"COMPARATIVE STUDY OF ADDUCTOR CANAL BLOCK IN ADDITION TO EPIDURAL INFUSION VERSUS EPIDURAL INFUSION ALONE ON POST-OPERATIVE ANALGESIA IN PATIENTS UNDERGOING KNEE SURGERIES- A RANDOMIZED CONTROL TRIAL"

BY

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LIST OF ABBREVIATIONS

ASA	**American Society of Anesthesiologists**
ACB	**Adductor canal blockade**
CACB	**Continuous Adductor Canal Block**
SACB	**Single-shot Adductor Canal Block**
SA	**Spinal Anaesthesia**
CSEA	**Combine Spinal Epidural Anaesthesia**
PCM	**Paracetamol**
NSAIDS	**Non-Steroidal Anti-Inflammatpry Drugs**
CVA	**Cerebral Vascular Accident**
PNBS	**Peripheral Nerve Blocks**
IV PCA/ PCIA	**Intravenous Patient Controlled Analgesia**
ACLR	**Anterior Cruciate Ligament Repair**
TKR	**Total Knee ReplacemenT**
TKA	**Total Knee Aethroplasty**
CEA	**Continuous Epidural Analgesia**
IA	**Intra-Articular**
IPACK	**Infiltration between the Popliteal A rtery and Capsule of the Knee**
LIA	**Local Infiltration analgesia**
USG	**Ultrasonography**

NRS	**Numerical Rating scale**
SSFNB	**Single-Shot Femoral Nerve Block**
CFNB	**Continous Femoral Nerve Block**
PACU	**Post Anaesthesia Care Unit**
POD	**Post-Operative Day**

ABSTRACT

Title : Comparative Study Of Adductor Canal Block In Addition To Epidural Infusion Versus Epidural Infusion Alone On Post-Operative Analgesia In Patients Undergoing Knee Surgeries- A Randomized Control Tria

Background: Knee surgeries, including total knee arthroplasty (TKA) and arthroscopy, are associated with significant postoperative pain, which can impact recovery and prolong hospital stays. Effective pain management strategies are essential to enhance early mobilization and improve patient outcomes. This study evaluates the efficacy of combining adductor canal block (ACB) with epidural analgesia in comparison to epidural analgesia alone for postoperative pain relief in knee surgeries.

Methods: The study analyzed postoperative pain relief in two groups: Group A, receiving epidural analgesia alone, and Group B, receiving epidural analgesia combined with ACB using ropivacaine. Pain levels were assessed using the Visual Analog Scale (VAS) at multiple postoperative time points (0 minutes, 30 minutes, 1 hour, 2 hours, 4 hours, 6 hours, 12 hours, and 24 hours). Additional parameters included the need for rescue analgesics, bolus administration, and the duration of analgesic action.

Results: Group B demonstrated significantly lower VAS scores than Group A within the first six hours postoperatively ($p < 0.05$), indicating superior pain control during the critical early recovery phase. At 12 and 24 hours, the pain scores were comparable between groups ($p > 0.05$). Tramadol consumption was lower in Group B, with fewer patients requiring higher doses. Bolus administration patterns further supported the efficacy of ACB, as Group B required significantly lower bolus volumes ($p < 0.001$).

The mean duration of analgesia was significantly longer in Group B (8.5 ± 1.71 hours) compared to Group A (4.4 ± 2.07 hours), demonstrating the prolonged analgesic effect of ACB.

Conclusion: The addition of ACB to epidural analgesia enhances postoperative pain relief, particularly in the immediate postoperative period, while reducing the need for additional analgesics. ACB preserves quadriceps function, facilitating early mobilization and potentially reducing opioid-related side effects. These findings support the integration of ACB into multimodal pain management protocols for knee surgeries. Further studies with larger sample sizes and long-term follow-ups are needed to validate these results and optimize pain management strategies.

Key-words: knee surgeries, post-operative pain, ropivacaine, CESA, Adductor canal block, rescue analgesia, bolus epidural

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INTRODUCTION

Knee procedures, such as arthroplasty or arthroscopy, are linked with mild to severe postoperative pain, which can lead to issues related to immobility and lengthen hospital stays. For this reason, post-operative pain and rehabilitation require efficient analgesia[2]. Oral opioids, NSAIDS, per-articular injections, PNBs, epidurals, & IV PCA are being used in pain management protocols after knee surgeries. PNBs are regarded as a crucial component of the current multimodal pain management strategy after knee arthroplasty because they offer efficient and complementary pain treatment when utilized as part of a multimodal regimen [4].

Knee surgeries are performed to replace the knee joint's weight-bearing component. For individuals with final stage knee arthritis who was not benefited with conservative therapies, total knee arthroplasty is the gold standard. Increased life span, physical inactivity, obesity & advancements in healthcare insurance framework and technology have led to an increase in number of joint replacement operations performed to enhance, quality of living [4].

