"A COMPARATIVE STUDY OF EPIDURAL ADMINISTRATION OF 0.25%BUPIVACAINE VERSUS 0.25% BUPIVACAINE+8MG DEXAMETHASONE ON POSTOPERATIVE ANALGESIAFOLLOWING GYNAECOLOGY SURGERY"

A Dissertation submitted to

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

In partial fulfilment of the requirements

for the award of the degree

M.D. (BRANCH-X)

ANAESTHESIOLOGY

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL

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APRIL 2017

DECLARATION BY THE CANDIDATE

I, **Dr.K.MAHENDIRAVARMAN,** solemnly declare that the dissertation, titled **"A COMPARATIVE STUDY OF EPIDURAL ADMINISTRATION OF 0.25%BUPIVACAINE VERSUS 0.25% BUPIVACAINE+8MG DEXAMETHASONE ON POSTOPERATIVE ANALGESIA FOLLOWING GYNAECOLOGY SURGERY" ,** is a bonafide work done by me during the period of MARCH 2016 TO AUGUST 2016 at Government Stanley Medical College and Hospital, Chennai under the expert supervision of **Dr.NAHEEDAZHAR, M.D., D.A.,** Professor, Department Of Anaesthesiology, Government RSRM lying in Hospital (Government Stanley Medical College), Chennai.

This thesis is submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the rules and regulations for the M.D. degree examinations in Anaesthesiology to be held in April 2017.

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CHAPTER 1

INTRODUCTION

The word pain is derived from the Greek term poine ("Penalty").[1] Pain is not just a sensory modality but it is an experience. The international association for the study of pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage". The father of the field of pain management as we know it today was John.J.Bonica and he founded the international association for study of pain in 1974. other than psychological trauma, pain is shown to affect the physiology of almost all the system including respiratory, cardiovascular and metabolic profile thereby increasing morbidity.[2]

Anaesthesia can be categorised local, conscious sedation, regional and general anaesthesia(GA). Regional anaesthesia further separated into neuraxialblock and peripheral nerve block. The type of anaesthesia a patient receives depends on the procedure being performed and his/her physical and emotional status,as well as medical and psychological health.

Intrathecal anaesthesia and epidural anaesthesia (EA) are the most popular regional anaesthesia techniques used for lower abdomen surgeries. Intrathecal anaesthesia also called as spinal anaesthesia has few limitations like, short duration of anaesthesia, extension of anaesthesia can be done for

prolonged surgeries but chances of life threatening complications are more,shorter duration of postoperative analgesia and troublesome complication of postdural puncture headache (PDPH)[3].

EA is becoming one of the most useful and versatile procedures in moderm anaesthesiology. It is unique in that it can be placed at virtually any level of the spine, allowing more flexibility in its application to clinical practice.it is more versatile than spinal anaesthesia, giving the clinician the opportunity to provide anaesthesia and analgesia, as well as treatment of chronic disease syndromes. it can be used to supplement GA, thereby providing a more hemodynamically stable operative course. It provides better postoperative pain control and rapid recovery from surgery. When combined with spinal anaesthesia in a technique called CSE , or combined spinal epidural, benefits of both techniques can be combined and shortcomings of each avoided. Hence EA is the most preferred anaesthetic technique for lower abdomen surgeries these days.

The advantages of EA being it,

- provides effective surgical anaesthesia and can meet the extended duration of surgical needs.
- provides prolonged postoperative analgesia

- reduces the incidence of hemodynamic changes as a result of sympathetic blockade as it can produce segmental anaesthesia, unlike subarachnoid block anaesthesia.
- the incidence of PDPH is not there as dura is not pierced.

The knowledge of specific opiate receptors in the substantia gelatinosa of the posterior horn of spinal cord resulted in widespread use of epidural opiods in tha treatment of acute and chronic pain(Pert and Snyder 1973) .though morphine has already established its role epidural administration for pain relief, its side effects like respiratory depression, nausea, vomiting, urinary retention etc., as made physician to search for a better drug for epidural employment.

Epidural steroids have been used successfully for long time for chronic painsyndrome. The safety of epidural steroids is well established. Based on the above evidences and concepts in this study we used dexamethasone epidurally to study the effects on acute postoperative pain.

HISTORY OF EPIDURAL ANESTHESIA & ANALGESIA

- Jean Enthuse Sicard (1872-1929) and Fernand Cathelin (1873-1945)independently introduced cocaine through the sacral hiatus in 1901,thereby becoming the first practitioners of caudal (epidural) anesthesia.
- Sicard - a neurologist, used the technique to treat sciatica and tabes, butCathelin used the technique for surgical anesthesia.
- Arthur Läwen (1876-1958)- an early proponent of regional anesthesia, successfully used caudal anesthesia with large volumes of procaine forpelvic surgery.
- Heile - published an extensive study of the epidural space in 1913. Hisunique approach was to enter the epidural space through the intervertebralforamina.
- In 1921, Fidel Pagés (1886-1923), a Spanish military surgeon- devised atechnique to introduce epidural procaine at all levels of the neuraxis. Hismethod was to use a blunt needle and then feel and hear entry of theneedle through the ligamentum flavum.
- An important innovation was Dogliotti's method of identification of theepidural space. His textbook illustrates the use of continuous pressure

onthe plunger of a saline filled syringe as the needle is advanced through theligamentous structures.

- Gutierrez of Argentina developed the “hanging drop” sign, which is stillused by some anesthesiologists to identify the epidural space. William T.Lemmon (1896-1974) used a 17-gauge, malleable, silver needle that wasconnected through a hole in the operating room table to rubber tubing anda syringe.

- Edward B. Tuohy (1908-1959) used a ureteral catheter threaded through alarge Huber-tipped spinal needle to provide continuous spinal anesthesia.

- Behar in 1979 first reported the use of epidural morphine for treatment ofpain.

- Robecchi and Capra in 1952 treated radiculopathy with periradicularhydrocortisone. It is the first documented use of epidural steroids .

ANATOMY OF EPIDURAL SPACE

Everything outside the dural sac but within the vertebral canal can be considered to constitute the epidural space.

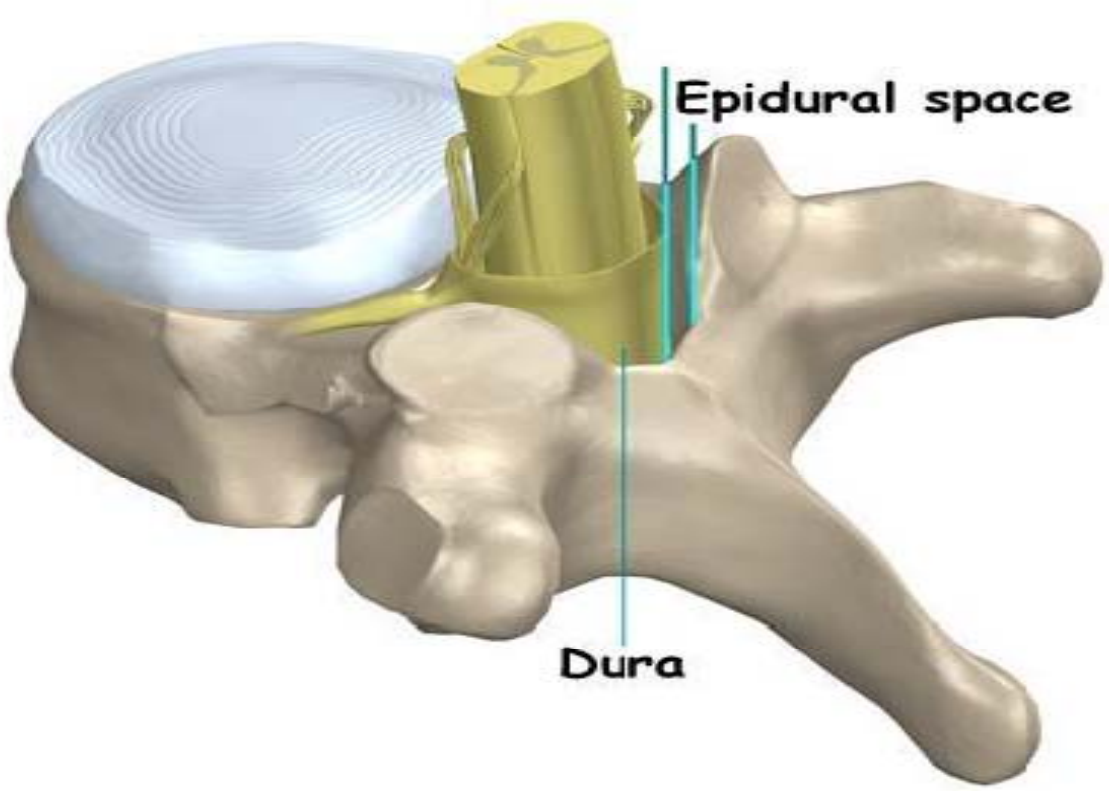
Boundaries of epidural space:

- The walls of vertebral canal including the vertebral bodies and discsanteriorly
- Pedicles laterally
- Lamina and ligamentum flava posteriorly

Epidural space is a potential space normally contains – fat, vessels andnerves. The cranial epidural space is entirely empty. The epidural fat which is nearly fluid in texture permits gliding movement of the neural structures and provides a padding effect. The distribution of epidural contents is highly non uniform.

Separated by these empty areas, the epidural contents occur as a series of metamerically and circumferentially discontinuous compartments. In contrast to this below L4, the dural sac tapers resulting in complete filling of epidural fat. Thus there will be difficulty in delivering local anaesthetic to the L5 and sacral nerve roots during epidural anaesthesia, since solution is not confined in close proximity with neural structures at these levels.

Figure 1 : Anatomy of epidural space



Posterior epidural compartment:

A triangular part of fat pad fills the dura posterior to epidural space. It is enclosed by ligamentum flava but also extends under the caudal most portion of lamina above. The largest posterior epidural compartment is at the mid lumbar level with progressive decrease in anteroposterior dimension at thoraciclevels[4]. Rostral to C7 level the posterior epidural space vanishes and theposterior dura lies in contact with the ligamentum flavum and the laminar bone.

A cleft like space between epidural fat and the canal wall allows passage ofcatheters and injected fluids with only a minor impediment in posterior midline.This arrangement of opposing non adherent tissue plane is ideally

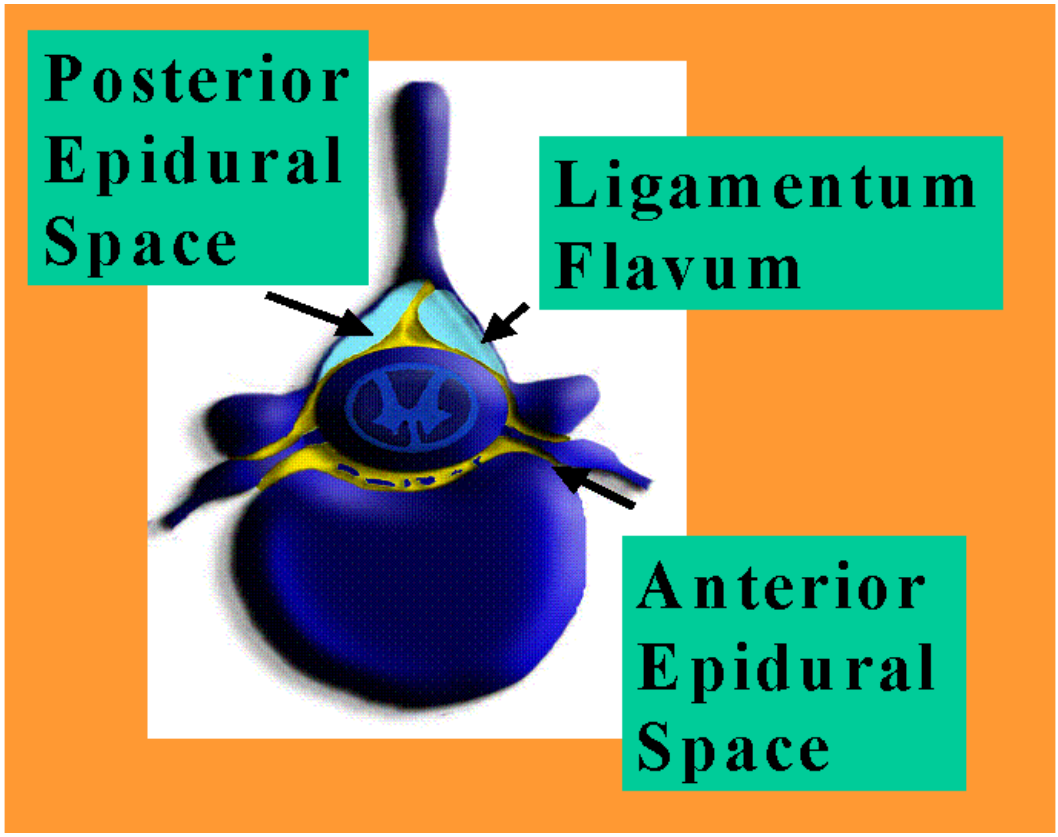
designed todemonstrate the normal subatmospheric pressure within tissues, generated bythe usual action of lymphatics and the balance of osmotic and hydrostatic forcesacross the capillary endothelium.

Lateral epidural compartment:

No epidural contents exist lateral to the dural sac where it is in contactwith the vertebral pedicles. This compartment forms just medial to eachintervertebral foramen and is filled with segmental nerves, vessels and fat.

The pressure in the epidural space closely reflects abdominal pressure because of the flexibility of tissues and lack of rigid barrier. Increasedabdominal pressure such as during a cough or pregnancy is therefore readily transmitted to the epidural space.

Figure 2 : Schematic appearance of epidural space



Anterior epidural space:

The anterior epidural compartment is separated from rest of vertebral column by fascia of posterior longitudinal ligament. The spread of injected drug anterior to plane of posterior longitudinal ligament is effectively blocked by this membrane. At the level of the narrow mid portion of the vertebral body this is almost occupied by internal vertebral plexus. Catheters that transgress into the anterior epidural space through the fascia of the posterior longitudinal ligament are likely to enter the venous plexus.

Functional implications of epidural space:

The spread of injected solutions is circumferential at a given level andpasses out of the intervertebral foramen and likewise freely passeslongitudinally within the vertebral canal.

As the catheter is advanced through the needle, there may be a briefresistance to advancement as the tip encounters the dura. CT scan shows thatcatheter tip inserted 3 cm into the vertebral canal most commonly travellaterally to the internal aspect of an intervertebral foramen because of thestiffness of the short segment of catheter that has emerged from the needle.Even when the catheter tip lies exterior to the intervertebral foramina in theparavertebral space, the distribution of the injected solution is preferentiallyback into the vertebral canal .

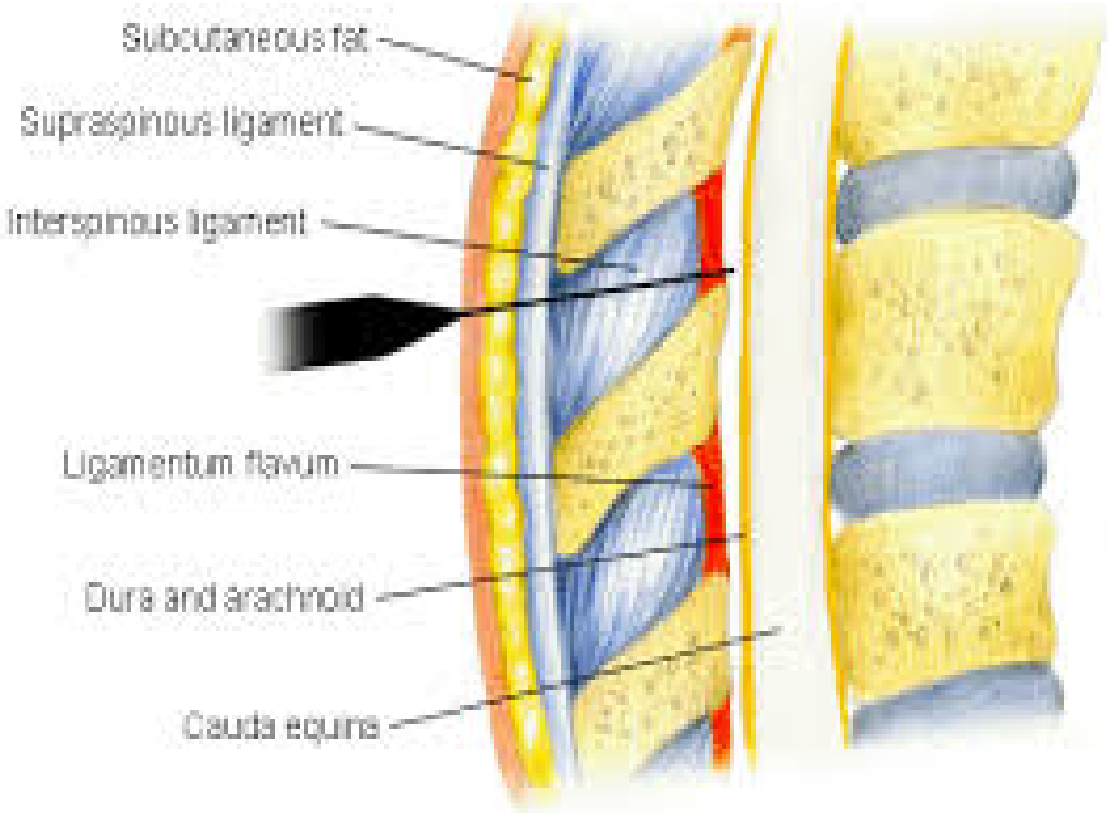
Table 1: Epidural space width and dural thickness

Region	Epidural space	Thickness of dura
Cervical	1.0 -1.5mm	1.5 mm -2.0 mm
Upper thoracic	2.5-3.0mm	1.0 mm
Lower thoracic	4.0 -5.0 mm	1.0 mm
Lumbar	5.0-6.0 mm	0.66-0.33mm

Table 2 ： Thickness of Ligamentum Flavum

Site	Thickness of ligamentum flavum
Cervical	1.5-3.0
Thoracic	3.0-5.0
Lumbar	5.0-6.0
Caudal	2.0-6.0

Figure 3: Layers pierced by epidural needle



PHYSIOLOGICAL EFFECTS OF EPIDURAL BLOCKADE

Epidural neural blockade implies sympathetic blockade accompanied by somatic blockade in the form of sensory and motor blockade alone or in combination.

