical workload, increased oxygen demand and hyperventilation . These result in increased catecholamine secretion leading to uterine vasoconstriction increased uterine contractility, hypoperfusion of the fetoplacental unit, fetal hypoxia and acidosis.

These responses can easily be obtunded by the providing analgesia during labour. Various methods have been experimented upon to provide pain relief to the laboring mother with minimal detriment on progress of labour and well being of the fetus.

Obstetricians and anaesthetists have always feared the incidence of instrumental deliveries in women receiving labour analgesia could be higher than in those who do not receive it.[55]

Ideally pain relief with regional techniques should be produced with the minimum disturbance to the progress of labour or to sympathetic functions, sensory functions (proprioception) and motor functions of CNS. Thus it is intriguing to the obstetric anaesthetist to strike a balance between patient satisfaction by providing good analgesia, reduce motor block thus making the parturient participate in labour and decrease instrumental deliveries due to prolonged second stage. [56]

Factors contributing to instrumental delivery include –

a) Diminished pain and sensation from uterine contraction leading to diminished Fergusson's reflex and of the perception of the need to push at full dilatation.

b) Reduced motor force due to weakened abdominal musculature and

c) Inadequate rotation of the presenting part due to weakened pelvic floor musculature.[57]

All these factors have generated intense interest in epidural analgesia in 3 forms.

1. Decreased local anaesthetic concentration
2. Combining with opioids
3. Combined spinal epidural technique.

Developments in drugs, needle designs and catheter technology have contributed to development of combined spinal epidural anaesthesia technique which aims at improving the quality, efficacy and safety of neuraxial blockade.

CSE consists of identification of the epidural space and insertion of epidural catheter plus initial intentional placement of an intrathecal dose at opioid, local anaesthetic or both. [9]

Combined spinal epidural analgesia is an effective method of analgesia in labour, intrathecal administration of combination of local anaesthetic and lipophilic opioid provides rapid analgesia. The techniques combines the advantages of speed of onset and reliability of block achieved by subarachnoid

anaesthesia with flexibility povided by the presence of an epidural catheter and avoids their individual disadvantages. [9]

Several different drugs and combination have been described for combined spinal – epidural analgesia in labour. The principal drug providing the intrathecal componenet of analgesia is the lipid soluble opioid. Opioids alone, injected intrathecally in the first stage of labour have variable results in terms of onset, efficacy, duration of analgesia and side effects. [9]

Synergism has been demonstrated when a local anaesthetic is administered together with an opioid allowing enhanced pain relief with fever adverse effects local anaesthetics are very effective for relieving pain of somatic origin. This is particularly important in late first and second stages when the visceral pain of the early first stage of labour gives way to somatic pain. The ability of spinal opioids alone to effectively control this somatic pain is limited.

It is not uncommon for intrathecal opioid alone to fail to provide adequate analgesia when cervical dilatation is more than 5cm and parturient complains of unrelieved perineal discomfort.

It has been demonstrated that addition of local anaesthetic significantly improves analgesia with faster time of onset, greater efficacy and longer duration of analgesia. [9]

Study design :

In the context of above mentioned developments we have undertaken a study to compare intrathecal bupivacaine 1.25mg and fentanyl 25µg with

bupivacaine 2.5 mg and fentanyl 25µg for combined spinal epidural analgesia in the first stage of labour.

Combined spinal epidural analgesia for painless labour was given to 60 parturients belonging to ASA grade I and II admitted to Chigateri General Hospital and Women Child Hospital, Davangere. The parturients were randomly allocated to 2 groups. Group I (n=30) receiving intrathecal bupivacaine 1.25mg and fentanyl 25µg and group II (n=30) receiving intrathecal bupivacaine 2.5mg fentanyl 25 µg followed by epidural top up.

Lee et al performed CSE at L2-3 or L3-4 intervertebral space using a single space needle through needle technique with 18G Tuohy needle and 25G Whitacre spinal needle. [9]

In our study combined spinal epidural technique was done under strict aseptic precautions in L2-L3 or L3-L4 interspace with patient in left lateral position using a single space, needle through needle technique with 18G Tuohy needle epidural space was identified with loss of resistance technique 15mm 25 guage Whitacre spinal needle was the then passed through Tuohy needle intrathecal space was confirmed by observation of free flow of CSF. Intrathecal injection of bupivacaine 1.25 mg with 25 µg (group I n=30) or bupivacaine 2.5 mg with fentanyl 25 µg (group II n=25) both made upto a total volume of 2ml with saline, epidural catheter was passed and secured top ups were given in sitting position when the patient complained of pain. Initiation of labour analgesia was done when parturients were in active phase of labour with good uterine contraction and appropriate cervical dilatation (4-5 in primigravida and 3-4 in multi gravdia).