One of the first indications of a pathological alteration in the meniscus, ligaments, cartilage, or bone of the joint is knee discomfort. A minimally invasive treatment called a knee arthroscopy can identify potential reasons of knee discomfort and surgically fix structural changes in the bone, cartilage, ligament, or meniscus. One of the most common procedures in sports medicine is arthroscopic knee surgery, which is especially useful for fixing bone spurs, torn cartilage, or torn meniscus.[14]

In recent years, advancements in pain management techniques have significantly impacted the practice of total knee replacement and arthroscopy surgeries. Providing effective postoperative pain relief with minimal side effects is

crucial for facilitating early mobilization and promoting recovery in patients undergoing knee surgeries.[3]

To treat post-operative pain, epidural analgesia is frequently utilized either by itself or in conjunction with oral or intravenous NSAIDS and opioid analgesics. After knee procedures, proper pain management speeds up recovery, lowers the chance of complications, and increases patient satisfaction[4].

Regional treatments include peri-articular infiltration, adductor canal block, and femoral nerve block have garnered more attention recently because of their ability to provide analgesia for longer periods of time with fewer adverse effects.

Although continuous epidural analgesia using an epidural catheter is regarded to be beneficial for managing postoperative pain following knee procedures, it frequ ntly necessitates the use of additional NSAIDS or opioids, which can have a number o f negative effects.

A new peripheral nerve block used to treat knee surgery pain is called adductor canal blockage (ACB). Early rehabilitation after knee surgery is made easier by ACB, a relatively new approach that mostly inhibits pain perceptions while maintaining quadriceps power. It has been shown that ACB is a useful substitute for femoral nerve blocks, offering comparable analgesic effects while notably preserving motor power[3].

Addition of ACB to regional blocks to epidural analgesia is synergistic in providing post-operative pain relief with the added advantage of decreasing side effect of epidural analgesia dosage.

Few studies comparing epidural analgesia with adductor canal block in relation to epidural analgesia alone for post procedural pain following knee surgeries were found in the literature search.

This study aims to compare epidural analgesia versus epidural analgesia combined with adductor canal block by injecting ropivacaine for post procedural pain relief following knee surgeries.

OBJECTIVES

Study objective

Primary Objective – To study the effect of adductor canal block to epidural analgesic infusion on the duration of post-operative analgesia and rescue analgesic requirements in patients undergoing knee surgeries under combined spinal epidural anaesthesia.

REVIEW OF LITERATURE

The ACB contends that by inhibiting the saphenous nerve (a sensory nerve) and a portion of the obturator nerve which passes through the adductor canal, local anesthetics injected into the canal will produce sufficient analgesia. The literature on ACB's effectiveness is expanding, and the data that is currently available suggests that ACB is effective at providing postoperative analgesia following knee surgery.[23]

One well-known analgesic technique is the use of epidural analgesia following major surgery. . Postoperative pain can be effectively and safely managed with epidural analgesia. There are many other positive advantages linked to this method Regular monitoring of epidural analgesia for postoperative pain treatment is necessary to assess patient satisfaction and prevent adverse effects.[24]

Girish joshi & authors observe the use of peripheral nerve block, either as a continuous infusion through a perineural catheter (cPNB) or as a single injection (sPNB), as part of multimodal analgesia is growing. In many surgical procedures, it demonstrated better pain management and reduced opioid use in subjects receiving PNB as opposed to those getting IV opioids. [13]In recent decades, epidural analgesia has become more and more popular, since neuraxial procedures in orthopaedic surgery have been shown to lessen blood loss and thromboembolic consequences.[13]

The efficacy & advance results of ACB versus CEA in participants who undergone solitary TKR were compared by Umut C et al. (2019) that involved 80 patients received single total knee arthroplasty. According to the findings, participants who experienced ACB were able to flex their knees during the first twenty-four hours after surgery. As stated by authors findings, utilizing ACB as a postoperative

analgesic following TKR is linked to greater early functional recovery, increased mobility, and a lower risk of serious postoperative sequelae [6].

Seham M. Moeen & authors (2025) compared ACB and IA (Intra-Articular**)** analgesia for post procedural pain management following AACLR in 72 patients. The participants were assigned randomly & injected 18 ml of 0.25% bupivacaine + 8 mg of dexamethasone in the ACB 30 minutes prior SAB or intra articular around the completion of operation. Pain scores, at rest & while mobilization, were evaluated at 6, 12, 18, and 24 hrs post procedure, until the first analgesic required & analgesics dose consumed were noted. The results showed significantly lower pain scores in IA class at 12, 18 & 24 hours post procedure, at rest and while mobility. Notably, no participants in the IA group required analgesia until 17 hours after surgery. Additionally, time to first ketorolac (1342.5 ± 132.7 vs. 1167.5 ± 297.36 minutes, P = 0.029) & morphine (1412.5 ± 87.94 vs. 1278.33 ± 274.04 minutes, P = 0.025) dosage were notably increased in IA participants contrast to ACB participants [15].