CARDIOVASCULAR EFFECTS:

Blockade ofsympatheticinnervation accounts for the cardiovascularresponses. Preganglionic sympathetic innervation – regulates regional bloodflow. Post ganglionic sympathetic innervations – controls cardiac function and vascular tone. Peripheral sympathetic blockade causes vascular dilatation inpelvis and lower limbs when lower thoracic and lumbar segments are blockedwith epidural anaesthesia.

Cardiovascular depression is atleast partly related to the level of sympatheticblockade. Vascular absorption of local anaesthetic and addition ofvasoconstrictor may result in significant hemodynamic changes after epidural

but not after subarachnoid blockade.

Lumbar epidural anaesthesia with sympathetic blockade below T10 resultsin minimal vasodilatory consequences because fewer vasoconstrictor fibres areincluded and neither the sphlanchnic nerves nor the nerve supply to the adrenalmedulla are affected. Since muscle veins lack sympathetic innervation,venodilatation of the extremities is limited to skin and so minimal capacitanceincrease results from blocks of the lower extremities[5].Lumbar epiduralanesthesia with a sympathetic blockade extending to the lower segments mayoccasionally be associated with profound bradycardia and circulatory collapsewithout any obvious precipitating event.

RESPIRATORY EFFECTS:

Following aspects may influence respiration.

- Sensory neural blockade reduces nociceptive afferent drive to respiratory center.
- Motor neural blockade of intercostals muscles, abdominal muscles and diaphragm.
- Sympathetic neural blockade with resultant change in cardiac output .
- Vagal dominance.

The potential for phrenic nerve palsy is rare with epidural block.Respiratory arrest is rare and commonly associated with extensive sympatheticblockade, reduced cardiac output and reduced oxygen to the CNS. In patientswith severe pain epidural block probably improves Vital capacity andFunctional residual capacity as well as PaO2. Thoracic epidural anesthesia doesnot impair the hypoxic drive. The inhibitory reflex of phrenic nerve motor driveis interrupted with thoracic epidural anesthesia resulting in increaseddiaphragmatic activity .

NEUROENDOCRINE EFFECTS OF EPIDURAL BLOCKADE:

Most of the surgically induced endocrine and metabolic changes areabolished by an appropriate level of sensory blockade produced by regionalanesthesia. Surgical stress responses during major upper abdominal and

thoracicprocedures are not effectively amileorated by epidural anaesthesia due toincomplete blockade of nociceptive pathways. Sympathetic block abolishes theincrease in renin activity in response to arterial hypotension. Vasopressinsystem is activated in response to hypotension

EPIDURAL BLOCKADE AND MOTOR FUNCTION:

The degree of motor blockade increases as dose of drug increases. Usage ofdilute concentration of local anesthetics facilitates ultra early ambulation. Motor blockade in lower limbs is assessed by bromage scale.

Table 3 : BROMAGE SCALE

No block (0%)	Full flexion of knees and feet possible
Partial (33%)	Just able to flex knees, still full flexion of feet possible
Almost complete(66%)	Unable to flex knees, still flexion of feet
Complete (100%)	Unable to move legs or feet

THERMOREGULATION AND SHIVERING:

Hypothermia is common in patients undergoing surgery with epiduralanesthesia and it results from heat loss to the cold environment due tosympathectomy induced vasodilatation and in part from redistribution of heat from central to peripheral regions.

Pregnancy may enhance the contribution of spinal thermoregulatory input.Injection of epidural pethidine 25mg or epidural fentanyl 50 μg abolishesshivering from epidural local analgesia.

EFFECTS ON GIT:

Epidural block extending from T6 to L1 effectively denervates thesphlanchnic sympathetic supply to the abdominal viscera. As a resultparasympathetic activity predominates resulting in contraction of gut. Thoracicepidural anesthesia with local anaesthetics shortens the duration ofpostoperative paralytic ileus. Unopposed parasympathetic activity withblockade of afferent nociceptive and thoracolumbar efferents produces ashortened postoperative colonic ileus.

Epidural anesthesia have protective action on gut due to improved mucosalblood flow. This increase in blood flow may contribute to the healing of gutanastomosis. Epidural anesthesia with local anaesthetic seems to be the bestmethod for relieving pain after gastrointestinal surgery.

EFFECTS ON BLOOD LOSS:

Patients receiving epidural block had operative blood losses that were halfthose associated with general anaesthesia. Blood loss can be reduced as far as30 to 40 % if epidural block is used for hip surgery. Factors that reduce bloodloss include mild reduction in arterial blood pressure, increase in venouscapacitance, prevention of high venous pressure in response to sympatheticactivity resulting from pain and use of appropriate position.

EPIDURAL ANESTHESIA & ANALGESIA

Epidural anesthesia is a central neuraxial block technique which providessegmental blockade. Improvements in equipment, drugs and technique havemade it a popular and versatile anesthetic technique, with applications insurgery, obstetrics and pain control. Its versatility means it can be used as an anesthetic, as an analgesic adjuvant to general anesthesia, and forpostoperative analgesia in procedures involving the lower limbs, perineum,pelvis, abdomen and thorax.

GENERAL INDICATIONS:

Epidural anesthesia can be used as sole anesthetic for proceduresinvolving the lower limbs, pelvis, perineum and lower abdomen. It is

possibleto perform upper abdominal and thoracic procedures under epiduralanesthesia alone, but the height of block required, with its attendant sideeffects, make it difficult to avoid significant patient discomfort and risk.

The advantage of epidural over spinal anesthesia is the ability tomaintain continuous anesthesia after placement of an epidural catheter, thusmaking it suitable for procedures of long duration. This feature also enablesthe use of this technique into the postoperative period for analgesia, usinglower concentrations of local anaesthetic drugs or in combination withdifferent agents.

SPECIFIC INDICATIONS:

Hip and knee surgery:

Internal fixation of a fractured hip is associated withless blood loss when central neuraxial block is used. The rate of deep venousthrombosis is reduced in patients undergoing total hip and knee replacement, when epidural anaesthesia is used.

Vascular reconstruction of the lower limbs:

Epidural anesthesia improves distal blood flow in patients undergoing arterial reconstruction surgery.

Amputation:

Patients given epidural anaesthesia 48-72 hours prior to lower limb amputation may have a lower incidence of phantom limb pain following surgery.

Thoracic trauma with rib or sternum fractures:

Adequate analgesia inpatients with thoracic trauma improves respiratory function by allowing the patient to breathe adequately, cough and cooperate with chest physiotherapy.

Obstetrics:

Epidural analgesia is indicated in obstetric patients in difficult or high-risk labour.Caesarean section performed under central neuraxial block is associated with a lower maternal mortality and better perioperative outcome.

CONTRAINDICATION OF EPIDURAL ANESTHESIA:

ABSOLUTE:

- Patient refusal
- Infection at the site of injection
- Coagulopathy or other bleeding diathesis
- Severe hypovolemia
- Increased intracranial pressure
- Severe stenotic valvular heart disease with low fixed cardiac output syndrome.
- Severe hypotension
- Known allergy to local anesthetics

RELATIVE:

- Sepsis
- Uncooperative patient
- Pre-existing neurological disease
- Severe spinal deformities
- Patients on anticoagulants.

ADVANTAGES:

- Use of perioperative epidural anesthesia and analgesia, especially with alocal anesthetic–based analgesic solution, can attenuate thepathophysiologic response to surgery and may be associated with areduction in mortality and morbidity when compared with analgesia withsystemic opioid agents. Use of epidural analgesia can decrease theincidence of postoperative gastrointestinal, pulmonary, and possiblycardiac complications by inhibiting sympathetic outflow, decreasing thetotal opioid dose, and attenuating spinal reflex inhibition of thegastrointestinal tract.

- Postoperative thoracic epidural analgesia can facilitate return ofgastrointestinal motility without contributing to anastomotic boweldehiscence. Patients who receive epidural local anesthetics have an earlierreturn of gastrointestinal motility after abdominal surgery.

- Perioperative use of epidural analgesia with a local anesthetic–basedregimen in patients undergoing abdominal and thoracic surgery decreasespostoperative pulmonary complications, presumably by preservingpostoperative pulmonary function by providing superior

analgesia andthus reducing splinting behavior and attenuating the spinal reflexinhibition of diaphragmatic function.

- Use of postoperative thoracic, but not lumbar epidural analgesia maydecrease the incidence of postoperative myocardial infarction[7], possiblyby attenuating the stress response hypercoagulability, improvingpostoperative analgesia and providing favorable redistribution of coronary blood flow.

FACTORS AFFECTING EPIDURAL BLOCKADE:

SITE OF INJECTION AND NERVE ROOT SIZE:

Injection of drug close to nerve roots results in rapid and intenseblockade. After lumbar epidural injection, a somewhat greater cranial thancaudal spread of analgesia occurs. The spread of analgesia is even when drugs are injected in midthoracic epidural injection.

Concentration of large number of nerve fibres within upper thoracic andcervical segments makes them resistant to blockade with epidural injections.Caudal epidural block spreads from S5 and the S1 segment is the last to beblocked.

VOLUME:

Segmental dose is the spread of the volume of anesthetic solution injected in mlper no of dermatomes blocked. The capacity of lower part of epidural space islarger.

For each pair of segment the following dose is recommended:

For cervical region – 1.5 ml

For thoracic region – 2 ml

For lumbar region - 2.5 ml

The per segment volume of anesthetic solution necessary in sacral and lower lumbar region is greater. For single injection technique the dose should range from 15 – 20 ml of anesthetic solution. For continuous technique the initial dose is 8 – 12 ml and subsequently 5 – 7 ml every hour.

AGE:

In the elderly, the areolar tissue around the intervertebral foramina becomesdense and firm partially sealing the foramina. The permeability of duramaterincreases with increase in age. Aging is associated with reduced beta

adrenergicresponsiveness. Increased levels of analgesia with increase in age is due :

- Progressive sclerosis of intervertebral foramina results in reducedleakage of injected solutions into paravertebral space.

- Increased permeability of duramater.

- Increased compliance of the epidural space.

- Decreased resistance of epidural space.

With aging neural population declines steadily within the spinal cord andperipheral nerves show a linear reduction in conduction velocity especiallymotor nerves. These changes makes older patients more sensitive to localanesthetics with altered motor block profile.

Thermoregulatory response declines with age as shown by decrease in coretemperature consequently rewarming process will occur more slowly in elderpatients.

CONCENTRATION AND DOSE OF LOCAL ANAESTHETIC:

Below concentrations of 1% lignocaine motor block is minimal regardlessof dose, unless injections are repeated at intervals. When dilute solutions inconcentration of 0.125% or 0.625% bupivacaine are injected repeatedly theintensity of sensory and motor blockade increase. This mechanism isparticularly important in obstetric analgesia. Increasing concentration results in reduction in onset time yet produces intense motor blockade.

If more potent analgesia with minimal motor block is required 0.5% bupivacaine, 0.5% ropivacaine, 0.5% levobupivacaine or 1% lignocaine may be chosen. The requirement of profound sensory block and excellent muscle relaxation are best met by 1% lignocaine with epinephrine or 0.75% to 1% ropivacaine. The toxic plasma concentration of lignocaine, bupivacaine, ropivacaine were >5, > 3, >4 ng / ml respectively.

DRUG	CLINICAL USE	CONCENTRATION(%)	DURATION (min)
Lignocaine	Infiltration	0.5 Peripheral	60 - 240
	Peripheral blocks	1	60-200
	Epidural	1.5 – 2	20 – 120
	Spinal	2 – 5	30 – 60
Bupivacaine	Infiltration	0.25	120 – 480
	Epidural	0.5	120 – 300
	Spinal	0.5	60 - 240
Ropivacaine	Infiltration	0.2 – 0.5	120 – 360
	Epidural	0.5 – 1	120 – 360
	Spinal	0.5 – 0.75	90 - 200

Table 4 : Concentration of Local anaesthetics.

POSITION OF THE PATIENT:

Comparison of sitting and lateral position for epidural block reveals no significant differences in cephalad spread. An exception is the obese patient who achieves a lower level of block when seated. The spread of analgesia is more intense in dependent portion when drugs injected in lateral position in both pregnant and non pregnant women. Motor and sensory block onset will be rapid in the dependent portion.

SPEED OF INJECTION:

Rapid injection of local anesthetics into epidural space has no effect on spread of analgesia and has only minimal effect on bulk flow of solution in the space. Rapid injections of large volumes of solution may increase CSF pressure, decreases spinal cord blood flow, increase intracranial pressure and pose a risk of spinal or cerebral complications. Headache is commonly reported if epidural solutions are rapidly injected.

NUMBER & FREQUENCY OF LOCAL ANESTHETIC INJECTIONS:

A single repeat dose (20% of total dose) given approximately 20 minutes after the main dose of local anaesthetic has been said to consolidate blockade within the level of blockade already established. Thus missed segments may be

filled in but the level of blockade may not be extended. A second dose of approximately 50% of initial dosage will maintain the initial segmental level of analgesia if given when the upper level of segmental analgesia has receded 1 to 2 dermatomes. In addition tachyphylaxis increases with the number of injections especially when short acting amides are used.

PHYSIOLOGY OF PAIN

PAIN:

International association for study of pain has defined pain as anunpleasant sensory and emotional experience associated with actual or potentialtissue damage or defined in terms of such damage.

There are two components of pain. Neurophysiologically mediatedsensory component and an emotional component.

There are two types of pain

1. Physiological pain is a transient sensation due to noxious mechanical,thermal, chemical stimulus each with a clearly defined threshold and without causing damage to the nervous system.

2. Pathological pain is an inflammatory response to tissue injury or damage tocentral nervous system with an alteration in perception. Pain followingsurgery is pathological.

There are two major theories of pain.

1. Specificity theory proposed by Von Frey states that pain is due to stimulation of specific end organs.

2. Intensive / Summation / Pattern theory proposed by Gold Scheider states that there are no specific pain receptors and any sensory stimulus if sufficiently severe would produce pain.

ORGANISATION OF PAIN PATHWAYS:

According to the recent theory, pain pathway is organized as follows

RECEPTORS:

Nociceptive receptors are fine, profusely branched, free nerve endingscovered by Schwann cells with little or no myelin. They are present in skin,viscera and other organs.

There are three types of receptors

1. Mechanosensitive nociceptors activated by mechanical stimuli.

2. Mechanothermal nociceptors activated by mechanical and thermal stimuli >43ºC.

3. Polymodal pain receptors respond to mechanical, thermal and chemical stimuli like hydrogen, potassium ions, histamine, serotonin, prostaglandins.

FIRST ORDER NEURONS:

Mechanosensitive and mechanothermal pain receptors transmit impulses through thinly myelinated A δ fibres of 1-5 μ diameter with conduction velocity of 15-30 metres per second. This is responsible for fast pain which is sharply localized. Polymodal pain receptors transmit impulses through unmyelinated C fibres of 0.4-1.1 μ diameter with conduction velocity of 0.5 – 2 meters per second. This is responsible for the poorly localized slow pain. Transmission through both these fibres causes the " Double response of Lewis". The peripheral afferent fibres have their cell body in the dorsal root ganglion and project via the lateral part of the dorsal root called " Tract of Lissauer". They terminate in dorsal horn of spinal cord within 1 to 2 segments of entry. A δ fibres terminate in lamina 1 (marginal cell layer of Waldeyer) and lamina 5 (wide dynamic range of neurons which respond to other modalities also). Unmyelinated C fibres terminate in lamina 2 and 3 (substantia gelatinosa).

SECOND ORDER NEURONS:

They arise from the cell and connect with ventral and lateral horn cells in the same and adjacent spinal segments which subserve both somatic and autonomic reflexes. Around 75% of other sensory neurons project contralaterally after decussating in the anterior commissure 1-3 segments higher than the root of entry and divide into two ascending tracts.

Neospinothalamic / Lateral spinothalamic tract:

It ascends in the anterolateral funiculus of spinal cord to brain stem and thalamus. It contains fast conducting fibres which transmit specific localised pain, identifiable in quality and intensity causing “First Pain “. The fibres are arranged in such a way that fibres from lower part of the body are superficial and from upper part of the body are innermost.

Palaeospinothalamic / Ventral spinothalamic / Spinoreticulothalamic tract:

It is medially placed and contains slowly conducting fibres responsible for “Second Pain” and has connections with brainstem, limbic and subcortical regions.