Onset and duration of sensory and motor blockade.

Collis and Colleagues, popularized the use of intrathecal bupivacaine 2.5 mg and fentanyl 25 µg.

Soresi performed the first intentional CSE in 1937, [10]

Lee's et al compared intrathecal bupivacaine 1.25 mg and Fentanyl 25µg and bupivacaine 2.5mg and fentanyl 25µg for combined spinal – epidural analgesia.

RE Collins, DWL Davies did comparison of combined spinal epidural analgesia Bupivacaine (2.5mg) and Fentanyl (25µg) followed by epidural top ups of 15ml, 0.1% Bupivacaine with 2µg/ml Fentanyl into epidural space. With standard epidural analgesia 25mg/10ml of 0.25 % Bupivacaine injected into epidural space followed by top up of 6-10 ml 0.25% Bupivacaine. Overall satisfaction was greater in combined spinal epidural group.[58] Comparison of maternal satisfaction with low dose CSE (group A) and higher dose standard bupivacaine (group B) epidural analgesia. They concluded onset of analgesia was more rapid in combined spinal epidural group 20 min VAS score 92/98 group A Vs 68/99 group B $p<0.0001$. [58]

Michael J. Paech et al did a randomized, double blinded controlled clinical trial aimed to determine whether the addition of subarachnoid clonidine 15-45µg to fentanyl 20µg and bupivacaine 2.5mg increased the duration of labour analgesia they concluded that onset of analgesia and duration was almost similar addition of clonidine had increased incidence of hypotension. In the group with fentanyl and bupivacaine thoracic sensory dermatomal level was

T5. Onset was within 5 min and duration was more than 90 min with small incidence of motor blockade. [59]

In our study the onset of analgesia was equally rapid with both doses of bupivacaine. Sensory onset of analgesia with mean of 204 sec in group I and 87 sec in group II and mean difference of 117 sec between both the groups.

Dermatomal level achieved at the end of 10 min was T9 in group I and T7-T8 in group II with p-value of <0.001, motor blockade grade 0 in 87% of cases and grade 1 in 13% of cases in Group I. In group II Grade 0 60% of patients and 30% of patients with grade I blockade.

Duration of analgesia for spinal component, mean of 82 min in group I and 104 min in group II with mean difference of 21 min, p-value of 0.001.

Quality of analgesia during early part of labour :

Lee et al VAS pain scores in the first 30 min were similar between the two groups. Median time to first request for additional analgesia was longer in group B (120 min) compared to group A (75 min) p(0.0013).[9]

RE Collis, DWL Davies concluded that overall satisfaction was greater in CSE group than in stand epidural group. CSE had faster onset of analgesia and more of this group were satisfied with analgesia at 20 min. [58]

In our study degree of pain relief was assessed by using visual analog score. There was no significant difference in pain relief between the groups 67% of patient in group I had score of 1-2 and 93% in group II and 33% of patient in group II had score of 3-4 and 7% in group II.

Mode of delivery :

Study by Wong CA et al revealed that neuraxial analgesia in early labour did not increase the rate of cesarean delivery and it provided better analgesia and resulted in shorter duration of labour than systemic analgesia. [60]

The comparative obstetric mobile epidural trial study, confirmed that low dose techniques influence the mode of delivery in both CSE and low dose infusion groups there was an increased percentage of spontaneous vaginal deliveries compared to traditional technique. [49]

Studies have shown CSE to be associated with shorter duration of first stage of labour among nulliparous women compared to epidural analgesia alone. The time of full cervical dilatation was shortened by 78 min in CSE group. [11]

In our study spontaneous vaginal delivery occurred in 84% of cases in group I and 63% of cases in group II. Instrumental delivery with forceps was conducted in 13% of cases in group I and 20% of cases in group II. Caesarean section was done in 3% of cases in group I and 17% of cases in group II. [11]

Effects on the fetus :

Fernando et al conducted a study of 40 nulliparous women receiving low dose CSE. In their study they measured maternal and umbilical plasma concentrations to fentanyl and bupivacaine at delivery. Their neonatal outcome measurements consisted of 1 and 5 min Apgar scores. Umbilical blood gases and neurologic and adaptive capacity scores at 2 and 24 hours post delivery.