Rasim Onur Karaoğlu (2024) evaluated 80 participators undergone KS aimed to differentiate analgesic efficacy of a combination of ACB + IPACK blocks with single-dose epidural analgesia. The results revealed that both IPACK, ACB, and epidural analgesia provided comparable pain relief for patients undergoing arthroscopic knee surgery. At the 8th and 24th hours postoperatively, the block group showed similar analgesic effectiveness to the epidural group. However, the combined spinal epidural group provided more effective analgesia at the 1st hour post-surgery [16].

In a research of 123 participators arranged for single leg TKR, Khalid AA et al. (2020) discover How quickly patients walked following surgery, length of hospital stay (LOS), use of local anesthetic and total opioids, postoperative blood drainage output, incidence of nausea and vomiting, pain scores, and the degree of knee flexion and extension during physiotherapy sessions on postoperative day 1 and discharge were the main outcomes assessed. Result In the first 24 hours after surgery, ACB was able to flex their knee ($P < 0.05$), and their overall drain output was also noticeably lower ($P < 0.05$). Using a Visual Analog Scale, the ACB group experienced less pain in the first 8, 24, and 48 hours ($P < 0.05$). Additionally, as compared to epidural analgesia, the administration of ACB resulted in a substantial decrease in LOS, total opioid intake, postoperative blood drain output, incidence of nausea and vomiting, and pain scores.[7]

In a study published in 2022, Xiaojuan Y et al. examined the effects of CACB & SACB in alliance with PCIA on pain management and inflammation in patients gone through TKA operation. Mean VAS scores, both at rest and during movement, were consistently lower in Group B compared to Group A across all postoperative hours. At POH30, the mean VAS score at rest was significantly lower in Group B (0.9 ± 0.4) than in Group A (1.1 ± 0.6) ($P = 0.048$). Similarly, the mean VAS score during movement was lower in Group B (2.2 ± 0.8) compared to Group A (2.6 ± 0.7) ($P = 0.001$). Additionally, the average number of patient-controlled bolus doses was 3.1 ± 1.3 (95% CI: 2.8–3.4) in Group B, which was significantly lower than the 4.3 ± 1.6 (95% CI: 3.9–4.6) recorded in Group A ($P < 0.001$). CONCLUTION: Which demonstrated that, in comparison to CACB, the combination of SACB and PCIA in the first two days after TKA had superior analgesic effects and more beneficial outcomes on inflammation and functional recovery.[8]

A study conducted by Bharath KK et al. (2021) involving 102 patients compared the effects of dexmedetomidine & clonidine as adjuvants to ropivacaine in USG aided ACBs. The results indicated that dexmedetomidine provided extended analgesia action, lesser pain levels & improved sedative outcomes compared to clonidine for post operation pain assurance following TKR interventions. Average period of analgesia was significantly elevated in the dexmedetomidine batch (Batch D) at 16.01 ± 0.5 hours, compared to 13.02 ± 0.5 hours in clonidine batch (Batch C) ($P < 0.0001$). At various time intervals (8, 10, 12, 14, 16, and 24 hours), the mean NRS scores were consistently less in Batch D than in Batch C ($P < 0.05$). Additionally, sedation scores were higher in Batch D, with peak sedation observed at the 2-hour mark, and better sedation levels maintained up to 4 hours in contrast to Batch C in contrast to Batch C [11].

A study conducted by Dong L et al. (2015) worked to found whether adductor canal block enhance superior quadriceps strength & same pain assure compared to femoral nerve block. A meta-analysis of nine studies involving 639 patients was conducted. They stated no significant differences in pain assessment at inactive state at 24 hours, ACB better conserved quadriceps muscle strength than FNB. Research studied ACB preserves quadriceps strength more effectively than FNB while providing same analgesic efficacy for post operation pain relief [10].

Sholahuddin & coworkers (2021) analyzed the application of ACB in cases who underwent knee surgery, involving 115 patients. The mean VAS scores before ACB on 5th, 7th, and 9th post procedural days were 7.4, 7.2, and 6.2, respectively. After the intervention, the VAS scores significantly decreased; however, the scores gradually increased until the 23rd day, after which they plateaued. The mean consumption of the analgesic (etoricoxib) was 102 mg, 96 mg, and 96 mg on the 5th,

7th, and 9th postoperative days, respectively. Analgesic consumption significantly decreased to 16 mg by the 15th postoperative day and gradually increased on the 17th, 19th, and 21st days. Only one patient reported a thigh hematoma following the ACB procedure. The study concluded that single-shot ACB, when administered in outpatient clinics, is a safe and effective intervention that significantly reduces both pain and analgesic consumption, potentially enhancing the postoperative rehabilitation process [11].