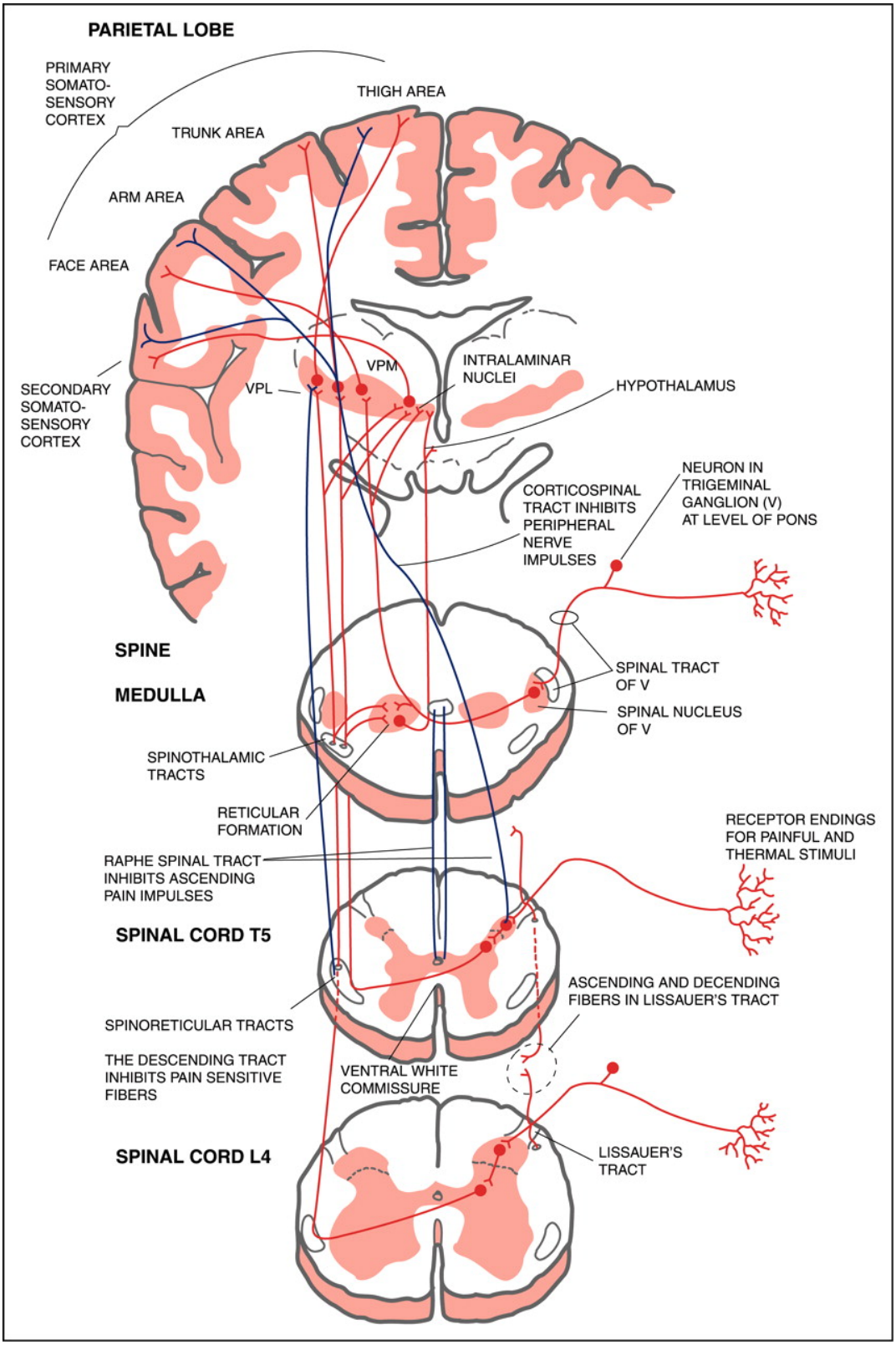
Thalamic terminus:

Most of the fibres of spinothalamic tract terminate in the nucleus ventroposterolateralis which is the major sensory relay nucleus. The other fibres terminate in the posterior group of nuclei, ventrobasal complex and hypothalamic nuclei.

THIRD ORDER NEURONS / THALAMOCORTICAL PROJECTIONS:

Posterior thalamic nuclei project to the post central cortex and upper bankof sylvian fissure and subserve tactile and proprioceptive stimuli withdiscriminative sensory function. Pain afferents received from mesencephalicoffset of anterolateral funiculus project to the amygdaloid nuclei and other areas related to affect the emotion.

Figure 4 : Pain pathway



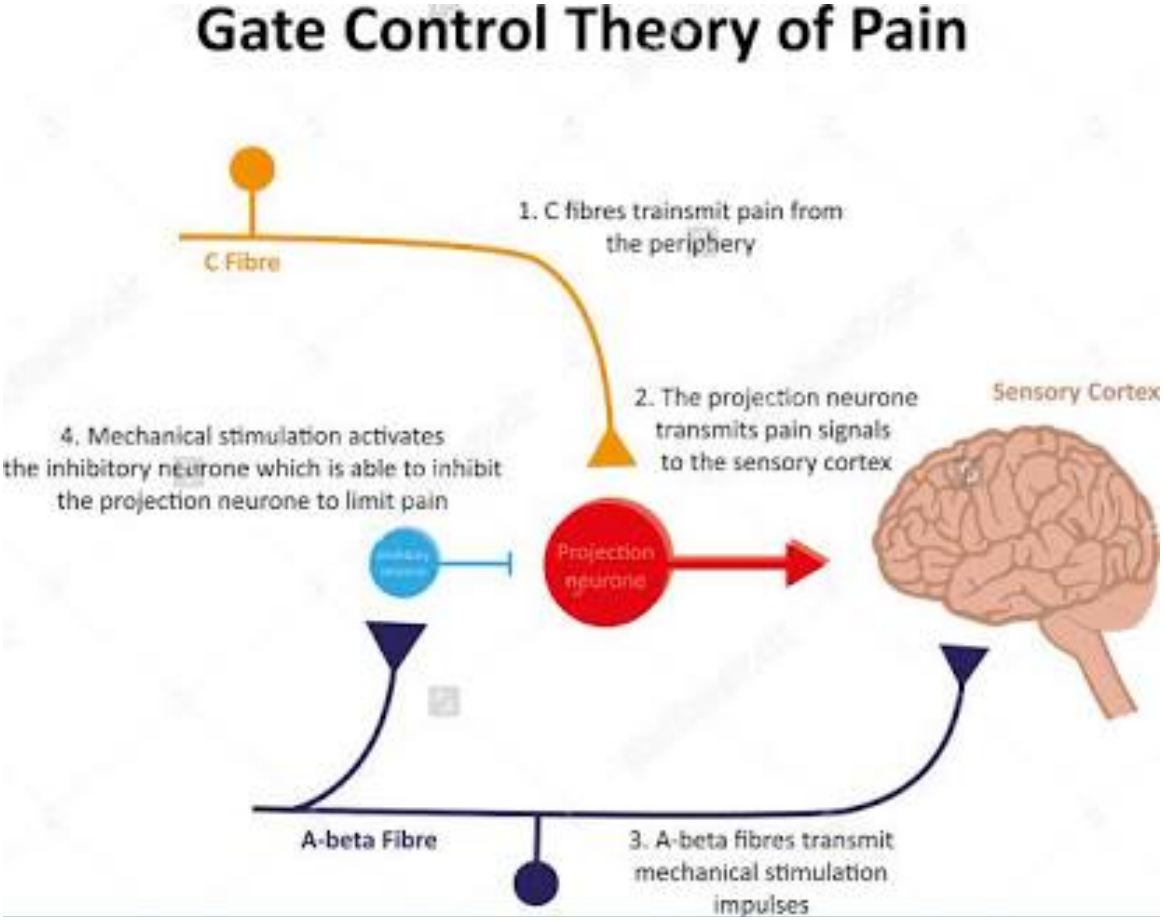
PERCEPTION OF PAIN :

The threshold for the perception of pain is the lowest intensity of stimulus recognized as pain. The conscious awareness or perception of pain occurs at the thalamic level and thalamic pain occurs when the thalamocortical pathway is destroyed. Somatosensory cortex is essential for the accurate localization, appreciation of intensity and other discriminative aspects of pain. Prefrontal cortex subserves the unpleasant affective and emotional reaction to pain.

GATE CONTROL THEORY OF PAIN:

It was propounded by Melzack and Walls in 1965. It states thatmodulation of pain impulses in the dorsal horn can control further synaptictransmission via the spinothalamic tract. It states that stimulation of large afferentfibres excite the I cells (inhibitory cells) in the lamina 2 and 3 of dorsal hornwhich in turn cause pre and post synaptic inhibition of secondary transmissionneurons (T cells) in lamina 5 of dorsal horn and interrupt pain pathway.Conversely stimulation of small pain afferents (C fibres) inhibit the I cellsleaving the T cells in the excitatory state thus facilitating transmission of pain.

Figure 5 : Gate control theory of pain



Endogenous opioids and spinal modulation of pain perception:

Hughes et al described endogenous morphine like substances with analgesic activity called endorphins. There are 5 endorphins, Metenkephalin, Leuenkephalin, Betaendorphin, L endorphin R endorphin.

Metenkephalin and Leuenkephalin:

They are inhibitory neurotransmitters at the primary afferent nociceptivesite. They act through release of substance P.

Dynorphins:

Control nociception at the spinal cord level through activation of kappareceptors. It is present in lamina 1 to 5 of dorsal horn.

L-endorphin and R- endorphins :

Breakdown products of beta endorphins.

PHARMACOLOGY OF STEROIDS

Two classes of steroids:

The corticosteroids, and androgens.

The corticosteroids are classified as glucocorticoid (carbohydrate metabolism–regulating) and mineralocorticoid (electrolyte balance–regulating). The important glucocorticoid and mineralocorticoid in human is cortisol and aldosterone respectively.

GENERAL MECHANISMS FOR CORTICOSTEROID EFFECTS:

Interaction with specific receptor proteins in target tissues upregulatethe expression of corticosteroid-responsive genes, which changes the levels and array of proteins synthesized by the various target tissues.

MOLECULAR MECHANISM OF ANTI INFLAMMATORY EFFECTS

OF GLUCOCORTICOIDS:

Glucocorticosteroids are potent anti-inflammatory agents. This anti-inflammatory effect may be produced via a variety of mechanisms. A group of

structurally related, calcium-dependent phospholipid-binding proteins,annexins, which were formerly known as lipocortins or calpactins, had beenshown to be inducible by glucocorticoids. Annexin I has been reported to inhibitsPLA_2 activity in vitro. These observations led to the hypothesis that theinhibition of $sPLA_2$ by annexins is the mechanism of the anti-inflammatoryaction of glucocorticoids[6].

The prolongation of analgesic duration of perineural administration ofdexamethasone may be secondary to local action on nociceptive- C fibresmediated via glucocorticoid receptors and upregulation of function of potassiumchannels in excitable cells

CARBOHYDRATE AND PROTEIN METABOLISM :

Stimulation of glucose synthesis from amino acids and glycerol andstorage as glycogen in liver. There is diminished glucose utilisation withincreased protein breakdown in the periphery resulting in increased bloodglucose. Glycemic control can be worsen in patients taking corticosteroids.

LIPID METABOLISM:

Redistribution of body fat results in increased fat accumulation insupraclavicular area, nape of the neck, face along with a loss of fat in theextremities. An increase in free fatty acid level occurs due to augmentation of lipolytic effects of growth hormone and adrenergic agonists.

ELECTROLYTE AND WATER BALANCE :

In patients with glucocorticoid deficiency there is increased secretion ofvasopressin, which stimulates water reabsorption in the kidney. Steroidsinterfere with Ca^{2+} uptake in the gut and increase Ca^{2+} excretion by the kidney leading to decreased total body Ca^{2+} stores. The most striking cardiovascular effects of corticosteroids result from mineralocorticoid-induced changes in renal

Na^+ excretion, leading to increased sodium and water retention in primaryaldosteronism there is enhanced response to vasoactive drugs.

SKELETAL MUSCLE:

In Addison‟s disease, weakness , fatigue and diminished work capacityare the prominent symptoms. In primary aldosteronism weakness and fatigueoccurs due to steroid myopathy.

CENTRAL NERVOUS SYSTEM:

Patients with adrenal insufficiency exhibit apathy, depression andirritability. Replacement therapy will alleviate such symptoms. Treatment withglucocorticoids may result in behavioural changes such as mania, insomnia and restlessness and these abnormalities disappear with cessation of therapy.

BLOOD AND FORMED ELEMENTS:

Corticosteroids exert minimal effects on erythrocytes and haemoglobin asevident by polycythemia in cushing syndrome, an normocytic normochromic anaemia in addisons disease. A single dose of hydrocortisone can decrease the circulating levels of these cells within 4-6 hours . This persists for 24 hours and it results from redistribution of cells away from periphery.

PHARMACOLOGY OF DEXAMETHASONE:

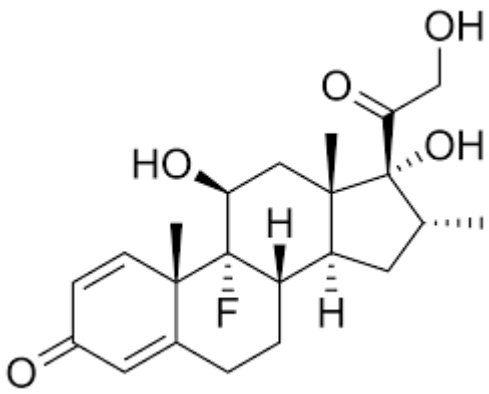


Figure 6 : Structural formula of dexamethasone

PHARMCOKINETICS OF DEXAMETHASONE

Bioavailability	→	80 – 90 %
Protein binding	→	70 %
Metabolism	→	hepatic
Half life	→	36 – 54 hours
Excretion	→	renal
Molecular weight	→	392.4 g / mol

Dexamethasone is a high potency, long acting glucocorticoid with littlemineralocorticoid effect. It has been used intravenously for prophylaxis ofpostoperative nausea. Single doses of epidural dexamethasone and

otherglucocorticoids have been reported to improve analgesia after various surgeries.Acute noxious stimulation of peripheral tissues leads to sensitization ofdorsal horn neurons of the spinal cord by the release of excitatory amino acidssuch as glutamate and aspartate. These amino acids activate N-methyl-Daspartatereceptors resulting in calcium ion influx. As a result, increasedintracellular calcium activates phospholipase A_2 which converts membranephospholipids to arachidonic acid. Simultaneously, there is up-regulation of theexpression of cyclo-oxygenase 2 in the spinal cord, leading to prostaglandinE_2 synthesis, which results in a hyperalgesia.

MECHANISM OF ACTION OF EPIDURAL STEROIDS:

Dexamethasone and other steroids act by suppression of transmission in thin unmyelinated C fibres while not affecting myelinated A_β fibres. It exerts these action through direct membrane stabilising effect and indirectly through mediators.These direct and indirect actions lead to decrease in intraneuronal edema and venous congestion thereby reducing ischemia and improving pain.

PHARMACOLOGY OF BUPIVACAINE

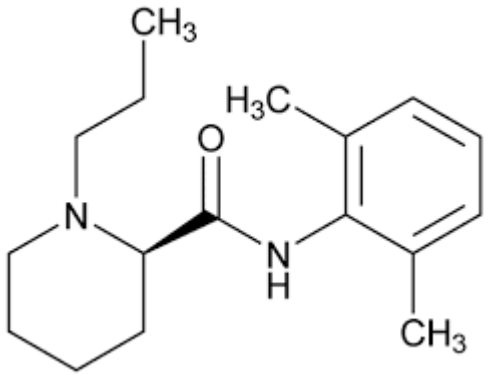


Figure 7 : Structural formula of bupivacaine

It is an amide local anaesthetic first synthesized in Sweden by Ekenstamand his colleagues in 1957 and used clinically L.J.Telivuo in 1963. Itsmolecular weight is 288. (1-butyl- N-(2,6, dimethyl phenyl piperidine-2-carboxamide)Prepared as a clear solution of 0.25%, 0.5% solution of bupivacainehydrochloride. The hyperbaric solution used for subarachnoid block contains 80mg / ml of glucose.

PHARMACOKINETICS:

At pH 7.4 only 15% exist in non ionised form. Absorption depends on thesite of injection, dosage and use of epinephrine.

pKa	→	8.1
Protein binding	→	95 %
Lipid solubility	→	28 %
Volume of distribution	→	73 litre
Clearance of drug from plasma	→	0.471 litre / minute
Elimination half life	→	210 minute
Onset time	→	5 – 7 minute

MECHANISM OF ACTION:

Local anesthetics such as bupivacaine block the generation andconduction of nerve impulses, presumably by increasing the threshold forelectrical excitation in the nerve, by slowing the prolongation of the nerveimpulse and reducing the rate of rise of the action potential. The progression ofanesthesia is related to the diameter, myelination and conduction velocity ofaffected nerve fibres. The analgesic effects are thought to be due to its bindingto the prostaglandin E2 receptors.

METABOLISM:

The possible pathway for metabolism of bupivacaine include aromatichydroxylation, N-dealkylation, amide hydrolysis and conjugation. Only the Ndealkylatedmetabolite N-desbutyl bupivacaine has been measured in the bloodor urine.5 % of the dose is excreted in the urine as pipcolloxylidine.16 % isexcreted unchanged.

ROUTES OF ADMINISTRATION:

May be administered by infiltration, intrathecally or epidurally and forperipheral nerve blocks. The total dose of bupivacaine should not exceed 2 – 3mg / kg (with or without epinephrine).

SYSTEMIC TOXICITY:

CARDIOVASCULAR SYSTEM:

Bupivacaine is markedly cardiotoxic. It binds to specific myocardialproteins. In toxic concentrations the drug decreases the peripheral vascularresistance and myocardial contractility producing hypotension andcardiovascular collapse.

Cardiotoxic plasma concentration is 8 – 10 μg / ml.20 % intra lipid can be given for bupivacaine toxicity. The dose is 1.5 ml /kg as initial bolus can be repeated 1 to 2 times for persistent asystole. Infusioncan be started at dose of 0.25 ml / kg / min for 30 – 60 min.

CENTRAL NERVOUS SYSTEM:

During accidental overdosage or direct vascular injections the clinical signsare numbness of tongue, light headedness, visual and auditory disturbances,muscle twitching, tremors. The signs may progress to generalised convulsionsof the tonic clonic nature. The typical plasma concentrations of bupivacaine associated with seizures is 4.5 – 5.5 μg / ml.

CHAPTER 2

AIM AND OBJECTIVES OF STUDY

To compare the efficacy of epidural bupivacaine with added dexamethasone in postoperative analgesia following gynaecology surgeries.