They concluded that the concentration of bupivacaine and fentanyl achieved during use of routine CSE for labour were not detrimental to the fetus. [47]

Porter et al detected no difference in Apgar scores, umbilical artery prior neonatal respiration between low dose combinations and plain bupivacaine infusion. [61]

Recent studies however suggest the incidence of caesarean sections linked to the use of opioids is no different whether they are administered intrathecally or epidurally intravenously or not at all.

Craig M. et al studied incidence of fetal heart rate changes after intrathecal fentanyl labour analgesia either 25 µg fentanyl alone or with combination of 2.5 mg Bupivacaine, low incidence of (6-12%) of FHR changes was found none of FHR changes identified resulted in need for caesarean deliveries. [54]

In our study there was not much difference in fetal heart rate changes.

Untoward effects of drugs :

Intrathecal narcotics commonly causes pruritis that can be treated with oral, intramuscular or intravenous narcotic antagonists that do not effect analgesia levels.

Nausea has often been attributed to intrathecal narcotics.

Buvanendran et al studied that intrathecal bupivacaine reduces pruritis and prolongs duration of fentanyl analgesia during labour.

In our present study only hypotension was present in 10% in groupI and 43% in group II which was easily treated with fluids.

Complications :

Complications following CSE are reported in numerous studies, the most dreaded are cardio respiratory catastrophies and inadvertent total spinal block. Norris MC et al quotes inadvertent dural puncture in epidural leading to total spinal in his series as 6.3% and PDPH in CSE in 1.2%.

In our study we did not come across cardiorespiratory catastrophes total spinal, inadvertent puncture of dura or kinking of catheter.

Conclusion

CONCLUSION

A comparative study of combined spinal epidural analgesia is labour with two different doses of intrathecal. Bupivacaine 1.25 mg and Fentanyl 25µg with bupivacaine 2.5 mg and fentanyl 25 µg showed that rapid onset of analgesia was one of the major advantage of combined spinal epidural analgesia and was associated with increased maternal satisfaction.

The onset of analgesia was equally rapid with both doses of bupivacine and the two groups achieved of excellent in major proportion with in 5 min. Duration of analgesia was longer in patients who received the larger dose of bupivacaine. This was associated with higher dermatome levels of sensory block which was reflected in corresponding longer time for regression of the block. However many required subsequent use of their epidural catheter to continue analgesia.

We found lower incidence of motor block with bupivacaine 1.25 mg compared with bupivacaine 2.5 mg. Our results also showed a significantly smaller decrease in arterial pressure with bupivacaine 1.25 mg. This is important clinically as maternal hypotension affects uteroplacental perfusion.

In summary we found that bupivacaine 1.25 mg was as effective as bupivacaine 2.5 mg when added to fentanyl 25 µg for combined spinal epidural analgesia in the first stage of labour, with less motor and sensory block and hypotension onset of analgesia was rapid and was achieved within 5-10 min.

Summary

SUMMARY

The present study was conducted to compare onset duration of sensory and motor blockade and quality of analgesia of intrathecal bupivacaine 1.25mg and fentanyl 25 µg in combined spinal epidural technique in early stage of labour.

60 primigravid or second gravid belonging to ASA grade I & II with singleton pregnancy, cephalic presentation in active labour were selected. Cervical dilatation was 3-4 cm written informed consent was obtained.

IV line was secured with 18G cannula in the non dominant hand, monitors like NIBP and pulse oximeter were connected. The parturient were placed in left lateral semiflexed position. Needle through – needle technique of CSE (18G Tuohy epidural needle 27G whitacre pencil point spinal needle and a 20G polyamide closed end epidural catheter). We used bupivacaine 1.25 mg and fentanyl 25 µg made to 2ml with normal saline in group I and bupivacaine 2.5 mg and fentanyl 25 µg made to 2ml with normal saline. In group II as the initial intrathecal component followed by epidural top up. Top ups were given as 6-8 ml of 0.125% Bupivacaine and 2µg of fentanyl.

FHS, uterine contraction and progress of labour were continuously monitored and active management was initiated in co-ordination with the attending obstetrician, operation theatre was kept ready for any eventuality.

The 2 groups were similar with respect to age distribution height and weight of the parturients.

Onset of analgesia was 204 sec in group I and 87 sec in group II after initial spinal component of CSEA.

The average dermatomal level of sensory blockade achieved was T9 in group I and T7-8 in group II.

Motor block was minimal no motor block in majority of cases 87% in group I and 60% in group II and grade I in 13% of cases in group I and 30% in group II.