Tong QJ et al. (2018) investigated the benefits of combining ACB with local infiltration analgesia (LIA) for pain relief, conserving quadriceps power & promoting advance rehabilitation preceding TKA. The survey consisted 40 cases between January 2014 and October 2015. In LIA set, 75 mL of a local infiltration containing 150 mg ropivacaine, 30 mg ketorolac, 10 mg normal saline & 200 mcg adrenaline was used. In ACB group, 30 mL of 0.5% ropivacaine was injected postoperatively. It stated that ACB set required significantly reduced morphine consumption at 24 & 48 hrs contrast to LIA group. However, no secondary effect noted, indicating comparable functional recovery between the two groups. In conclusion, ACB revealed much effectiveness than LIA in lessen pain in initial 24-48 hours next to operation, while both methods provided similar functional outcomes in TKA patients.[19]

The functional recovery and analgesic effectiveness of FNB & ACB in unicompartmental knee arthroplasty were assessed and compared in a study by Simon HA et al. (2020). According to the study's findings, which included 80 patients, the FNB group's quadriceps strength was noticeably weaker than that of the ACB group, especially 12 hours post procedure. Thus analysis proved no discernible contrast in pain alleviation, ACB maintained quadriceps muscle strength better than FNB, leading to fewer falls. Thus, when utilized as an additional post procedure pain

administration preceding unicompartmental knee arthroplasty, ACB is regarded as a good substitute for FNB.[4]

After total knee replacement, Keith R. & workers analysed intraarticular analgesia with epidural plus FNB in a research including 90 patients. The findings demonstrated that the ropivacaine group had greater mean maximum VAS pain levels than the epidural group over the first 12 & 24 hrs preceding procedure. The two groups' pain values were comparable after a day. On operation day, ropivacaine batch consumed much more narcotics, but no notable difference in groups overall in-hospital narcotic usage. At no time point were there any appreciable variations in functional recovery. Although there were no differences in in-hospital falls, patients in the epidural batch showed elevated knee buckling & slower reactions than those in ropivacaine batch [12].

BASIC SCIENCES

Applied Anatomy

Ananaesthesiologist requires to have an accurate and in depth knowledge of the anatomy of vertebral column and its contents for a safe and successful administration of epidural anaesthesia, not only in terms of performance but also in terms of spread of drug in epidural space and level of block achieved.

Vertebral column

Main function of vertebral column is to protect the spinal cord. There are 33 vertebrae in vertebral column which includes

- Cervical - 7
- Thoracic - 12
- Lumbar - 5
- Sacrum - 5(fused)
- Coccyx - 4(fused)

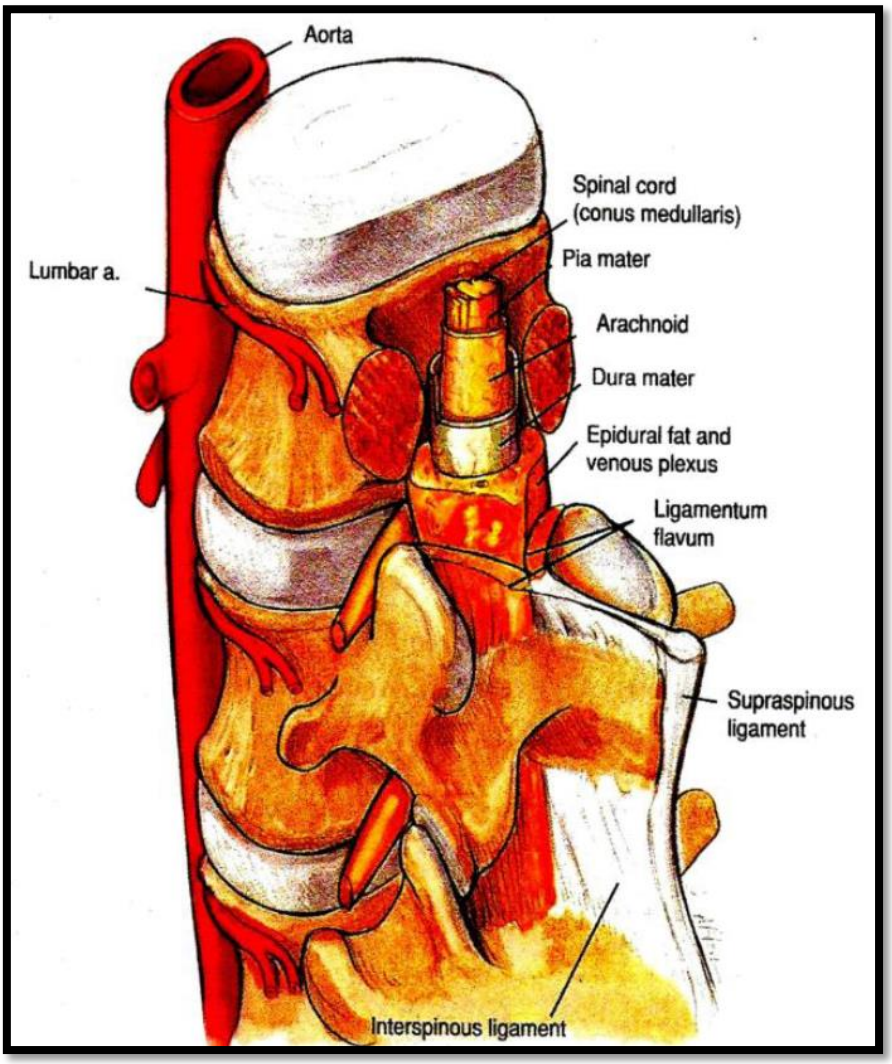
Curves of spine

In adults, curves of vertebral column have significant effect on spread of drugs in subarachnoid space and these curves are:[13]