Following points will be considered for the comparison

1) Time for first anaelgesic request

2) Duration of surgery

3) Duration of sensory analgesia using VISUAL ANALOG SCORE

4) Hemodynamic parameters

5) Adverse effects if any

CHAPTER 3

REVIEW OF LITERATURE

1. Khafagy et al(23) studied the effect of epidural dexamethasone in patientsundergoing lower abdominal surgeries. In that study group I patientsreceived epidural 10cc of 0.25% bupivacaine and fentanyl 50µgepidurally and group II patients received 10cc of 0.25 bupivacaine withdexamethasone 4 mg. He found that addition of epidural dexamethasoneimproved postoperative pain relief and decreased analgesic requirement.

2. Thomas et al (16) evaluated the efficacy of epidural dexamethasone inreducing post operative analgesic requirements following laprascopiccholecystetcomy. In that study group I patients received IVdexamethasone 5 mg and 8 cc of 0.25% bupivacaine epidurally. Group IIpatients received IV normal saline 2 cc with 8 cc of 0.25% bupivacaineand dexamethasone epidurally. He concluded in the study that group IIpatients receiving epidural dexamethasone 5 mg had effective postoperative pain relief and less systemic opioid requirements followinglaprascopic cholecystectomy.

3. Younyi jo et al[13] compared outcomes of epidural ropivacaine 0.25% andepidural ropivacaine 0.25% with dexamethasone 5 mg in patientsundergoing radical subtotal gastrectomy.He compared VAS scores andrescue analgesic requirements among these group of patients andconcluded that VAS scores and rescue analgesic requirements were lessin dexamethasone treated group.

4. Farshad M Ahadian et al[14] in his stuy compared the efficacy of threedifferent doses (4 mg, 8 mg, 12mg) of transforaminal epiduraldexamethasone in relieving radicular pain.He measured the outcomes interms of VAS scores and subject satisfaction scale.It showed thatimprovement in radicular pain with no difference in efficacy of differentdoses of dexamethasone.

5. Wang et al[15] compared the effect of epidural dexamethasone in relievingpost epidural backache in patients undergoing hemorrhoidectomy. In thestudy group I patients received 25 cc of 2% lignocaine with 1 cc normalsaline. Group II patients received 25 cc of 2% lignocaine with 5 mgdexamethasone. He found decreased severity of backache in group IIpatients.

6. Jehan et al (17) studied the effect of preoperative epidural dexamethasone and magnesium sulphate in patients undergoing abdominal surgeries. Inthe study, group I patients received 12cc of 0.5% bupivacaine withmorphine 2 mg and magnesium sulphate 50mg epidurally. Group IIpatients received 12 cc of 0.5%bupivacaine with morphine 2 mg anddexamethasone 6 mg epidurally. He concluded that with coadministration of magnesium sulphate 50 mg or dexamethasone 6 mg as asingle dose in preoperative period was associated with less postoperativenarcotic consumption and VAS scores.

7. W.Neill et al (18) studied the effects of epidural methyl prednisolone 40mg and morphine 5mg along with control group having normal saline inpatients undergoing surgery for spinal stenosis. He found that thepostoperative analgesic requirement was less in group of patientsreceiving epidural morphine or methylprednisolone or combination ofboth.

8. Atsuhiro Kikuchi et al19) studied the effect of intrathecal and epiduralmethyl prednisolone in relieving the severity of pain due to post herpeticneuralgia. Group I patients received 40 mg of methylprednisoloneepidurally and group II patients received 40 mg of methylprednisoloneintrathecally. He concluded that pain severity was less

in patientsreceiving intrathecal methylprednisolone due to decreased inflammatoryreaction in CSF.

9. SaeidAbrishamkar et al[20] studied the effect of epiduralmethylprednisolone 40 mg and local anesthetic (1 cc of 0.5%bupivacaine) impregnated in adipose tissue in relieving low back painand radicular pain in lumbar disc surgery. He found that combination ofmethylprednisolone and local anaesthetic increased the duration of painfree interval.

10. Park CH[21] compared the effects of transforaminal injection ofdexamethasone 7.5 mg and triamcinolone 40 mg in patients with lumbardisc herniation and found that triamcinolone is more effective inrelieving lumbar radiculopathy than dexamethasone.

CHAPTER 3

MATERIALS AND METHODS

After getting Ethical committee approval from Government Stanley MedicalCollege Hospital. Chennai 1, we conducted the study in Government RSRM lying hospital in 60adult female patients aged between 25 – 70 years belonging to ASA Physicalstatus I and II undergoing elective gynaecological procedures, epidural anaesthesia afterobtaining written informed consent.

STUDY DESIGN:

Double blinded randomized prospective study.

Patients were randomly allocated into one of the two groups (30 patientsper group) by lotting method.

METHOD OF BLINDING:

Patients and the person performing the epidural technique was unawareof the epidural drug composition. The drug solution was prepared by ananaesthesiologist assistant in the operating room and was labelledaccordingly.

PATIENT SELECTION:

All the 60 patients were evaluated clinically, biochemically and assessed for elective gynaecological procedures under epidural anesthesia considering the inclusion and exclusion criteria. Then the patients were randomised into two groups.

OBSERVATION PERIOD:

For 24 hours postoperatively.

INCLUSION CRITERIA:

- Adult male patients aged 18-70 years
- ASA physical status I & II
- For elective gynaecological surgery

EXCLUSION CRITERIA :

- Patient unwilling for the procedure
- Bleeding disorders
- Allergy to amide type local anaesthetics
- Diabetes mellitus

- Infection at the injection site
- Those received corticosteroids or immune suppressive drugs in the last 6 months
- Those with contraindications to steroids
- Patients on anticoagulants
- Pregnancy or breast feeding females
- Severe obesity(BMI >35Kg/$m^{2)}$
- Psychiatric disorders
- Patients with past history of musculoskeletal disorders
- Spine or chest wall deformity
- Previous history of thoracic surgeries

PATIENT GROUPS:

60 patients enrolled in the study were randomly allocated into two groups.

- Group BS: Patients receiving 10 cc of 0.25 % bupivacaine plus normal saline 2 cc epidurally.
- Group BD: Patients receiving 10 cc of 0.25 % bupivacaine plus 8 mgpreservative free dexamethasone epidurally.

All patients received a total volume of 15 ml of study drug including 3 ml of test dose plus 1 ml of adjuvant. The level of blockade was then noted.

MATERIALS USED:

- 16 Gauge Tuohy needle
- 18 Gauge epidural catheter
- Loss of resistance syringe
- 10 ml syringe
- Local anesthetic solution (3 ml of 1.5%lignocaine with epinephrine 1 in 2,00,000 dilution) for test dose.
- 0.25% bupivacaine
- Inj. Dexamethasone sodium phosphate (preservative free)
- 22 G needle for pin prick test

PARAMETERS TO BE OBSERVED:

1. Demographic parameters
2. Baseline parameters
3. Duration of surgery
4. VAS score
5. Duration of analgesia
6. Incidence of side effects like nausea,vomiting,hypotension, bradycardia

CONDUCT OF STUDY:

In the pre anesthetic visit, study plan was explained in detail to all thepatients. Written informed consent obtained after explaining the study intheir ownlangage.After getting informed consent, patient was prepared for the surgery withfasting period of 8 hours. Antacid prophylaxis was given with inj. Ranitidine50 mg IV 2 hours before surgery. Baseline vital parameters were recorded inthe patient waiting room.

CONDUCT OF EPIDURAL BLOCK:

In the operating room patient was connected to five lead ECG, NonInvasive Blood Pressure, Pulse Oximeter and baseline parameters wererecorded. An intravenous line was established with 18 gauge venflon andpreloaded with 15 ml / kg of ringer lactate.Under strict aseptic precautions with the patient in right lateral positionlocal anaesthetic infiltration was given with 2 % lignocaine. Epidural space wasidentified at L2 – L3 space through 16 gauge Tuohy needle by loss of resistancetechnique. An 18 gauge epidural catheter was inserted in L2 – L3 space and 5cm of catheter kept inside epidural space. Test dose was given with 3 ml of 0.25% bupivacaine with epinephrine 1:2,00,000 dilution via catheter before it isfixed to rule out intravascular or intrathecal placement.

After confirming the epidural placement of the catheter, 12 ml of blindedstudy solution was given and level of blockade was noted at 5 min .In both the groups anaesthesia was induced with thiopentone 5mg/kg and fentanyl 2mic/kg ,intubated with atracurium0.5 mg/kg and anaesthesia was maintained withatracurium 0.1mg/kg and volatile Nitrous oxygen mixture.Intra operative analgesia was maintained with intermittent doses of fentanyl.after surgery got over ,patient reversed with residual neuromuscular blockade with neostigmine 50mic/kg,then the patient was shifted to PACU.(post anaesthesia care unit).

ASSESSMENT OF PAIN SCORE:

In the PACU, pain score was observed for every hour for first 6 hours,then every 6 hours for 24 hours on a 10cm Visual analoguescale („no pain‟ at 0 cm end and „worst pain ever‟ at 10cm end) and foroccurrence of side effects like nausea, vomiting,pruritus respiratorydepression, sedation and changes in hemodynamic variables.

ONSET AND DURATION OF ANALGESIA:

The time since injection of drug into epidural space to the time requiredto obtain sensory blockade upto T8 (loss of pin prick to 22 gauge needle) was noted as onset of analgesia. The time between the onset of analgesia and return to baseline VAS of 5 was noted as the duration of analgesia .

RESCUE ANALGESIA IN THE POSTOPERATIVE PERIOD:

When the VAS score was more than 5 or when the patients complained ofpain, Since the study was concluded Inj. Diclofenac 50mg was givenintramuscularly and epidural catheter was removed.The patients were followed for a period of 24 hours in PACU for anyoccurrence of nausea, vomiting, sedation, pruritus, respiratory depression(RR<10/min), and parameters like duration of analgesia, hemodynamicvariables etc were noted.

Statistical analysis was done on collected data. Analysis of variances(ANOVA) was used for comparison of mean values between more than twogroups. Posthoc test was used to find any significance between the individualgroups.

VISUAL ANALOGUE SCALE:

" Please make a mark on this line that describes how much pain you are having"

No pain 0___1___2___3___4___5___6___7___8___9___10 Worst pain

Pain

0 - No nausea/vomiting

1 - Nausea

2 - Vomiting

0 - No pruritus

1 - pruritus

Bradycardia HR < 50 / min

0 - No bradycardia

1 - Presence of bradycardia

Respiratory depression

RR < 10 / minute

0 – No respiratory depression

1 – Presence of respiratorydepression

Desaturation Spo2 < 95 %

0 – No desaturation

1 – Presence of desaturation

Hypotension

Systolic blood pressure < 80mm hg

Mean arterial pressure < 60mm hg

VISUAL ANALOGUE SCALE

A Visual Analogue Scale (VAS) is a measurement instrument that triesto measure a characteristic or attitude that is believed to range across acontinuum of values and cannot easily be directly measured (example –the amount of pain that a patient feels ranges across a continuum from none to anextreme amount of pain).From the patient's perspective this spectrum appearscontinuous and their pain does not take discrete jumps, as a categorization ofnone, mild, moderate and severe would suggest. It was to capture this idea ofan underlying continuum that the VAS was devised.Operationally a VAS isusually a horizontal line, 10cm / 100 mm in length. The patient marks on theline the point that they feel represents their perception of their current state.The VAS score is determined by measuring in centimetres / millimetres fromthe left hand end of the line to the point that the patient marks.

CHAPTER 5

OBSERVATION AND RESULTS

STATISTICAL TOOLS

The information gathered from the selected cases were noted in the master chart.The collected data were analysed with IBM.SPSS statistics software 23.0 Version.To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups the Unpaired sample t-test was used for normal data and Mann-Whitney U test for skewed data.To find the significance in categorical data Chi-Square test was used. In all the above statistical tools the probability value .05 is considered as significant level.

COMPARISON OF AGE:

Figure 8: Comparison of age

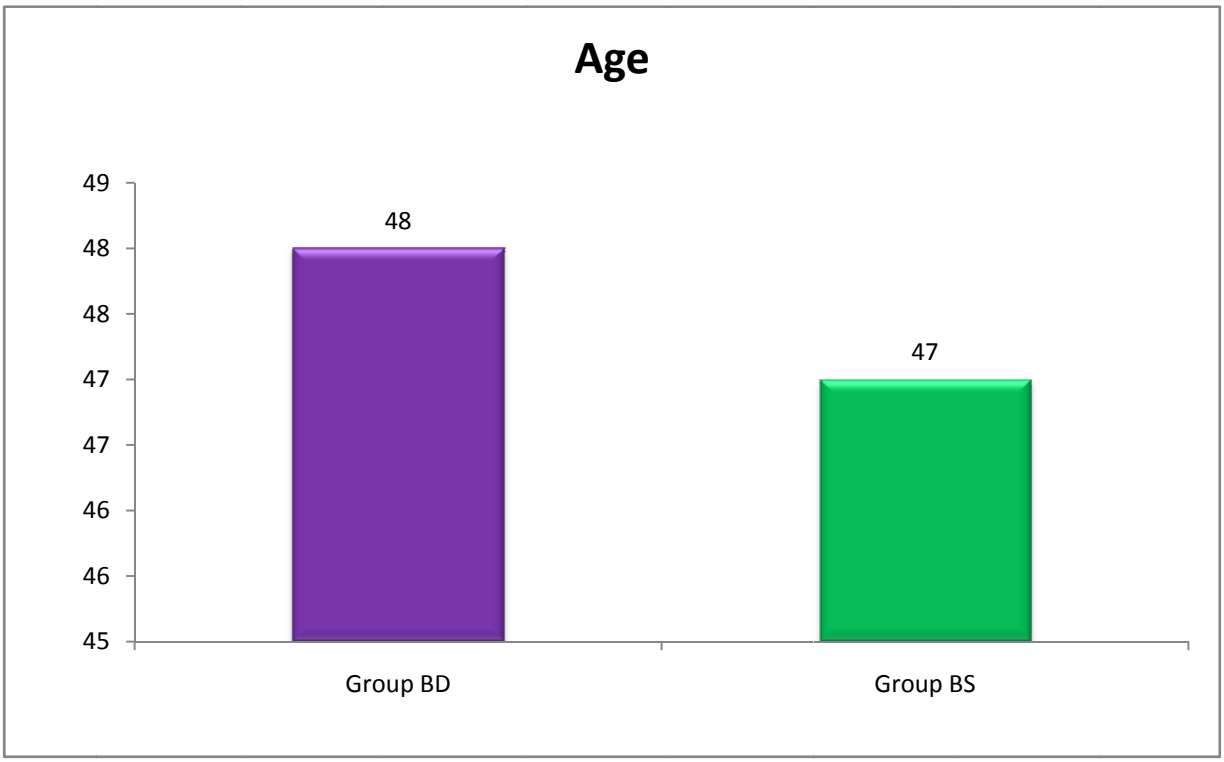


Table.5 : Comparison of age

GROUPS		N	Mean	Std. Deviation	p value
AGE	Group BD	30	48	9.576	0.863
	Group BS	30	47	9.774	

The mean age of Group BD and BS were 48 and 47 respectively. There was no statistically significant difference with the p value of 0.863 between the mean age of two groups which shows these two groups were similar with respect to age.

COMPARISON OF HEIGHT:

Figure. 9 : Comparison of Height

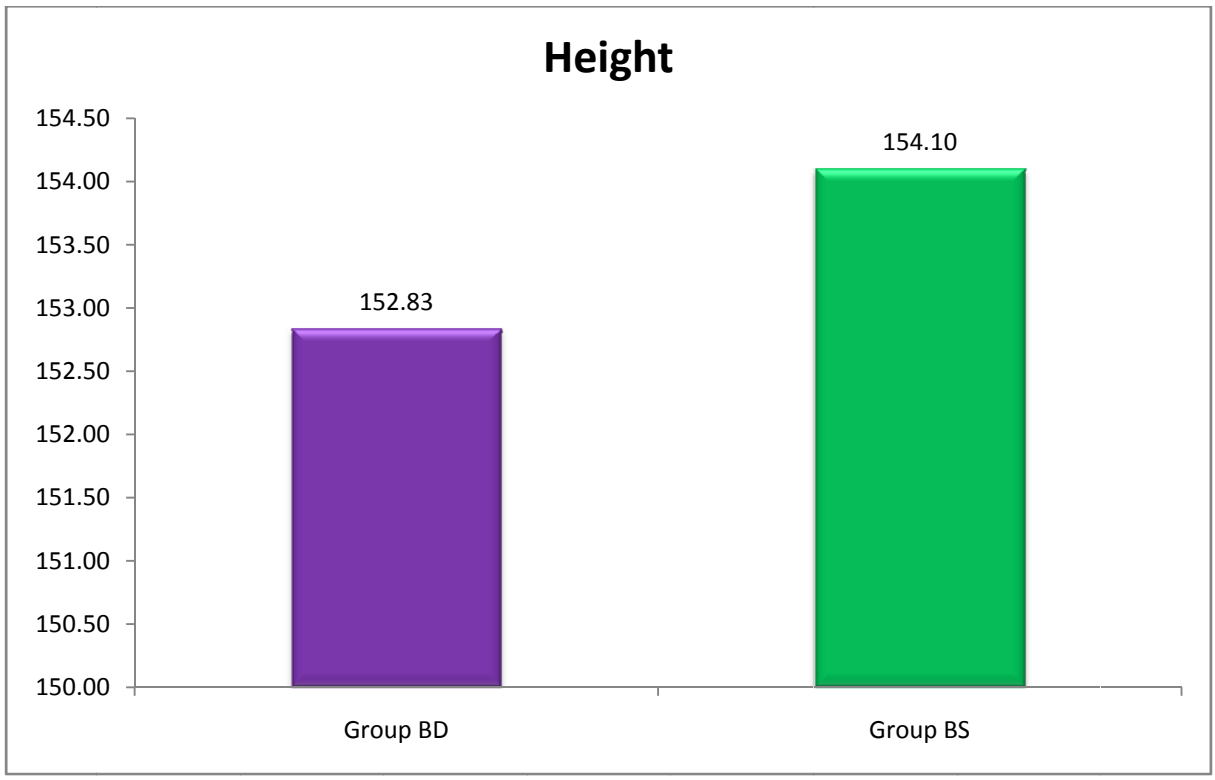


Table 6 : Comparison of Height

GROUPS		N	Mean	Std. Deviation	p value
HEIGHT	Group BD	30	152.83	3.966	0.254
	Group BS	30	154.10	4.536	

The mean height of group BD and BS were 152.83cm and 154.10 cm respectively. There is no statistically significant difference(p value = 0.254) between these groups which shows that they are comparable with respect to height.