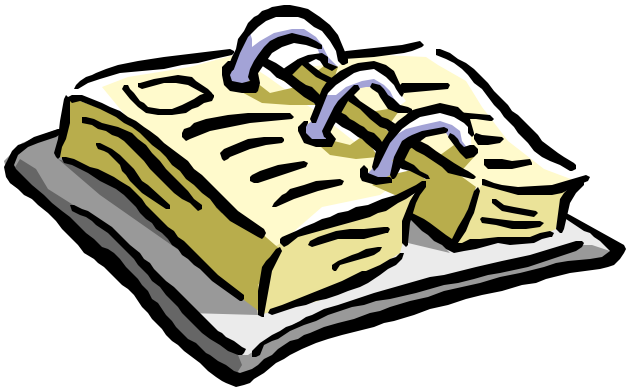
Variation in pulse and blood pressure was minimal more in group II.

In group I 84% of patients normal spontaneous vaginal delivery and 13% instrumental delivery and 3% caesarean section and in group II, 63% had normal spontaneous vaginal delivery and 20% instrumental delivery and 17% caesarian section.

No variations were observed in the APGAR scores.

Complications were observed in form of hypotension sedation and pruritis.

Thus needle through needle technique of CSEA has rapid onset of action with 1.25 mg of bupivacaine and fentanyl 25 µg has minimal motor blockade and sensory blockade and hypotension with rapid onset of analgesia achieved within 4-10 minutes.



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Annexures

PROFORMA

Name :	Age:	IP No.:
Hospital	D.O. Admission	D.O. Delivery

OBSTETRIC HISTORY

Gravida	LMP:	EDD:

PAST HISTORY : TB/HT/DM / Previous epidurals

FAMILY HISTORY : TB/HT/DM

GENERAL PHYSICAL EXAMINATION :

Height / Weight

Pallor / Icterus / Clubbing / Cyanosis / Lymphadenopathy / Oedema.

Pulse:	BP.
Airway	Spine
RS	CVS

INVESTIGATIONS

Hb%	Urine	VDRL
BT	RBS	USG
CT	HbsAg	HIV

Blood grouping & Typing

OBSTETRIC EXAMINATION

Cervical dilatation

MANAGEMENT OF LABOUR ANALGESIA

CSE KIT

Epidural needle 18 G Tuohy needle

Spinal needle 27G whitacre pencil point

Epidural cathether 20G polyamide closed end

Time of spinal injection Site

Time of epidural top up direction of catheter

DRUG ADMINISTERED

Initial dose	Bupivacaine 1.25mg with 25µg of Fentanyl	Bupivacaine 2.5 mg with 25µg of fentanyl
Time of administration		
Time of next request for analgesia.		
Epidural top ups required Time Dose	Yes / no	Yes/ no

Pain relief

- Onset of analgesia
- Peak effect
- VAS

Level of Blockade

Sensory : Dermatomal Levels

Time in mins

Motor : Bromage Scale

Maternal & foetal Monitoring

Time in mins	Pulse	SDP	DBP	SpO_2	FHR	Uterine contraction	Complication
0							
1							
5							
10							
15							
30							
45							
60							
90							
120							

Mode of delivery : Spontaneous / forceps / caesarian with indication

Assessment of the New born

Weight / sex / APGAR – 1 min 5 min

Complication :

Assessment of the technique by the mother

- Degree of overall satisfaction : Excellent / Good / Poor

CONSENT FORM FOR

ANAESTHESIA/OPERATION

I …….. with Hosp. No…….. in my full senses hereby give my consent for …….. or any other procedure deemed fit which is and / or diagnostic procedure / biopsy / transfusion / operation to be performed on me / my son / my daughter / my ward …… age …… under any anaesthesia deemed fit. The nature and risks involved in the procedures have been explained to me to in my own language. The operation / procedure may be televised or photographed for academic and scientific purposes.

Date:

Signature / Thumb Impression of

the Patient / Guardian

Name:

Place: Guardian

Relationship

Full Address

KEY TO MASTER CHART

Ht	-	Height
cm	-	centimeter
wt	-	weight
Kg	-	Kilogram
G	-	Second Gravida
P	-	Primigavida
MSBP	-	Mean Systolic Blood Pressure
MDBP	-	Mean Diastolic Blood Pressure
FHS	-	Foetal Heart Sounds
Hr	-	Hours
Min	-	Minutes
Group I	-	Bupivacaine 1.25mg and Fentanyl 25µg
Group II	-	Bupivacaine 2.5 mg and Fentanyl 25 µg
mg	-	milligrams
gm	-	grams
µg	-	Microgram
VAS	-	Visual Analogue Scale
+	-	Present
_	-	Absent
SI.NO	-	Serial no
IP . NO	-	Inpatient number
T	-	thoracic segment
N	-	normal
C	-	caesarean
F	-	forceps
Y	-	yes