- Cervical curve - Convexity anterior
- Thoracic curve - Concave anterior
- Lumbar curve - Convexity anteriorly

Cervical (C) five and lumbar (L) five are the highest points of cervical and lumbar curves in supine position and the lowest points of thoracic and sacral are at thoracic (T) five and sacral (S) two respectively.[13]

Figure 1: Vertebral Column



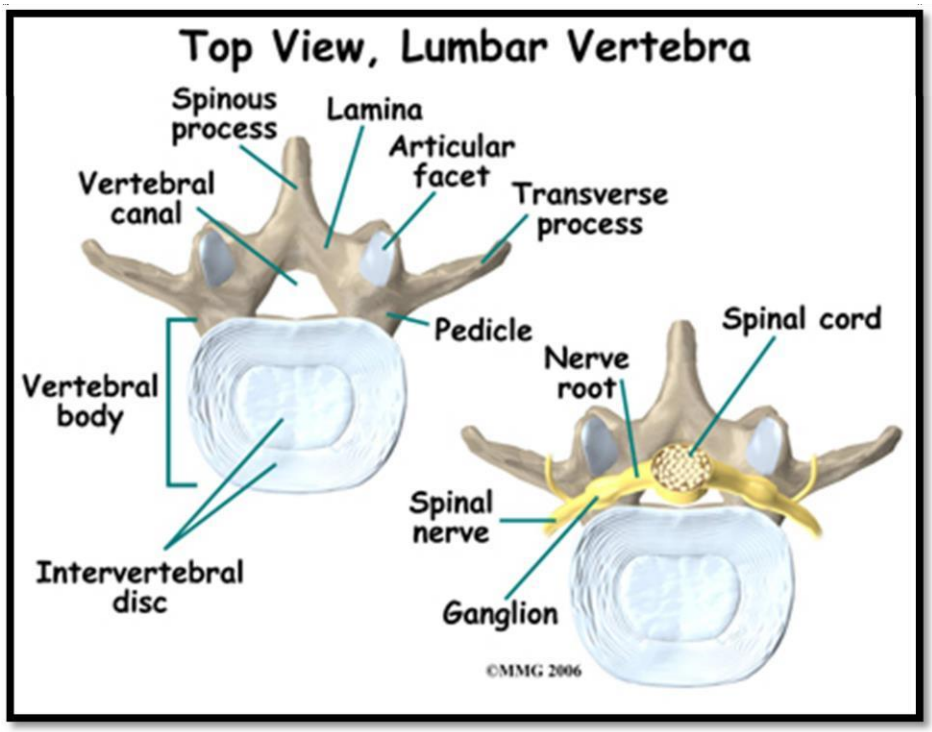
Lumbar vertebrae

Atypical lumbar vertebra consists of:

- A kidney shaped body.
- Two pedicles directed backwards from the upper part of the body.
- Two transverse processes

- Two laminaen meeting posteriorly and enclosing the triangular vertebral foramen.
- Thick, broad and quadrilateral spinous processes.
- Two upper and lower articular processes which prevent rotation but allow limited flexion and extension between contiguous vertebrae.

Figure 2: Typical lumbar vertebra



Thoracic vertebrae:

- A heart shaped body
- A small costal demifacet on superior border of lateral side of body and a larger demi facet on the inferior surface
- Shallow superior vertebral notches and deeper inferior vertebral notches
- Transverse processes are directed backwards and laterally, carrying a costal facet for articulation with ribs.

Vertebral ligaments

The following overlapping ligaments provide stability to the vertebral column and protect the spinal cord :

Supraspinous ligament: This is a strong fibrous cord which connects a pieces of spinous processes from sacrum to C_5 where it is continued as the ligamentum nuchae . The width depends upon the width of the spinous process– in lumbar region it might be upto 1 cm wide. In elderly people and manual labourers this ligament calcifies thus making the midline approach difficult.