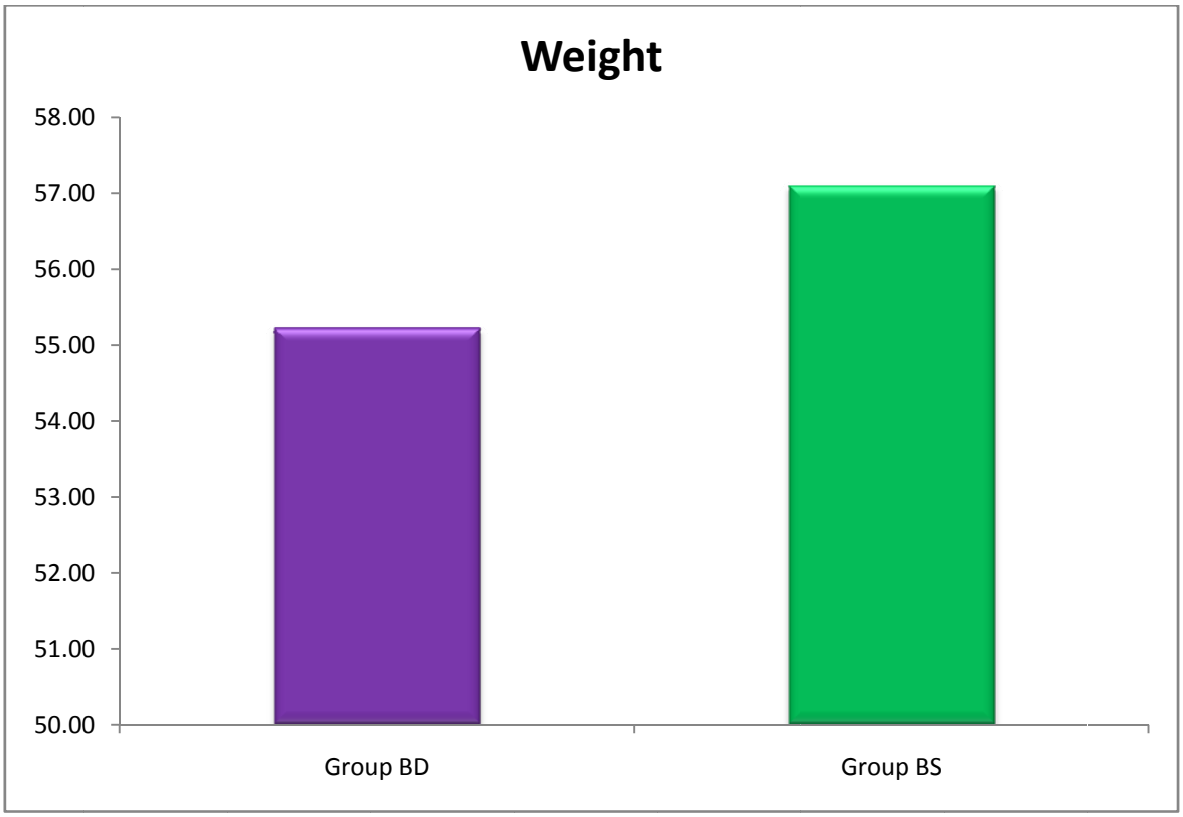
COMPARISON OF WEIGHT :

Table.7 : Comparison of weight

GROUPS		N	Mean	Std. Deviation	P value
WEIGHT	Group BD	30	55.23	4.360	0.119
	Group BS	30	57.10	4.759	

The mean weight of the Group BD and BS were 55.23 kg and 57.10 respectively. There is no statistically significant difference (p value = 0.254) between these groups which shows that they are comparable with respect to height.

Figure 10 : Comparison of weight



COMPARISON OF ASA :

Figure 11: Comparison of ASA

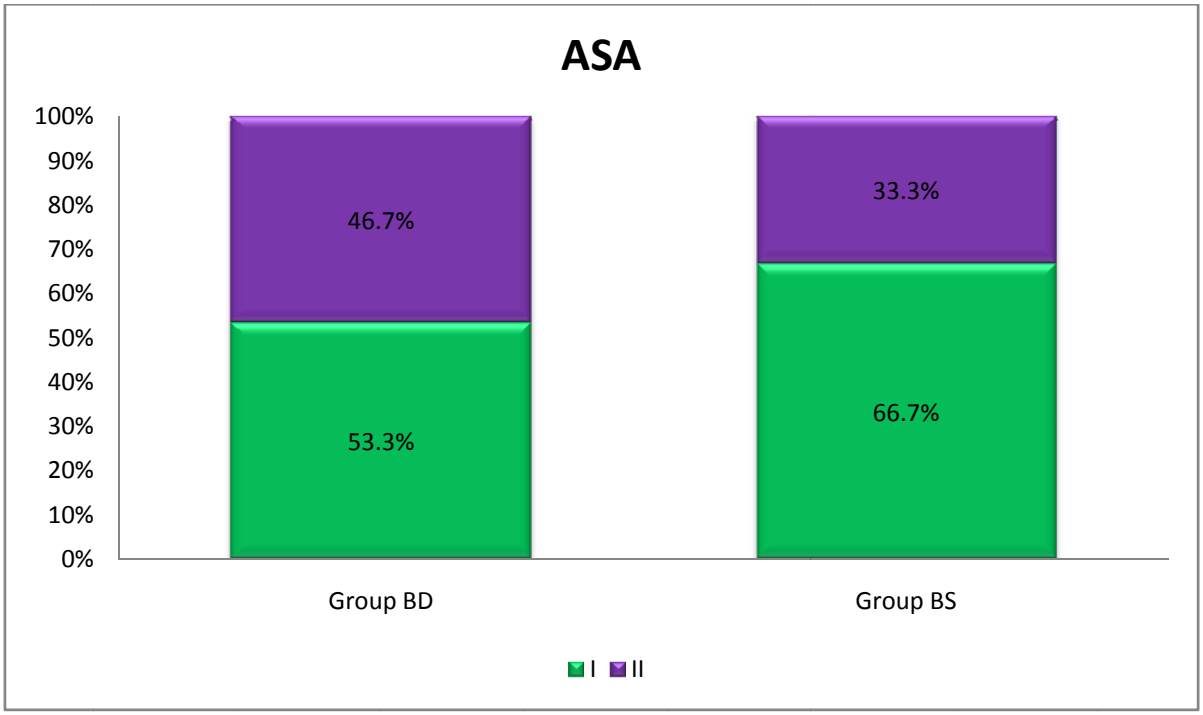


Table 8 : Comparison of ASA

		GROUPS		Total
		BD	**BS**	
ASA	I	16	20	36
		53.3%	66.7%	60.0%
	II	14	10	24
		46.7%	33.3%	40.0%
Total		30	30	60
		100.0%	100.0%	100.0%
p value		0.292		

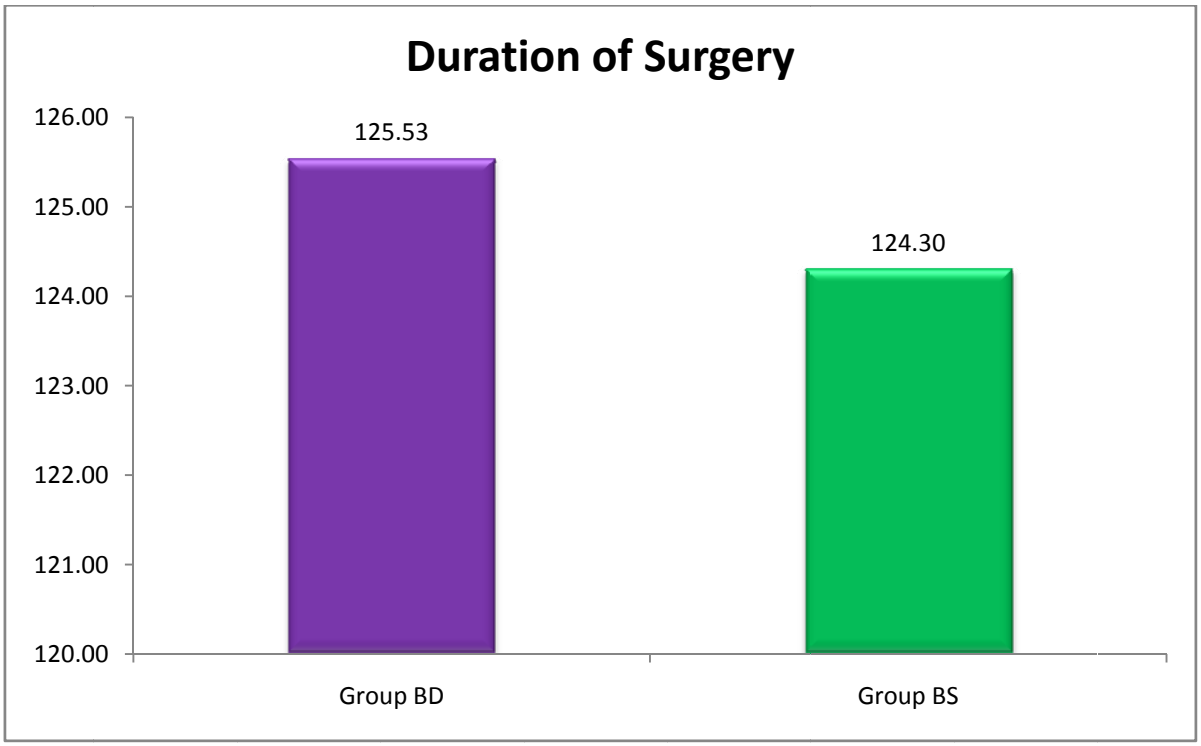
DURATION OF SURGERY:

Table 9: Duration of surgery

GROUPS		N	Mean	Std. Deviation	p value
DURATION OF SURGERY	Group BD	30	125.53	14.450	0.725
	Group BS	30	124.30	12.477	

The mean duration of surgery in Group BD and BS were 125.53 minutes and 124.30 minutes respectively. p value is 0.725 which is not statistically significant and hence they are comparable with each other.

Figure 12 : Duration of surgery



TIME FOR FIRST ANALGESIC REQUEST :

Figure 13: Time for first analgesic request

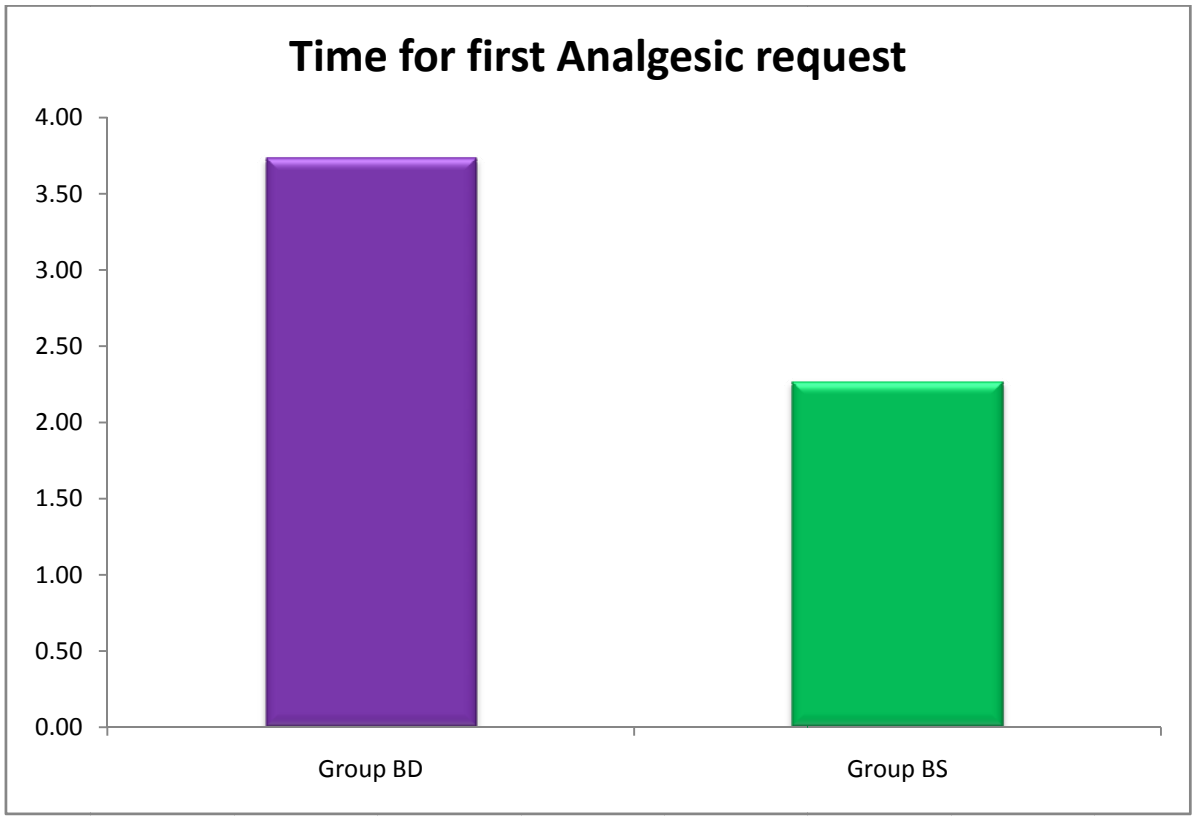


Table 10 : Time for First Analgesic request

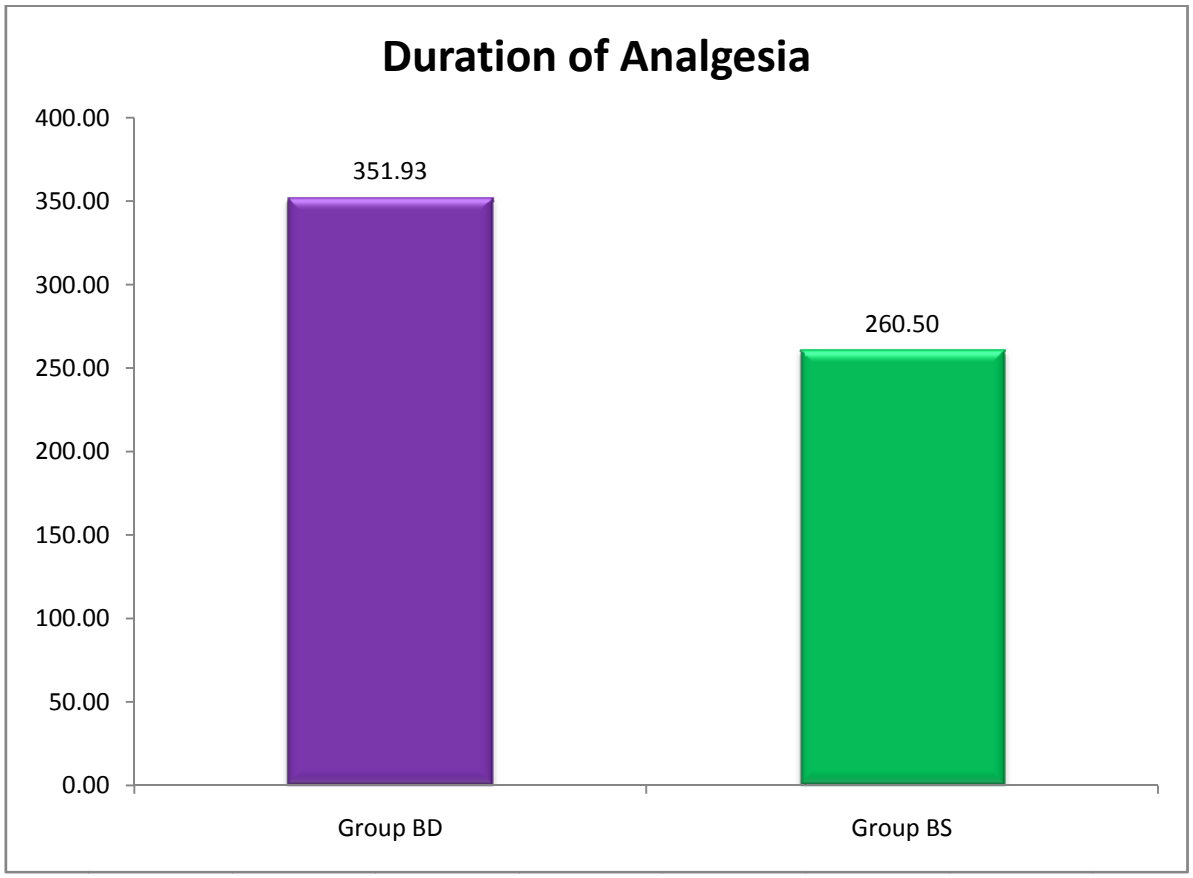
GROUPS		N	Mean	Std. Deviation
TIME FOR FIRST ANALGESIC REQUEST	**Group BD**	**30**	**3.53**	**1.484**
	Group BS	**30**	**2.27**	**.944**
p value	0.000			

On comparing the time for first analgesic request postoperatively between two groups, the patients in Group BS requested first analgesic dose earlier than the patients from group BD.