MASTER CHART [Group - 1)

Sl. No.	Group	Name	IP No	Height(cm)	Weight (kg)	Parity	Age (years)	Cervical dilatation	Sensory onset of action	grade of Motor blockade	Dermatomal level	Outcome	VAS after spinal	Time of 2 segment regression	Epidural needed	Heart rate (min)									Blood presure mm of Hg																		APGAR score	Sedation	Fetal bradycardia	Nausea	Vomiting	Pruritis	Hypotension
																0	1	5	15	30	45	60	90	180	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic									
1	I	JAYAMMA	2552	154	64	P	23	4	190	0	T9	N	2	100	Y	110	90	80	88	87	72	74	86	80	110	78	120	80	110	84	120	86	114	80	116	80	120	84	106	84	108	86	7/9	-	-	-	-	-	-
2	I	NASREEN TAJ	3341	152	61	P	27	5	240	0	T10	N	2	100	Y	88	78	86	77	74	80	76	70	71	110	80	90	60	120	80	80	60	110	60	120	80	110	80	112	80	112	80	5/8	+	-	+	-	+	-
3	I	NOORJAN	3524	156	71	G	22	4	180	0	T9	N	1	75	Y	100	90	91	89	82	86	84	83	80	130	80	110	60	112	64	112	68	114	80	120	80	130	80	120	80	120	80	7/9	-	-	-	-	-	-
4	I	VISHLA	3325	157	59	G	26	4	300	0	T11	N	3	65	Y	110	82	81	80	81	80	82	80	79	120	80	110	60	112	64	14	62	116	70	114	68	118	70	120	80	126	82	6/9	-	-	+	-	+	-
5	I	ASMA BANU	4532	153	60	P	20	5	240	0	T10	N	1	75	Y	110	88	80	74	78	74	76	83	80	114	70	10	70	120	80	130	80	120	90	110	80	120	90	120	80	120	80	7/9	-	-	-	-	-	-
6	I	JAYAMMA	2082	158	63	P	19	5	180	0	T9	F	1	70	Y	98	90	94	82	86	82	80	80	79	128	80	120	80	120	80	110	80	128	90	120	90	10	90	120	80	126	82	6/9	+	-	-	+	-	-
7	I	BHAGYA	3228	149	50	G	22	4	240	0	T9	N	4	75	Y	84	90	88	80	74	78	74	77	76	120	80	110	84	114	60	116	64	118	60	120	64	116	68	118	64	116	60	5/9	+	-	+	-	-	-
8	I	MAMATHA	2120	152	55	G	28	5	120	0	T8	N	3	75	N	110	68	76	74	72	80	84	90	92	120	80	110	84	112	64	112	68	114	80	126	80	130	80	126	80	120	80	6/9	-	-	-	-	-	-
9	I	LATHA	5155	160	74	P	25	4	180	0	T9	N	4	105	Y	89	68	62	64	65	67	68	80	90	130	80	90	60	100	80	106	80	120	80	120	80	130	80	140	60	130	80	6/8	-	-	-	-	+	-
10	I	SUNITHA	2104	158	65	P	20	5	200	0	T9	F	3	90	Y	99	80	74	76	81	84	76	81	80	120	80	120	90	110	60	10	80	100	88	100	60	110	88	120	80	120	80	6/9	-	-	-	-	-	-
11	I	RAJIMA BANU	3525	156	60	G	27	4	120	0	T9	F	2	65	Y	98	81	80	79	78	76	77	75	76	126	70	120	80	126	70	114	80	120	80	114	62	118	66	116	64	120	68	6/8	+	-	-	-	-	-
12	I	CHANDRAMA	2121	150	53	P	25	4	180	I	T9	N	1	60	Y	120	80	88	87	89	90	100	80	82	120	80	110	84	114	86	16	88	118	80	120	90	114	86	110	88	120	90	4/8	-	+	-	-	-	-
13	I	SUVARNA	3215	160	59	G	24	5	180	0	T8	N	3	75	Y	97	80	81	82	84	84	86	90	92	120	80	100	70	90	50	110	70	120	80	128	84	110	80	112	60	112	64	7/9	-	-	-	+	-	+