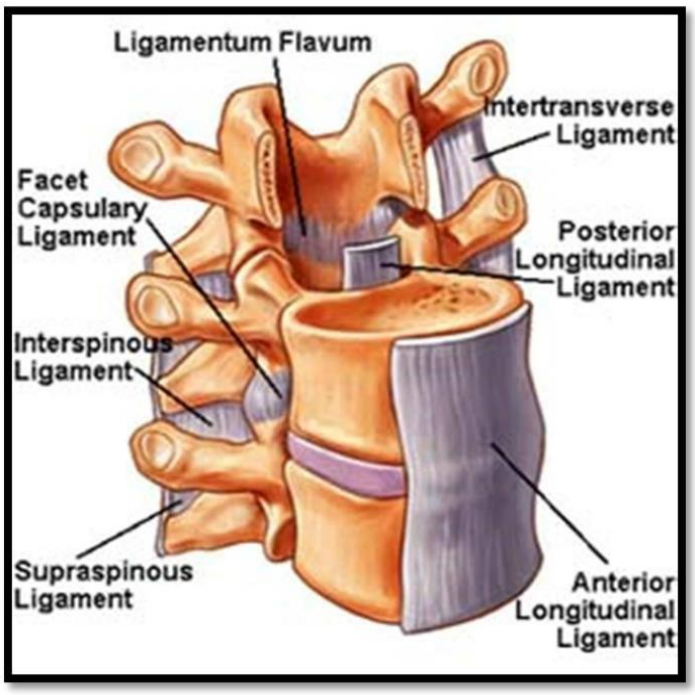
Interspinous ligament: This is a thin membranous ligament running obliquely and connecting spinous processes blending anteriorly with ligamentum flavum and posteriorly with supraspinous ligament. In the lumbar region, this ligamentis rectangular in shape leading to the characteristic and identifiable “loss of resistance” feel to air or saline.

Ligamentum flavum: This ligament comprises of yellow elastic fibres and connects adjacent laminae. Laterally, this ligament begins at the root of articular processes and extends posteriorly and medially to the point where laminae join to form spinous process. It provides the classic springy resistance in the lumbar region.

Longitudinal ligaments: There are two longitudinal ligaments (anterior and posterior) that bind vertebral bodies together.

For epidural anaesthesia, needle pierces the first three ligaments when midline approach is used, in paramedian approach only the ligamentum flavumis encountered.

Figure 3: Vertebral ligaments



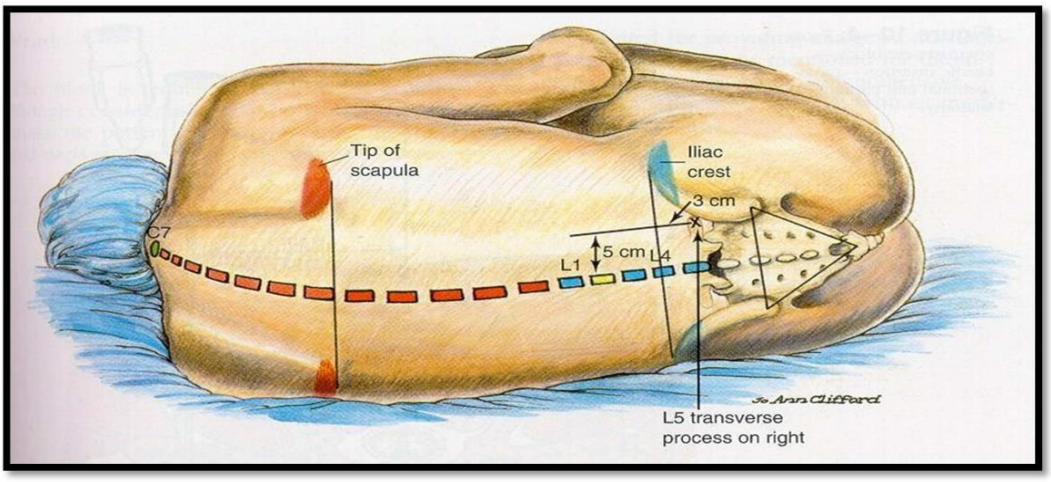
Intervertebral Discs[14]

These are principle connecting link between vertebral bodies. They form about 25% of the length of the spine. They consist of two parts – The outer fibrous part called the *annulus fibrosus* (made up of fibrous tissue), while the *nucleus pulposus* is the softer core. The discs serve as shock absorbers and lend flexibility to the vertebral column.

Topographical Line of Tuffier

This is a horizontal line across the back between the crests of the iliac bone passing over the spine of the 4th lumbar vertebra in the upright position. In a patient lying in the lateral position it may also pass through L4 and L5 interspaces. The superior iliac crest is used to identify the L4 and L5 interspace during epidural anesthesia.

Figure4: Topographical line of Tuffier



Vertebral canal:

The vertebral canal is bound by the vertebral bodies and intervertebral discs anteriorly, the laminae , ligamentum flavum and laterally by pedicles and laminae.

The contents of vertebral canal are as follows:

- Spinal cord
- Spinal nerve roots
- Meninges
- Cerebrospinal fluid
- Vessels
- Fat
- Loose areolar tissue

Spinal cord

The average length of the spinal cord in males is 45 centimetres (cms) and in females it is 42 cms. The average weight is approximately 30 gm.

The spinal cord is a continuation of the medulla oblongata below the level of foramen magnum and it tapers off into a conical extremity known as conus medullaris. Filum terminale descends to the back of first segment of coccyx from apex of conus medullaris.