Table 11 : Duration of Analgesia

GROUPS		N	Mean	Std. Deviation	P Value
Duration of Analgesia	Group BD	30	351.93	92.018	0.000
	Group BS	30	260.50	58.728	

Figure 14 : Duration of Analgesia



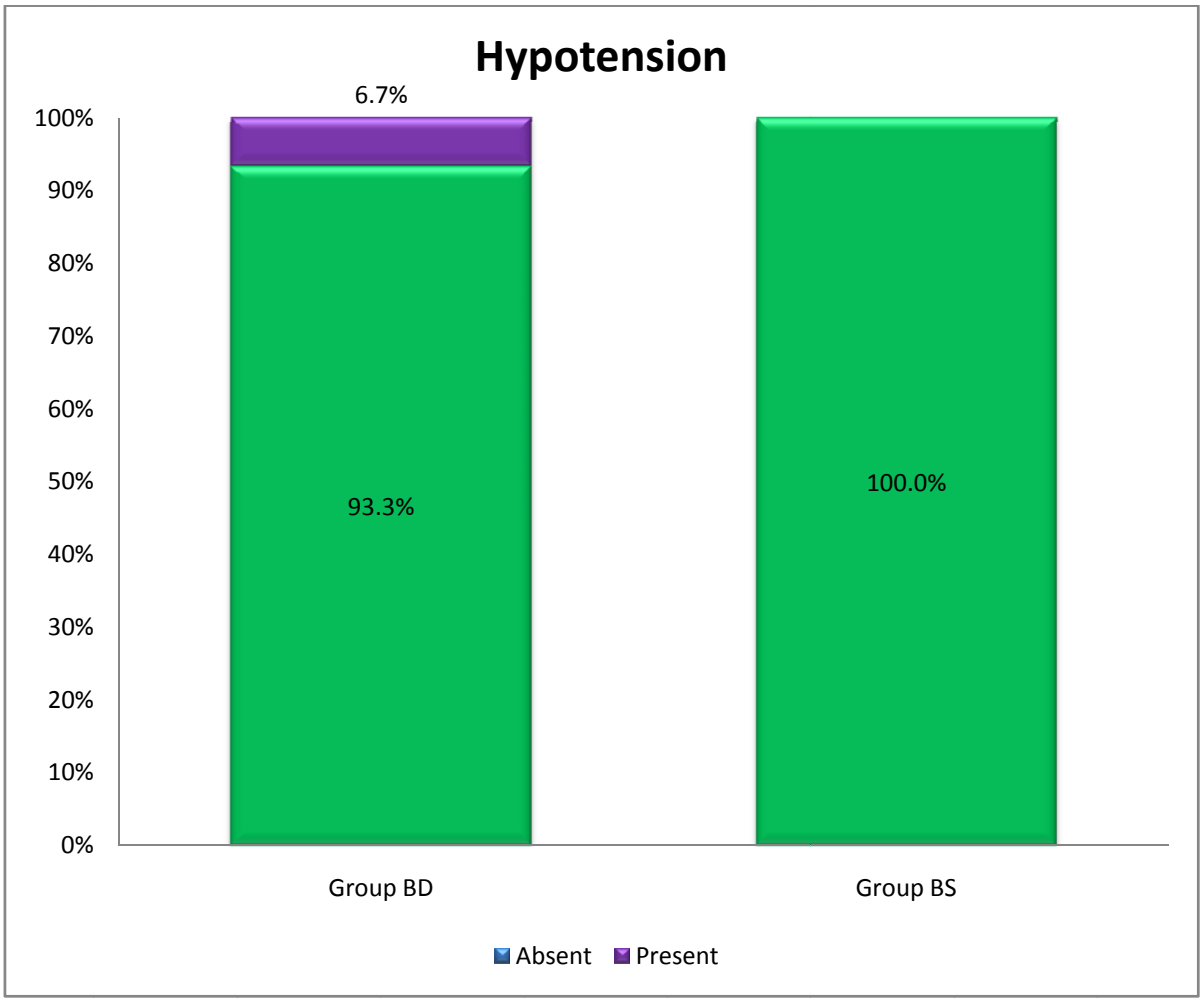
On comparing the mean duration of analgesia among the two groups, patients in Group BD receiving dexamethasone (351.93 mins) had a prolonged duration of analgesia than group BS(260.50 mins). The p value is statistically significant (0.000)

HYPOTENSION :

Table 12 : Incidence of Hypotension

			GROUPS		Total
			BD	BS	
HYPOTENSION	NIL	Count	28	30	58
		% within GROUPS	93.3%	100.0%	96.7%
	YES	Count	2	0	2
		% within GROUPS	6.7%	0.0%	3.3%
p value	0.150				

Figure 15 : Incidence of Hypotension



The incidence of hypotension in Group BD is 6.7 %. There is nil incidence of hypotension in Group BS. P value is 0.150 which is not statistically significant.

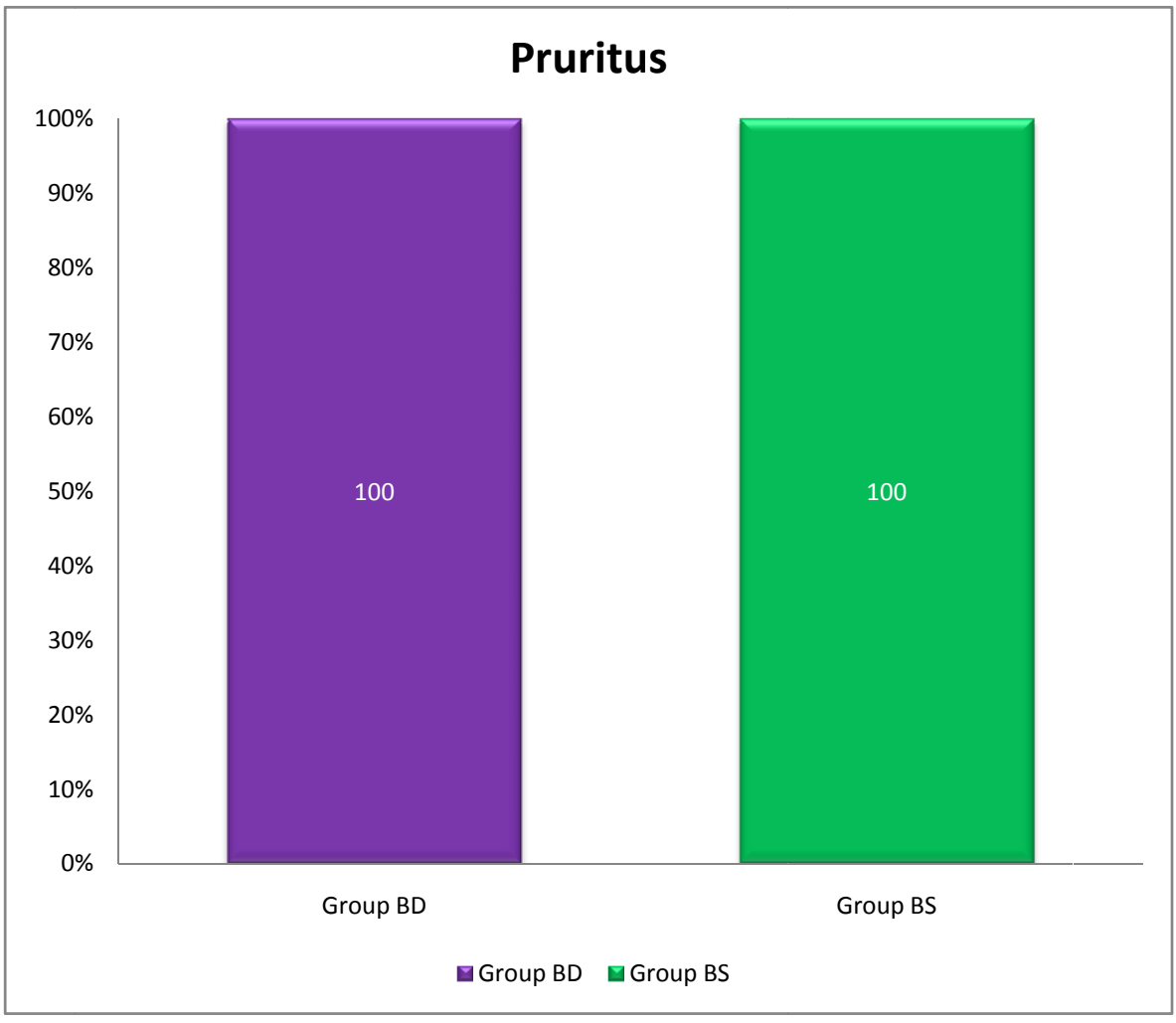
PRURITUS:

Table 13 : Incidence of Pruritus

			GROUPS		Total
			BD	BS	
PRURITUS	NIL	Count	30	30	30
		% within GROUPS	100.0%	100%	100%
	YES	Count	0	0	0
		% within GROUPS	0.0%	0.0%	0.0%
Total		Count	30	30	60
		% within GROUPS	100.0%	100.0%	100.0%

There is nil incidence of pruritus in both the Groups.

Figure 16 : Incidence of Pruritus



NAUSEA :

Figure 17 : Incidence of Nausea

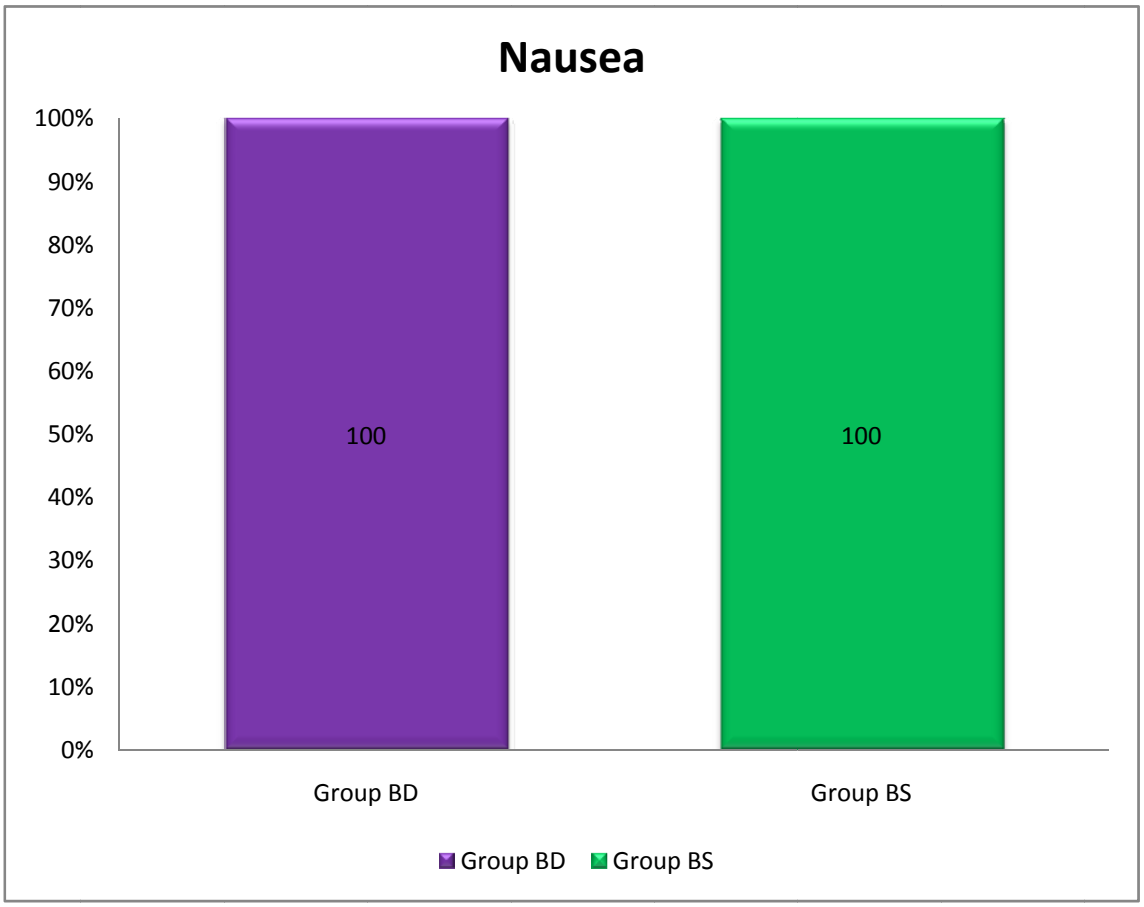


Table 14 : Incidence of nausea

			GROUPS		Total
			BD	BS	
PRURITUS	NIL	Count	30	30	30
		% within GROUPS	100.0%	100%	100%
	YES	Count	0	0	0
		% within GROUPS	0.0%	0.0%	0.0%
Total		Count	30	30	60
		% within GROUPS	100.0%	100.0%	100.0%

No patients had complaints of nausea in both the groups.

CHAPTER 6

DISCUSSION

Epidural anaesthesia is superior to spinal as the desired block levels can be achieved without significant hemodynamic disturbances and topup doses of anaesthetics and analgesics can be given.In modern anaesthetic practiseepidural anaesthesia is being widely used,especially in patients undergoing procedures involving lower abdominal surgeries.to fulfil this demand there is a need for local anesthetic with desired properties like longer duration of sensory blockade and shorter duration of motor blockade.

Traditionaly epidural bupivacaine was used for postoperative analgesia.the epidural bupivacaine 0.5%causes motor,sensory and sympathetic blockade ,0.25% causes sensory and autonomic blockade.0.125 causes autonomic blockade only. Epidural administration of various anaelgesics gain increasing popularity following the discovery of opite receptors in the spinal cord capable of producing potent analgesia by taksh and rudy in 1976.

A Study entitled a comparative study between epidural “A Comparative Study Of epidural administration of 0.25% bupivacaine versus 0.25% bupivacaine+8mg dexamethasone on postoperative analgesia following gynaecology surgery”

After obtaining informed consent from 60 patients ASA1and ASA2

Posted for various gynaecological surgeries,were grouped randomly into either Bupivacaine with saline(BS) and bupivacaine with dexamethasone(BD) .Epidural space was identified with loss of resistance technique in T12 L1 space in right lateral decubitus position with skin to space distance varying from 4 to 5 cm.catheter tip placed 9-11 cms inside .Epidural activation is done before skin incision.Bupivacaine 0.25% plus study drug either normal saline or 8mg dexamethasone given as single shot 10ml+2ml study drug .

Based on observations and results obtained in our study involving 30 patients in each group are discussed in detail by comparing with the available evidences in the literature. The analgesic efficacy of epidurally administered 10 ml of 0.25 % Bupivacaine + 2 ml Normal saline (Group BS) ,10 ml of 0.25% Bupivacaine + 8 mg of Dexamethasone was studied.

All the demographic tools like Height, Weight, Age were comparable to each other. There is no statistically significant difference between these parameters.

MEAN TIME FOR FIRST ANALGESIC REQUEST

On comparing the time for first analgesic request post operatively between two groups, the patients in Group BS (3.53 hours) requested first analgesic dose earlier than the patients from group BD(2.27 hours). This was found to be statistically significant with the p value of 0.000

The onset of pain was earlier in theBS group when compared to BD group. This study confirmed that onset of pain is earlier with the local anaesthetic alone than with the combination of dexamethasone and local anaesthetic. This correlates with the study of **Youn Yi Joun et al** who concluded that epidural administration of dexamethasone 5 mg in patients undergoing radical subtotal gastrectomy resulted in less VAS score and rescue analgesic requirements than the control group.

MEAN DURATION OF ANALGESIA:

The duration of postoperative analgesia was prolonged in Group BD patients receiving Dexamethasone (Mean 351.93) than in the Group BS patients receiving Normal saline(Mean 267.5).

The results of our study correlates with the study done by **Khafagy et al.** In his study he concluded that epidural dexamethasone resulted in a low post-

operative pain score and analgesic requirements, and pronged analgesic duration. The results of our study also correlates with the study of **Thomas &Beevi et al**who concluded that patients receiving epidural dexamethasone had les postoperative VAS score and analgesic consumption. Dexamethasone had action at spinal cord level in addition to its action on the peripheral tissues after systemic absorption from the epidural space.

SIDE EFFECTS :

The incidence of hypotension in Group BD receiving Dexamethasone is 6.7 %. There is nil incidence of hypotension in Group BSreceiving Normal saline. p value is 0.150 which is not statistically significant.

There is nil incidence of pruritus in both the Groups.

No patients had complaints of nausea in both the groups.

SUMMARY

After getting ethical committee approval the study was conducted in 60 patients undergoing gynaecological procedures belonging to ASA physical status 1 &2 .the 60 patients enrolled in the study were divided into two groups.

The data were statistically analysed ,compared and discussed.the results obtained are summarised below:

1) The demographic data like age,weight and height were comparable to each other in both the groups.

2) Time for first anaelgesic request was significantly earlier in group BS patients receiving normal saline(2.27 hrs) and it was delayed in dexamethasone receiving group(3.53)hrs

3) The duration of analgesia was significantly prolonged in group in BD patients receiving dexamethasone (351.53)when compared to group BS patients receiving normal saline(260.50)

4) Regarding side effects two patients receiving dexamethasone had hypotension . This was treated with fluid bolus and single dose of injection ephedrine 6mg iv and hypotension wasn't found in patients receiving normal saline.

5) There is nil incidence of nausea and pruruitus in both the groups.

CHAPTER 7

CONCLUSION

We conclude that epidural administration of dexamethasone – bupivacaine admixture resulted in better postoperative analgesia in terms of lower postoperative pain score, prolonged postoperative analgesia and patient comfort with fewer side effects when compared with the other two groups.

We also conclude that this resulted in prolonged postoperative analgesia without any side effects like nausea, pruritus except hypotension in few patients.