14	I	ASHA	3222	150	61	G	20	4	300	I	T10	N	1	60	Y	96	90	97	84	92	94	87	88	82	130	90	120	86	110	82	114	86	16	82	10	84	108	88	112	90	120	86	7/9	-	+	-	-	+	-
15	I	MADHULEMMA	2045	154	59	P	20	5	220	0	T10	F	3	105	Y	110	80	81	82	72	78	70	81	72	120	80	90	60	106	80	120	80	98	80	100	70	120	80	118	66	116	64	5/8	+	-	-	-	+	-
16	I	MANJULA	3422	159	66	G	25	4	300	0	T8	N	1	60	Y	100	80	81	82	83	84	80	81	80	126	70	120	80	126	70	114	80	120	80	114	62	118	66	116	64	120	68	5/7	+	-	-	+	+	-
17	I	SABEENA BANU	2114	152	52	P	22	4	240	0	T10	N	2	120	Y	100	82	84	82	78	81	74	81	82	110	72	106	70	110	74	104	70	106	80	170	78	100	70	108	70	120	68	5/9	-	-	+	-	-	-
18	I	ZAMAN BI	3521	163	68	G	30	4	180	0	T10	N	2	75	Y	89	92	68	72	84	74	76	80	90	120	90	110	80	90	60	70	50	110	60	120	60	130	60	140	60	130	80	6/8	-	-	+	-	-	+
19	I	LALITHAMMA	3479	160	70	G	25	5	240	0	T9	N	1	80	Y	82	76	74	70	80	82	80	81	80	110	80	120	80	110	80	112	84	118	80	120	64	130	68	120	80	120	80	6/9	+	-	-	-	-	-
20	I	SHARADA	2077	149	49	P	27	4	200	0	T8	N	1	65	Y	96	86	96	84	82	84	82	75	76	130	80	110	70	102	60	105	70	90	60	90	60	110	60	116	64	120	68	6/8	-	-	+	-	+	-
21	I	JABINA	2095	153	58	P	26	5	230	0	T10	N	1	75	Y	96	62	60	80	90	94	80	90	92	120	80	120	80	120	82	130	86	110	86	120	82	110	88	112	60	112	64	7/9	+	-	-	-	-	-
22	I	ROOPA	3475	153	59	G	22	4	120	0	T10	N	4	80	Y	110	80	82	86	70	72	76	81	82	110	60	120	60	110	68	108	70	106	68	108	70	106	68	108	70	120	68	7/9	-	-	-	-	+	-
23	I	ROOPA	3537	157	62	G	25	5	240	0	T9	N	3	80	Y	100	90	86	82	84	80	80	81	72	130	60	120	66	110	68	108	70	18	80	120	64	114	62	118	66	116	64	5/8	-	-	+	-	-	-
24	I	NAGARATHNA	4569	151	52	P	25	4	220	0	T10	N	2	105	Y	84	76	74	72	68	64	68	81	80	110	80	100	90	10	88	116	89	120	88	114	88	120	86	116	64	120	68	5/7	-	-	+	-	-	-
25	I	SHAMIN BANU	3577	148	49	G	21	5	120	I	T8	N	1	90	Y	102	80	81	82	70	71	72	80	82	120	80	114	78	116	76	112	70	102	70	108	74	106	80	110	88	120	90	5/8	+	-	-	+	-	-
26	I	MUBIAA BANU	3603	151	57	G	22	4	90	0	T9	N	1	100	Y	89	70	71	76	75	81	82	86	80	120	78	116	80	118	76	120	70	110	70	108	76	108	80	106	84	108	86	7/9	-	-	-	-	-	-
27	I	FATIMA	4526	152	57	P	20	4	220	0	T9	N	3	75	Y	72	68	75	92	88	84	72	88	82	120	80	100	70	90	80	80	50	100	60	110	70	100	80	112	90	120	86	7/9	-	-	-	-	+	-
28	I	HEMALATHA	2083	153	59	P	23	4	200	I	T11	C	2	85	Y	78	68	64	80	84	80	71	77	76	120	80	110	82	110	88	110	88	110	82	120	80	112	88	118	64	116	60	5/9	+	+	-	-	-	+
29	I	NEELAMMA	3665	158	68	G	22	4	220	0	T10	N	2	105	Y	92	80	78	72	70	68	69	70	71	120	80	100	86	90	88	110	84	120	80	128	86	110	88	112	80	112	80	5/8	-	-	-	-	+	-
30	I	SAKAMMA	4559	147	54	P	20	5	240	0	T9	N	2	90	Y	90	88	80	74	78	74	76	90	92	120	88	120	80	110	86	116	80	124	84	114	80	120	86	126	80	120	80	6/9	-	-	-	+	-	-