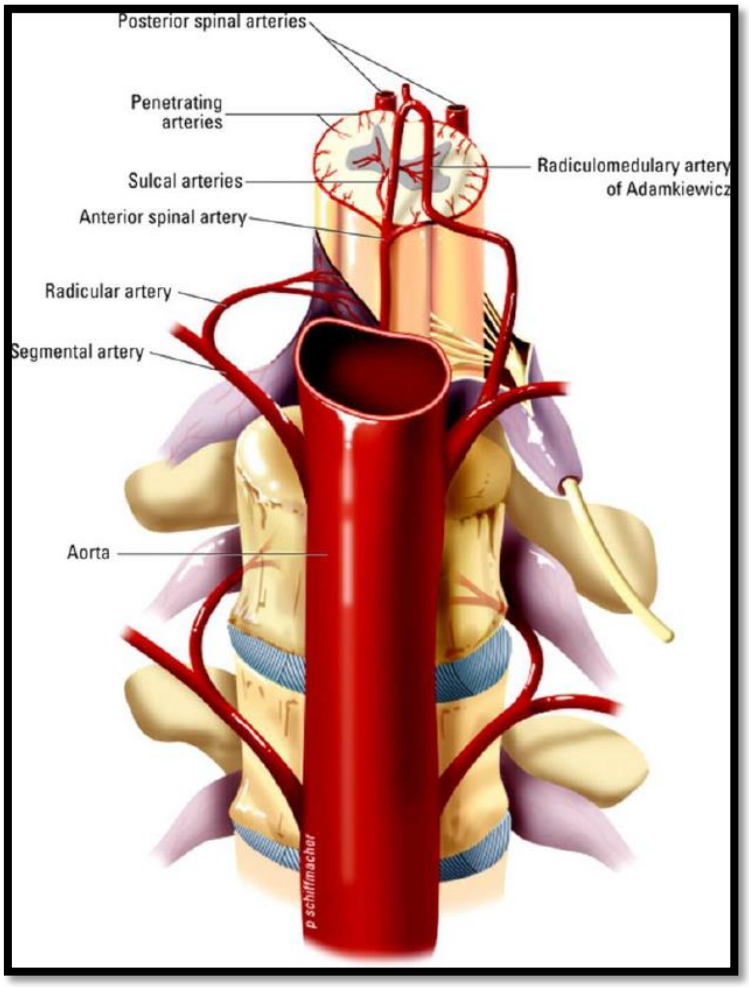
At birth, Spinal cord ends at the level of lower border of lumbar (L) three vertebra and in adults, it is as follows;

- LowerborderofL1
- UpperborderofL2
- UpperborderofL3

From the spinal cord arise 31pairs of spinal nerves, each made of a ventral and a dorsal root. These anterior and posterior roots after crossing the subarachnoid space, pass through the dura and extradural space independently and unite at the level of intervertebral foramen to form spinal nerve trunks, which further divide into anterior and posterior primary divisions.

The amount of white matter declines progressively from the cervical region down to the lumbar region. The gray matter is greatly increased in the both the lumbar and cervical enlargement.

Figure 5: Blood supply of spinal cord



Blood Supply of Spinal Cord:

The spinal cord receives its blood supply from anterior and posterior spinal arteries. The anterior spinal artery is a single vessel lying in front of the anteriormedian fissure. It is formed by two small arteries, one given off from each vertebral artery at the level of the foramen magnum. It receives small communications from the intercostal and lumbar arteries; to provide the extra blood supply needed in the cervical, thoracic and lumbar enlargements.

There are two posterior spinal arteries-one on each side. They are derived from the vertebral artery or more often from a primary branch of each vertebral artery.

They supply the posterior one-third of the spinal cord. This supply is augmented by spinal branches of vertebral, ascending cervical, posterior intercostals, lumbar and lateral sacral arteries, which pass through the intervertebral foramina.

Venous drainage is through a plexus of anterior and posterior veins in the neck, azygous veins in the thorax, lumbar veins in the abdomen ,and lateral sacral veins in the pelvis. There is no anastomosis between the anterior and posterior spinal arteries.

The longest of the feeder arteries is the radicularis magna (artery of Adamkiewicz), which supplies the anterior spinal artery in the area of the lumbar enlargement of the cord. It enters by way of a single intervertebral foramen (78% of the time on the left) between the T8 and L3 foramina.

Meninges

The spinal cord is covered by three membranes from in ward to outward, they are the pia mater, the arachnoid mater and the dura mater. The dural sac is the continuation of meningeal layer of the cranial dura mater. It is a circular sac or sleeve surrounding the spinal cord. Above, it is attached firmly to the circumference of the foramen magnum.

Duramater

It is the outermost membrane, the fibres of which run longitudinally. Although continuous, it can be described in two parts : the cranial and the spinal. The cranial dura consists has two layers, outer endosteal layer, which lines the skull, and an inner meningeal layer, which invests the brain and folds inward to form the falx cerebri and tentorium cerebelli.