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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work	:	A prospective randomized comparative study of Epidural Administration of 0.25% bupivacaine versus 0.25% bupivacaine+8mg Dexamethasone on postoperative analgesia following Gynaecology surgery
Principal Investigator	:	Dr. Mahendiravarman
Designation	:	PG, MD (Anaesthesiology)
Department	:	Department of Anaesthesiology Government Stanley Medical College, Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 24.03.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

20/9/16

STUDY CASE

NAME AGE/SEX UNIT IP No

DIAGNOSIS SURGERY

PRE OP ASSESSMENT

Weight
Height
BMI
COMORBIDS
MEDICATIONS
ALLERGY
ASA PS

Air way
PR
SPO2

Hb

RFT

LFT

COAGULATION PROFILE

INTRAOP

IV LINE
SIZE
SITE

MONITORS
ECG
NIBP
SPO2
ETCO2
TEMP
OTHERS

EPIDURAL

POSITION
SKIN TO SPACE
CATHETER IN SPACE
CATHETER TO SKIN

LORT
NEGATIVE ASPIRATION
TEST DOSE _____ TIME
DRUG _____ TIME

GA/CV

Preoxygenation
Glycopyrrolate
Midazolam
Fentanyl
Thiopentone Sodium

Atracurium
succinycholine

ETT
C/L
OELM
GEB
N2O _____ lt Neostigmine _____ _____
O2 _____ lt Glycopyrrolate _____ _____
Inhalational
Agent _____

INTRA-OP MONITORING

	TIME	PR	BP	SPO2	IVF
Baseline					
After Epidural					

AT SHIFTING

PR
BP
SPO2

PROTECTIVE REFLEXES

VAS SCORE

POST - OP

	VAS SCORE	INTERVENTION / TIME / DOSE
ON ARRIVAL IN IACU		

சுய ஒப்புதல் படிவம்

பெண்மை பிணியியல் (Gynaecology) அறுவை சிகிச்சையின் போது ஏற்படும் வலியை குறைக்க பயன்படுத்தப்படும் எபிடுரல் (Epidural) முறையிலான சிகிச்சையில் கொடுக்கப்படும் பூபிவெகைன் மருந்துடன் டெக்ஸாமெத்தசோன் சேர்த்து கொடுப்பதால் ஏற்படும் விளைவுகள் பற்றிய ஆய்வு.

ஆய்வாளர் : **மரு. கா. மகேந்திரவர்மன்,**
முதுநிலை பட்டமேற்படிப்பு மாணவர்,
மயக்கவியல்துறை,
ஸ்டான்லி மருத்துவ கல்லூரி,
சென்னை - 600 001.

வழிகாட்டி : **பேராசிரியர் நஹரித்அசார், எம்.பி.பி.எஸ். எம்.டி.,**
மயக்கவியல்துறை,
ஸ்டான்லி மருத்துவ கல்லூரி,
சென்னை - 600 001.

பெயர் : வயது : உள்ளிருப்பு எண் :

இந்த ஆய்வின் விவரங்கள் எனக்கு அளிக்கப்பட்டது. என்னுடைய சந்தேகங்களை தீர்க்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்பளிக்கப்பட்டது.

நான் இந்த ஆய்வில் தன்னிச்சையாக பங்கேற்கிறேன். எக்காரணத்தினாலும் எந்த கட்டத்திலும் எந்த சட்ட சிக்கலும் இன்றி இந்த ஆய்விலிருந்து விலகிக் கொள்ளலாம் என அறிந்து கொண்டேன்.

நான் ஆய்விலிருந்து விலகிக் கொண்டாலும் ஆய்வாளர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கோ அல்லது உபயோகிக்கவோ என் அனுமதி தேவையில்லை என்றும் அறிந்து கொண்டேன். என்னைப் பற்றிய தகவல்கள் இரகசியமாக பாதுகாக்கப்படும் என்பதையும் அறிவேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் ஆய்வாளர் அவர் விருப்பத்திற்கு ஏற்ப பயன்படுத்தி கொள்ளவும், அதனை பிரசுரிக்கவும் முழு மனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் ஆய்வாளருக்கு உண்மையுடன் இருப்பேன் எனவும் உறுதியளிக்கிறேன்.

உடல்நலம் பாதிக்கப்பட்டாலோ, வழக்கத்திற்குமாறாக ஏதேனும் நோய்குறி தென்பட்டாலோ அதனை தெரிவிப்பேன் என உறுதி கூறுகிறேன்.

இந்த ஆய்வில் எனக்கு எவ்விதமான பரிசோதனைகளையும் சிகிச்சைகளையும், மேற்கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன்.

இப்படிக்கு

ஆய்வாளர் கையொப்பம்

நோயாளியின் கையொப்பம்

நோயாளியின் தகவல் தாள்

பெண்மை பிணியியல் (Gynaecology) அறுவை சிகிச்சையின் போது ஏற்படும் வலியை குறைக்க பயன்படுத்தப்படும் எபிடுரல் (Epidural) முறையிலான சிகிச்சையில் கொடுக்கப்படும் பூபிவெகைன் மருந்துடன் டெக்ஸாமெத்தசோன் (Dexamethasone) சேர்த்து கொடுப்பதால் ஏற்படும் விளைவுகள் பற்றிய ஆய்வு.

ஆராய்ச்சியின் நோக்கமும், ஆதாரங்களும் :

அறுவை சிசிக்சை செய்வதற்கு மயக்க மருந்து அவசியமானது. அவ்வாறான மயக்க மருந்துகளும் மயக்க முறைகளும் பலவகை உண்டு. அவற்றில் எபிடுரல் வகை என்பது, நீண்ட நேர அறுவை சிகிச்சைகளுக்கு வலியை குறைத்து பயன் தரக் கூடியதாக அமையும். அவ்வகை மயக்கமுறையில், பூபிவெகைன் மருந்தோடு டெக்ஸாமெத்தசோன் (Dexamethasone) எனும் மருந்தை சேர்த்து கொடுப்பதால் ஏற்படும் விளைவுகளை பற்றி இந்த ஆய்வு மேற்கொள்ளப்படுகிறது.

ஆய்வுமுறை :

நீங்கள் இரு குழுக்களாகப் பிரிக்கப்படுவீர்கள், ஒரு குழுவிற்கு எபிடுரல் மயக்கமுறையில் பூபிவெகைனும் மற்றொரு குழுவிற்கு பூபிவெகைனோடு சேர்த்து டெக்ஸாமெத்சோனும் கொடுக்கப்பட்டு வலியின்மையின் நேரம் கணக்கிடப்படும் அறுவை சிகிச்சைக்குப் பிறகான முதல் வலி நிவாரணி தேவைக்கான கால அளவும் கணக்கிடப்படும்.

மேலும் மயக்கமருந்து கொடுக்கப்பட்ட 24 மணி நேரத்திற்கு ஏதேனும் பக்க விளைவுகள் ஏற்பட்டதா என்று கண்காணிக்கப்படும்.

உண்டாக கூடிய இடர்கள் :

இந்த ஆய்வின் போது பயன்படுத்தப்படும் புபிவெகைன் மற்றும் டெக்ஸாமெத்தசோன் மருந்தினால் இரத்த அழுத்தம், இதயத் துடிப்பு, சர்க்கரை அளவில் மாற்றமோ அல்லது வலிப்பு ஏற்படவோ வாய்ப்புகள் உண்டு.

ஆய்வில் உள்ள உரிமைகள் :

உங்கள் மருத்துவ பதிவேடுகளின் இரகசியம் காக்கப்படும். இந்த ஆய்வின் முடிவுகள் மருத்துவ இதழில் வெளிடப்படலாம். ஆனால் உங்கள் பெயர் அடையாளம் காட்டப்படமாட்டாது. இந்த ஆய்வில் பங்கேற்பது தன்னிச்சையானது மற்றும் வேறு காரணங்களால் நீங்கள் முன்னறிவிப்பின்றி எப்பொழுது வேண்டுமானாலும் விலகிக் கொள்ளலாம். ஏதேனும் பக்க விளைவுகள் ஏற்படின் முழு சிகிச்சையும், மருத்துவ குழுவினரால் உடனடியாக வழங்கப்படும்.

நோயாளியின் கையொப்பம்

அனிதா. M

தேதி :

இடது பெருவிரல் ரேகை

(மருத்துவரால் தெளிவாக படித்து காட்டப்பட்டது)

PATIENT INFORMATION SHEET

1. We are conducting a study on "RANDOMISED CONTROLLED DOUBLE BLINDED STUDY ON EPIDURAL ADMINISTRATION OF PLAIN BUPIVACAINE VERSUS BUPICAINE WITH ADDED DEXAMETHASONE FOLLOWING GYNAECOLOGY SURGERY.

2. We are selecting certain patients and if you are found eligible, we may be using you to perform procedures which will not harm you.

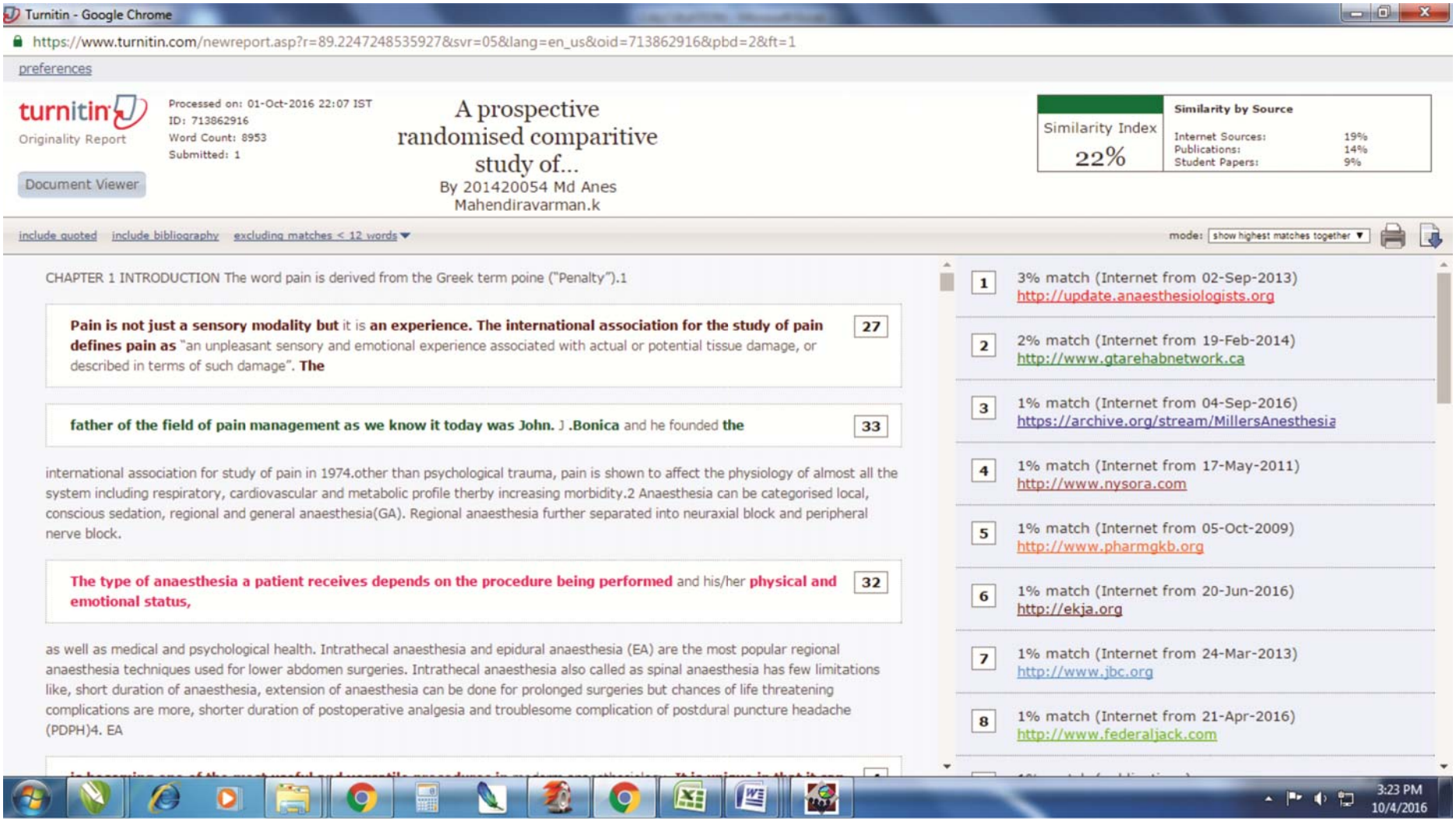
3. The privacy of patients in this research will be maintained throughout the study. In the event of any publication (or) presentation resulting from the research, no personally identifiable information will be shared.

4. Taking part in this study is voluntary, you are free to decide whether to participate in this study (or) to withdraw at any time, your decision will not result in any loss of benefits to which you are otherwise entitled.

Date :

Signature of Investigator

Signature of Participant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CHAPTER 1 INTRODUCTION The word pain is derived from the Greek term poine ("Penalty").1
Pain is not just a sensory modality but it is an experience. The international association for the study of pain defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage". The
27
father of the field of pain management as we know it today was John. J .Bonica and he founded the
33
international association for study of pain in 1974.other than psychological trauma, pain is shown to affect the physiology of almost all the system including respiratory, cardiovascular and metabolic profile therby increasing morbidity.2 Anaesthesia can be categorised local, conscious sedation, regional and general anaesthesia(GA). Regional anaesthesia further separated into neuraxial block and peripheral nerve block.
The type of anaesthesia a patient receives depends on the procedure being performed and his/her physical and emotional status,
32
as well as medical and psychological health. Intrathecal anaesthesia and epidural anaesthesia (EA) are the most popular regional anaesthesia techniques used for lower abdomen surgeries. Intrathecal anaesthesia also called as spinal anaesthesia has few limitations like, short duration of anaesthesia, extension of anaesthesia can be done for prolonged surgeries but chances of life threatening complications are more, shorter duration of postoperative analgesia and troublesome complication of postdural puncture headache (PDPH)4. EA
1 3% match (Internet from 02-Sep-2013) http://update.anaesthesiologists.org
2 2% match (Internet from 19-Feb-2014) http://www.gtarehabnetwork.ca
3 1% match (Internet from 04-Sep-2016) https://archive.org/stream/MillersAnesthesia
4 1% match (Internet from 17-May-2011) http://www.nysora.com
5 1% match (Internet from 05-Oct-2009) http://www.pharmgkb.org
6 1% match (Internet from 20-Jun-2016) http://ekja.org
7 1% match (Internet from 24-Mar-2013) http://www.jbc.org
8 1% match (Internet from 21-Apr-2016) http://www.federaljack.com
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S.NO	NAME	AGE	GENDER	IP.NO	HEIGHT	WEIGHT	ASA	GROUP	DATE	DIAGNOSIS	PROCEDURE		VAS SCORE								
												DURATION OF SURGERY	0 HOUR	1 HOUR	2 HOURS	3HOURS	4 HOURS	5 HOURS	6 HOURS	12 HOURS	18 HOURS
1	Dhanalakshmi	40	F	4357	145cms	52kgs	ps1	BS	05/01/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	120 mins	3	4	6	2	2	2	6	3	4
2	Mallika Hussain	40	F	3198	152cms	53kgs	ps2	BS	05/03/2016	AUB	Total abdominal Hysterectomy with BSO	127mins	4	6	2	6	2	2	4	6	2
3	Thabirisha	45	F	4576	151cms	54kgs	ps1	BD	05/04/2016	Ovarian cyst	Cystectomy	110mins	2	4	6	2	6	2	2	5	7
4	Shenbagavalli	35	F	4252	147cms	49kgs	ps3	BD	05/05/2016	Fibroid uterus	Total abdominal Hysterectomy	130mins	2	4	4	6	2	2	2	5	2
5	Zamruth	40	F	4920	147cms	62kgs	ps2	BS	05/06/2016	Fibroid uterus	Total abdominal Hysterectomy	120mins	2	6	4	2	2	6	4	4	6
6	Indirani	45	F	4342	150cms	54kgs	ps1	BD	05/09/2016	Fibroid uterus	Total abdominal Hysterectomy	145mins	3	7	2	3	4	4	7	3	2
7	Vijaya	41	F	3749	152cms	65kgs	ps2	BD	05/10/2016	Mod- dysplasia of ce	Total abdominal Hysterectomy with BSO	130mins	1	2	2	6	2	7	2	2	4
8	Anjalai	42	F	4819	155cms	55kgs	ps1	BS	05/11/2016	Ovarian cyst	Staging Laparotomy	100mins	2	7	1	1	1	2	4	7	2
9	Shakira	42	F	4268	156cms	49kgs	ps2	BD	05/12/2016	B/L Ovarian cyst	Total abdominal Hysterectomy	130mins	3	4	4	4	4	4	7	3	3
10	Govindammal	43	F	5217	154cms	56kgs	ps1	BS	13/5/2016	Fibroid uterus	Total abdominal Hysterectomy	110mins	1	2	4	6	2	2	4	7	2
11	Suganthi	45	F	4623	148cms	55kgs	ps2	BS	16/5/2016	Fibroid uterus	Total abdominal Hysterectomy	115mins	2	7	2	2	2	2	6	1	2
12	Vijaya Roja	41	F	5358	149cms	53kgs	ps2	BD	17/5/2016	Fibroid uterus	Total abdominal Hysterectomy	135mins	2	3	3	7	2	2	2	2	2
13	Anitha	45	F	5151	151cms	52kgs	ps1	BD	18/5/2016	III UV prolapse	Vaginal Hysterectomy with PFR	140mins	2	2	2	3	3	5	2	2	2
14	Latha	42	F	5508	160cms	49kgs	ps1	BD	19/5/2016	III UV prolapse	Vaginal Hysterectomy with PFR	126mins	3	3	3	3	4	6	2	6	3
15	Kumari	35	F	5575	161cms	52kgs	ps1	BD	20/5/2016	Fibroid uterus	Total abdominal Hysterectomy	110mins	2	3	3	6	3	3	3	6	2
16	Nisha	49	F	5980	160cms	53kgs	ps2	BS	23/5/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	128mins	3	3	4	6	2	3	3	6	2
17	Suguna	40	F	5666	159cms	61kgs	ps1	BS	24/5/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	132mins	2	4	6	2	4	4	4	6	4
18	Valarmathy	45	F	8741	154cms	62kgs	ps1	BS	25/5/2016	Left Ovarian cyst	Laparotomy and proceed	96mins	1	2	4	6	2	2	4	7	2
19	Nagammal	43	F	6701	155cms	63kgs	ps1	BS	26/5/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	116mins	2	7	2	2	2	2	6	1	2
20	Harilakshmi	46	F	5589	156cms	58kgs	ps1	BS	27/5/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	118mins	1	2	4	6	2	2	4	7	2
21	Rajeshwari	45	F	6009	156cms	59kgs	ps2	BS	31/5/2016	Endometrial polyp	Total abdominal Hysterectomy	132mins	2	7	2	2	2	2	6	1	2
22	Sathyavani	48	F	6083	158cms	48kgs	ps1	BS	06/01/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	147mins	2	7	1	1	1	2	4	7	2
23	Sulochana	38	F	6147	149cms	51kgs	ps1	BS	06/02/2016	III UV prolapse	Vaginal Hysterectomy with PFR	128mins	1	2	4	6	2	2	6	2	2
24	Kasthuri	47	F	6248	150cms	52kgs	ps1	BS	06/03/2016	UV Prolapse	Vaginal Hysterectomy with PFR	132mins	2	2	4	6	2	3	4	6	2
25	Aruna	59	F	6307	149cms	53kgs	ps2	BD	06/06/2016	UV Prolapse	Vaginal Hysterectomy with PFR	136mins	2	2	4	6	2	2	4	6	2
26	Elakiya	25	F	6784	160cms	54kgs	ps1	BS	06/07/2016	Right Complex Ovaria	Laparotomy and proceed	111mins	2	2	6	2	2	4	6	4	6
27	Thievam	39	F	5692	155cms	55kgs	ps1	BD	06/08/2016	Fibroid uterus	Total abdominal Hysterectomy	94mins	2	2	6	2	2	2	6	4	2
28	Dhanalakshmi	52	F	6896	155cms	54kgs	ps2	BD	06/09/2016	Fibroid uterus	Total abdominal Hysterectomy	126mins	2	3	6	2	2	2	6	2	4
29	Muniammal	43	F	6890	156cms	65kgs	ps1	BD	06/10/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	116mins	2	3	6	2	2	2	6	2	4
30	Noorjahan	40	F	6982	157cms	64kgs	ps1	BS	13/6/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	128mins	2	2	2	6	3	3	4	2	2
31	Prema	45	F	6994	158cms	63kgs	ps1	BS	06/10/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	136mins	2	2	4	6	2	3	4	6	2
32	Nalini	40	F	6561	159cms	62kgs	ps1	BS	06/10/2016	III UV prolapse	Vaginal Hysterectomy with PFR	122mins	2	2	2	6	3	3	4	2	2
33	Alamelu	40	F	6410	160cms	63kgs	ps1	BS	13/6/2016	Fibroid uterus	Total abdominal Hysterectomy	156mins	2	2	4	6	2	3	4	6	2
34	Kasthuri	60	F	6716	154cms	65kgs	ps2	BD	13/6/2016	Papillary Ca cervix	Werthins Hysterectomy	126mins	3	4	4	4	4	4	7	3	3
35	Anjali devi	60	F	6854	153cms	56kgs	ps2	BS	15/6/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	130mins	2	3	6	2	2	2	6	2	2
36	Nalini	36	F	6870	152cms	57kgs	ps1	BD	15/6/2016	Fibroid uterus	Total abdominal Hysterectomy	110mins	2	2	2	3	3	5	2	2	2
37	Kavitha	29	F	7271	153cms	55kgs	ps1	BD	16/6/2016	Right Ovarian Cyst	Right ovarian cystectomy	90mins	3	3	3	3	4	6	2	6	3
38	Sundari	48	F	6366	154cms	58kgs	ps1	BD	17/6/2016	UV Prolapse	Vaginal Hysterectomy with PFR	136mins	2	3	3	6	3	3	3	6	2
39	Saroja	70	F	6406	156cms	58kgs	ps2	BD	20/6/2016	III UV prolapse	Vaginal Hysterectomy with PFR	140mins	2	3	3	3	4	6	1	2	2
40	Ponni	45	F	6492	154cms	59kgs	ps1	BS	20/6/2016	Post menopausal ble	Total abdominal Hysterectomy with BSO	112mins	2	2	2	6	3	3	4	2	2
41	Mugila	60	F	6501	152cms	55kgs	ps2	BD	21/6/2016	Ca Ovary	Staging Laparotomy	152mins	2	2	3	4	6	1	2	2	2
42	Shameena	55	F	7008	150cms	49kgs	ps2	BD	22/6/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	126mins	2	2	4	6	2	2	4	6	2
43	Gulzar	45	F	6571	149cms	53kgs	ps1	BS	22/6/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	118mins	2	2	2	6	3	3	4	2	2