MASTER CHART (Group - 2)

Sl. No.	Group	Name	IP No	Height(cm)	Weight (kg)	Parity	Age (years)	Cervical dilatation	Sensory onset of action	Motor grade of blockade	Dermatomal level	Outcome	VAS after spinal	Time of 2 segment regression	Epidural needed	Heart rate (min)									Blood presure mm of Hg																		APGAR score	Sedation	Fetal bradycardia	Nausea	Vomiting	Pruritis	Hypotension
																0	1	5	15	30	45	60	90	180	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic									
1	II	SEETHA	3358	152	60	P	22	4	60	0	T7	N	1	110	Y	102	10	98	96	92	89	80	78	78	118	78	100	82	116	80	110	88	112	80	114	62	116	70	100	80	114	62	6/9	+	-	-	-	-	-
2	II	GIRIJA	2438	150	57	P	22	4	100	I	T9	F	1	90	Y	104	96	92	89	80	78	76	78	82	121	86	126	80	120	70	118	60	90	60	78	50	112	80	110	80	78	50	8/9	-	-	-	+	-	+
3	II	KAUSAI BANU	1996	155	58	G	20	4	90	I	T9	N	1	90	N	102	90	86	72	85	76	65	66	69	114	80	116	80	120	80	112	84	100	88	110	86	114	86	112	84	110	86	8/9	-	-	+	-	-	-
4	II	ROOPA	2098	151	50	G	25	5	90	I	T8	N	1	110	Y	98	93	85	83	70	68	68	71	70	120	80	112	80	113	82	100	60	112	70	120	80	116	70	110	80	120	80	6/9	+	-	-	-	+	+
5	II	BASAMMA	3339	146	55	P	30	5	60	I	T6	N	1	100	Y	92	81	83	86	90	78	82	80	89	110	80	110	80	114	80	116	82	100	84	102	80	104	82	108	84	102	80	5/8	-	-	-	-	-	-
6	II	ROOPA	2098	153	56	G	25	4	60	0	T7	N	1	100	N	96	77	78	80	63	62	61	64	71	116	80	114	78	118	76	116	80	114	84	112	88	110	86	120	86	112	88	8/9	-	-	-	-	-	-
7	II	SUMA	2362	151	61	P	25	4	90	I	T8	C	3	150	N	98	80	81	82	90	91	86	80	78	112	88	100	88	112	76	108	78	108	88	106	84	118	80	106	86	106	84	5/9	-	-	+	-	+	-
8	II	SAVITHA	3277	157	55	P	20	5	90	0	T7	N	1	90	Y	92	78	80	81	82	84	86	80	78	128	80	124	70	100	70	90	60	84	56	110	80	110	82	120	84	110	80	6/9	+	-	-	-	-	+
9	II	KALPANA	3052	152	56	P	21	5	60	I	T8	C	3	120	Y	87	81	86	72	83	85	74	75	78	124	82	120	80	124	82	122	84	120	78	122	80	118	80	120	82	122	80	5/9	-	-	-	-	-	-
10	II	ASMA	2089	158	60	G	24	4	60	0	T7	N	1	90	Y	88	80	78	77	76	79	75	74	71	110	80	104	98	124	72	120	78	116	80	114	84	100	84	112	86	114	84	8/9	+	-	-	-	+	-
11	II	SAHEENA BANU	3558	157	58	P	20	5	80	0	T10	F	1	90	Y	98	90	91	92	93	84	86	87	88	112	84	116	86	100	82	110	86	100	76	90	60	112	60	120	80	90	60	4/9	-	-	-	-	-	+
12	II	VISHALA	3583	161	65	P	22	5	180	0	T7	N	1	80	Y	86	84	73	82	76	77	71	80	79	122	78	116	76	122	74	114	78	110	80	118	82	116	84	118	78	118	82	4/9	+	-	+	-	+	-
13	II	CHAYA	2093	151	53	G	21	4	90	0	T8	N	1	130	Y	102	92	81	82	94	78	64	77	72	130	90	120	80	100	60	96	60	82	50	110	80	120	80	112	80	110	80	6/9	-	-	-	+	-	+
14	II	SABREENBANU	2107	154	57	G	21	4	90	II	T7	F	1	80	Y	88	81	79	84	90	89	66	65	76	120	80	100	60	90	56	84	60	118	80	120	80	130	80	120	80	120	80	5/9	+	+	-	-	+	+