44	Malliga	55	F	7297	148cms	52kgs	ps2	BD	23/6/2016	PMB	Total abdominal Hysterectomy with BSO	112mins	2	3	3	6	3	3	3	6	2
45	Susheela	50	F	6986	149cms	58kgs	ps1	BD	24/6/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	130mins	2	3	3	3	4	6	1	2	2
46	Aruvammal	45	F	7790	147cms	59kgs	ps1	BD	28/6/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	122mins	1	1	2	2	2	2	2	2	3
47	Parvathy	55	F	7884	146cms	58kgs	ps2	BD	28/6/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	112mins	1	2	2	4	6	2	2	2	2
48	Ramya	60	F	6500	149cms	54kgs	ps1	BS	29/6/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	117mins	2	2	2	6	3	3	4	2	2
49	Radhika	44	F	6499	150cms	52kgs	ps1	BD	29/06/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	126mins	2	4	6	2	6	2	2	5	7
50	Rekha	47	F	6920	154cms	51kgs	ps1	BS	30/06/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	132mins	2	7	2	2	2	2	6	1	2
51	Parvathi	52	F	6821	155cms	55kgs	ps1	BD	30/06/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	128mins	3	3	3	3	4	6	2	6	3
52	Prema	54	F	6710	156cms	56kgs	ps2	BD	30/06/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	116mins	2	3	3	6	3	3	3	6	2
53	Uma	50	F	7001	158cms	57kgs	ps2	BD	01/07/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	128mins	2	3	6	2	2	2	6	2	4
54	Muniyamma	67	F	7212	159cms	55kgs	ps2	BS	01/07/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	130mins	2	2	2	6	3	3	4	2	2
55	Rani	70	F	6543	160cms	58kgs	ps2	BS	02/07/2016	Uv prolapse	Vaginal Hysterectomy with PFR	138mins	2	2	4	6	2	3	4	6	2
56	Kuppu	64	F	6781	154cms	55kgs	ps1	BD	02/07/2016	UV Prolapse	Vaginal Hysterectomy with PFR	148mins	2	3	3	3	4	6	1	2	2
57	Subulakshmi	37	F	7002	159cms	54kgs	ps1	BD	04/07/2016	UV Prolapse	Vaginal Hysterectomy with PFR	136mins	1	1	2	2	2	6	2	2	3
58	Sornam	59	F	7126	149cms	53kgs	ps2	BS	04/07/2016	UV Prolapse	Vaginal Hysterectomy with PFR	130mins	1	2	2	4	6	2	2	2	2
59	Sellam	61	F	7234	148cms	63kgs	ps2	BS	04/07/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	128mins	3	4	6	2	2	2	6	3	4
60	Panjali	60	F	7312	156cms	65kgs	ps1	BS	05/07/2016	Fibroid uterus	Total abdominal Hysterectomy with BSO	120mins	4	6	2	6	2	2	4	6	2

	TIME FOR FIRST	uration of Analgesi	SIDE EFFECTS				SYSTOLIC BP							DIASTOLIC BP							PULSE RATE						
24 HOURS	NALGESIC REQUES		YPOTENSIC	RADYCARD	PRURITUS	TORY DEPI	BASELINE	TER EPIDUF	30 MIN	60 MIN	90 MIN	120 MIN	150 MIN	BASELINE	TER EPIDUF	30 MIN	60 MIN	90 MIN	120 MIN	150 MIN	BASELINE	TER EPIDUF	30 MIN	60 MIN	90 MIN	120 MIN	150 MIN
6	2 hours	240mins	NIL	NIL	NIL	NIL	112	124	110	108	115	116	110	62	65	66	69	62	71	69	78	92	80	78	84	86	84
4	1 hour	197 mins	NIL	NIL	NIL	NIL	132	128	124	125	122	120	115	70	80	82	72	80	68	65	80	88	88	90	92	86	85
4	2 hours	230mins	NIL	NIL	NIL	NIL	122	126	100	106	122	124	120	75	60	66	62	70	67	65	78	96	85	88	83	80	79
4	3 hours	310 mins	YES	NIL	NIL	NIL	136	140	86	102	90	100	122	85	90	56	60	64	52	60	86	102	75	74	72	70	74
2	1 hour	180mins	NIL	NIL	NIL	NIL	118	124	110	115	110	116	118	66	62	70	60	68	64	65	72	78	78	70	68	74	68
6	1 hour	205mins	NIL	NIL	NIL	NIL	128	130	120	122	120	118	115	68	64	74	72	63	70	72	74	88	78	80	70	84	80
4	3 hours	310mins	NIL	NIL	NIL	NIL	128	132	110	118	122	125	134	82	77	70	74	79	80	73	84	92	80	79	77	80	76
4	1 hour	160mins	NIL	NIL	NIL	NIL	125	128	118	120	118	120	112	78	80	78	82	77	75	72	84	96	80	88	82	85	83
5	6 hours	490mins	NIL	NIL	NIL	NIL	128	132	110	114	118	122	112	73	70	68	68	70	67	72	76	104	79	83	80	78	81
2	3 hours	290mins	NIL	NIL	NIL	NIL	138	140	126	128	125	130	128	75	80	84	85	70	68	72	68	82	70	72	66	68	70
2	1 hour	175mins	NIL	NIL	NIL	NIL	116	122	100	98	100	114	117	81	62	66	64	70	69	69	78	88	72	78	80	72	74
6	3 hours	315mins	YES	NIL	NIL	NIL	134	106	96	77	76	82	90	83	74	52	50	46	66	56	74	90	77	69	70	72	75
2	5 hours	440mins	NIL	NIL	NIL	NIL	118	124	120	115	122	128	100	85	90	80	85	85	80	88	80	84	78	80	78	72	70
3	5 hours	426mins	NIL	NIL	NIL	NIL	122	136	110	112	120	118	112	77	80	82	85	80	75	85	84	92	80	77	71	82	80
2	3 hours	290mins	NIL	NIL	NIL	NIL	128	130	110	122	130	132	122	68	66	68	70	72	75	68	76	78	72	80	78	76	74
2	3 hours	308mins	NIL	NIL	NIL	NIL	135	140	124	127	125	134	128	85	90	86	85	92	80	88	80	90	70	74	70	72	76
4	2 hours	252mins	NIL	NIL	NIL	NIL	120	116	98	102	117	116	120	82	66	70	69	71	73	70	70	78	67	73	69	77	75
2	3 hours	276mins	NIL	NIL	NIL	NIL	126	128	120	125	118	114	122	70	80	84	88	80	75	85	84	92	86	84	88	84	82
2	1 hour	176mins	NIL	NIL	NIL	NIL	137	140	130	127	125	128	132	86	90	88	85	85	95	88	78	86	80	84	78	82	76
2	3 hours	298mins	NIL	NIL	NIL	NIL	128	126	116	122	125	132	125	64	68	70	62	70	72	70	78	90	82	86	82	84	82
2	1 hour	198mins	NIL	NIL	NIL	NIL	132	140	110	115	114	126	110	75	72	68	69	67	65	69	70	88	70	71	73	76	69
4	1 hour	207mins	NIL	NIL	NIL	NIL	135	140	110	132	122	127	110	73	67	70	69	66	70	68	72	76	74	75	67	68	77
4	3 hours	308mins	NIL	NIL	NIL	NIL	118	122	114	116	122	126	120	75	80	80	85	88	75	83	88	112	92	90	88	92	90
4	3 hours	312mins	NIL	NIL	NIL	NIL	127	130	116	120	122	118	122	81	74	78	76	70	72	75	76	78	70	72	75	70	69
4	3 hours	316mins	NIL	NIL	NIL	NIL	132	140	96	100	112	116	114	77	60	64	63	69	75	68	85	88	83	81	79	76	77
2	2 hours	231mins	NIL	NIL	NIL	NIL	130	132	120	122	126	128	120	86	87	80	85	84	87	88	80	92	84	82	86	84	82
4	2 hours	214mins	NIL	NIL	NIL	NIL	126	128	110	118	115	119	120	72	67	68	65	68	63	69	92	96	93	90	88	86	90
6	2 hours	246mins	NIL	NIL	NIL	NIL	112	118	110	116	118	114	108	74	70	68	69	67	69	65	84	86	85	82	88	84	91
6	2 hours	236mins	NIL	NIL	NIL	NIL	132	134	124	128	125	124	128	72	72	74	77	73	75	72	92	102	94	96	92	88	96
2	3 hours	308mins	NIL	NIL	YES	NIL	127	130	124	126	130	128	128	85	78	76	85	77	80	82	88	110	88	92	90	86	86
4	3 hours	316mins	NIL	NIL	NIL	NIL	125	126	120	128	127	125	121	83	80	82	77	70	71	75	88	108	86	88	84	86	86
2	3 hours	302mins	NIL	NIL	NIL	NIL	136	140	120	122	117	121	123	79	74	72	73	75	77	72	80	92	74	79	85	84	86
4	3 hours	336mins	NIL	NIL	NIL	NIL	112	116	110	108	112	116	108	71	68	70	69	72	75	70	80	96	85	90	87	82	77
5	6 hours	486mins	NIL	NIL	NIL	NIL	132	136	116	122	135	130	128	84	80	78	78	75	77	73	74	84	79	81	88	80	78
4	2 hours	250mins	NIL	NIL	NIL	NIL	136	140	124	128	120	130	122	82	77	76	80	82	80	76	88	96	88	86	88	92	87
2	5 hours	410mins	NIL	NIL	NIL	NIL	108	110	122	112	110	115	112	68	60	68	66	62	64	69	82	88	74	78	88	82	85
3	5 hours	390mins	NIL	NIL	NIL	NIL	128	130	108	98	118	124	120	85	65	66	69	71	73	76	74	92	79	82	84	79	77
2	3 hours	316mins	NIL	NIL	NIL	NIL	104	108	100	96	112	120	125	75	69	72	72	69	70	75	76	84	81	84	83	80	77
3	5 hours	440mins	NIL	NIL	NIL	NIL	132	136	132	128	127	124	122	70	67	68	66	69	65	71	72	90	90	84	80	79	91
2	3 hours	292mins	NIL	NIL	NIL	NIL	126	128	128	120	126	127	118	75	72	72	72	78	68	70	84	88	80	84	82	84	86
4	4 hours	352mins	NIL	NIL	NIL	NIL	118	120	128	116	114	120	118	78	90	88	82	78	80	75	82	102	84	88	86	84	88
4	3 hours	306mins	NIL	NIL	NIL	NIL	128	130	135	122	120	127	130	75	82	84	85	77	79	82	78	84	92	90	86	88	82
2	3 hours	298mins	NIL	NIL	NIL	NIL	120	124	104	115	122	116	118	82	84	86	85	77	73	80	88	100	88	88	86	86	84

2	3 hours	392mins	NIL	NIL	NIL	NIL	136	138	124	128	132	124	126	77	72	74	69	73	75	73	84	88	84	88	85	89	93
3	5 hours	430mins	NIL	NIL	NIL	NIL	127	130	124	126	120	128	117	81	74	76	74	71	77	73	74	97	73	73	77	70	74
4	6hrs	482 mins	NIL	NIL	NIL	NIL	117	120	110	115	120	115	116	71	80	78	74	77	81	74	70	83	78	70	66	74	72
2	4 hours	352mins	NIL	NIL	NIL	NIL	138	140	124	132	130	132	128	68	60	64	62	67	59	66	78	91	78	74	76	78	78
2	3 hours	297mins	NIL	NIL	NIL	NIL	136	138	132	128	124	130	128	73	70	66	69	67	68	67	84	85	86	79	77	75	79
4	2 hours	246mins	NIL	NIL	NIL	NIL	122	126	100	106	122	124	120	75	60	66	62	70	67	65	78	96	85	88	83	80	79
2	1 hour	182mins	NIL	NIL	NIL	NIL	116	122	100	98	100	114	119	81	62	66	64	70	69	68	78	88	72	78	80	72	74
3	5 hours	428mins	NIL	NIL	NIL	NIL	122	136	110	112	120	118	120	77	80	82	85	80	75	85	84	92	80	77	71	82	80
2	3 hours	296mins	NIL	NIL	NIL	NIL	128	130	110	122	130	132	122	68	66	68	70	72	75	76	76	78	72	80	78	76	74
6	2 hours	248mins	NIL	NIL	NIL	NIL	132	134	124	128	125	124	128	72	72	74	77	73	75	72	92	102	94	96	92	88	96
2	3 hours	310mins	NIL	NIL	NIL	NIL	127	130	124	126	130	128	128	85	78	76	85	77	80	82	88	110	88	92	90	86	86
4	3 hours	318mins	NIL	NIL	NIL	NIL	125	126	120	128	127	125	125	83	80	82	77	70	71	75	88	108	86	88	84	86	86
3	5 hours	448mins	NIL	NIL	NIL	NIL	127	130	124	126	120	128	117	81	74	76	74	71	77	73	74	97	73	73	77	70	74
4	6hours	508 mins	NIL	NIL	NIL	NIL	117	120	110	115	120	115	116	71	80	78	74	77	81	74	70	83	78	70	66	74	72
2	4 hours	370mins	NIL	NIL	NIL	NIL	138	140	124	132	130	132	128	68	60	64	62	67	59	66	78	91	78	74	76	78	78
6	2 hours	248mins	NIL	NIL	NIL	NIL	112	124	110	108	115	116	110	62	65	66	69	62	71	69	78	92	80	78	84	86	84
4	1 hour	180mins	NIL	NIL	NIL	NIL	132	128	124	125	122	120	122	70	80	82	72	80	68	70	80	88	88	90	92	86	85