15	II	CHANDRAKALA	2028	152	60	P	23	5	70	0	T9	F	1	110	Y	90	78	72	76	78	72	70	81	82	120	86	110	82	100	80	90	70	80	60	98	70	110	70	120	80	98	70	7/9	+	-	-	+	-	+
16	II	SAKSHI BAI	2560	160	70	G	24	5	60	0	T8	N	2	80	N	112	91	82	66	69	64	63	61	68	110	60	100	60	90	50	80	50	100	60	120	80	124	85	116	80	120	80	7/9	-	-	-	-	-	+
17	II	LAKSHMI	2126	160	76	P	20	5	70	0	T7	C	1	140	N	98	80	78	79	75	76	81	82	79	114	90	110	72	120	74	100	76	112	78	116	80	114	82	112	80	116	80	5/7	-	-	+	-	+	-
18	II	MOSINA KHAN	2063	159	63	P	20	4	90	I	T8	N	1	120	Y	88	78	74	76	72	72	78	82	80	132	84	128	80	110	70	90	60	92	62	84	62	110	70	112	80	84	62	7/9	-	+	-	-	-	+
19	II	MANJULA	2410	154	60	G	21	5	70	0	T8	F	1	120	N	98	89	81	78	73	72	71	68	69	120	80	100	60	90	50	80	50	100	60	120	60	110	60	120	60	120	60	8/9	-	+	+	-	-	+
20	II	PAVITHRA BAI	2518	159	68	G	22	5	90	II	T10	N	1	100	Y	92	87	81	75	74	76	83	72	71	130	80	110	82	114	78	116	76	118	84	122	82	120	80	124	74	122	82	7/9	+	-	-	+	+	-
21	II	SUNITHA	2022	161	60	G	25	4	110	0	T6	N	1	110	N	98	95	87	74	75	71	73	74	67	130	80	120	80	110	80	100	60	90	60	80	60	110	60	120	80	80	60	8/9	-	+	+	-	-	+
22	II	KANOOR	2044	148	49	G	22	4	180	0	T7	N	2	130	Y	102	88	82	73	77	72	67	62	60	120	80	108	80	112	84	102	82	114	80	118	78	116	76	118	74	118	78	6/9	+	+	-	-	+	-
23	II	LATHA	3076	151	54	P	19	4	90	I	T7	F	1	105	N	102	80	81	78	77	79	82	83	84	124	80	122	82	120	80	118	78	116	76	120	74	118	80	118	76	120	74	8/9	-	+	-	-	-	-
24	II	AFREEN BANU	2912	158	58	P	20	5	110	0	T7	N	2	90	Y	98	88	84	82	78	88	82	81	80	112	80	102	82	114	80	112	84	108	82	92	64	90	60	112	80	92	64	8/9	-	-	-	+	-	+
25	II	GEETHA	3477	153	50	P	20	4	90	0	T9	C	1	105	Y	92	78	74	75	86	88	82	83	80	120	80	114	80	112	74	116	76	118	80	118	82	120	74	124	70	118	82	5/9	+	-	+	-	+	-
26	II	ULFATH UNNISA	2119	162	67	G	24	5	60	0	T7	N	1	120	Y	78	82	83	78	80	70	67	73	73	130	80	118	84	116	82	114	80	120	85	122	84	124	86	120	88	122	84	8/9	-	-	+	-	-	-
27	II	NAGAMMA	2243	153	52	G	22	4	60	0	T8	N	1	100	Y	80	78	81	81	71	68	74	69	75	120	80	114	78	116	76	120	74	116	76	118	78	118	78	116	80	118	78	7/9	-	-	-	-	+	-
28	II	MUSHRATH	2789	150	52	P	22	4	120	I	T8	C	1	120	Y	104	90	92	91	89	90	98	78	70	110	78	108	70	116	76	114	78	116	84	118	80	108	82	104	86	118	80	7/9	-	-	-	-	-	-
29	II	RASHIDA BANU	2632	160	61	G	32	5	90	0	T8	N	1	80	Y	86	72	84	71	72	64	66	70	74	130	80	100	80	112	80	90	60	84	60	120	80	130	80	100	80	120	80	8/9	+	-	-	-	-	+
30	II	MANJAMMA	2442	159	60	G	25	4	60	0	T8	N	1	70	N	89	70	71	81	82	83	85	75	72	136	80	120	78	128	76	126	74	130	76	120	74	128	72	122	70	120	74	6/9	-	-	-	-	-	+	-