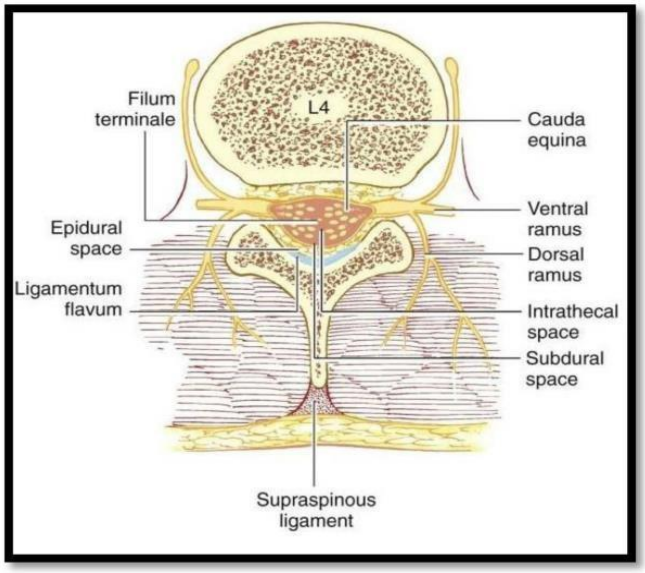
Arachnoid Mater

The arachnoid mater is a delicate non-vascular membrane applied closely to the dura mater. The lower extent of dural sac is as follows;

Below this the dura continues as the filum terminale. The subarachnoid space is the space between the arachnoid and piamater. This space is occupied by the cranial and spinal nerves and by the cobweb trabeculae. The space is annular in the cranial and thoracic vertebrae and is about three mm deep. Below the first lumbar vertebrae it is circular in shape.

EPIDURAL SPACE[15]

Figure 6: Epidural Space



Boundaries of the epidural space

The epidural space is bounded

Superior: by foramen magnum, where periosteal and spinal layers of dura mater fuse.

Inferior: by sacroccygeal membrane and sacral hiatus.

Anterior: by the posterior longitudinal ligament, vertebral bodies and discs

Posterior: by ligamentum flavum, periosteum of anterior surface of laminae and connecting ligaments.

Lateral: by periosteum of pedicles and intervertebral foramina.

Rarely, a fold of duramater divides the space into ventral and dorso – medial compartments leading to patchy or unilateral analgesia or missed segments.

Shape and size: These are largely determined by the shape of the lumbar vertebral canal and the position and size of the dural sac within it.

Cervical:1.5mm

Upper thoracic : 2.5 – 3 mm Lower thoracic : 4-5 mm Lumbar : 5-6 mm

Types of epidural space

The epidural space can be categorized into cervical, thoracic, lumbar and sacral epidural spaces. These spaces can be defined according to their margins. At the cervical epidural space, there is a fusion of the spinal and periosteal layers of dura mater at the foramen magnum to lower margin of the 7th cervical vertebra. While the thoracic epidural space is formed by the lower margin of C_7 to the upper margin of L_1, the lumbar epidural space is formed by the lower margin of L_1 vertebra to the upper margin ofS_1 vertebra. The sacral epidural space is formed by the upper margin of S_1 to sacrococcygeal membrane and sacral hiatus.

Contents of the epidural space:

Contains semi liquid fat , lymphatics , arteries ,loose areolar tissue spinal nerve roots and a very rich plexus of veins.

Fat

The epidural space is filled with semi fluid , lobulated fat tissue. Fat cells are also abundant in the dura that forms the sleeves around spinal nerve roots but they are not embedded within the laminae that form the dura mater of the dural sac. The fat in the epidural space buffers the pulsatile movements of the dural sac and protects nerve structure, creates a reservoir of lipophilic substances, and facilitates the movement of the dural sac over the periosteum of the spinal column during flexion and extension. The areolar tissue of this space has a very rich blood supply with small capillaries forming a network in its substance . Drugs stored in fat, inside dural sleeves, could have a greater impact on nerve roots than drugs stored in epidural fat, given that the concentration of fat is proportionally higher inside nerve root sleeves than in the epidural space, and that the distance between nerves and fat is shorter. Similarly, changes in fat content and distribution caused by different pathologies may alter the absorption and distribution of drugs injected in the epidural space. The maximum amount of fat is present posteriorly, where it assumes triangular capsular shapes and is linked to the midline of the ligamentum flavum by a vascular pedicle. Drugs with thoracic epidural space is formed by the lower margin of C_7 to the upper margin of L_1, the lumbar epidural space is formed by the lower margin of L_1 vertebra to the upper margin ofS_1 vertebra. The sacral epidural space is formed by the upper margin of S_1 to sacrococcygeal membrane and sacral hiatus.

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The anterior epidural space is entirely occupied by a rich venous plexus (valveless system of veins). The plexus communicates with the intracranial sigmoid sinus, basilar venous sinus, basi vertebral vein, occipital vein, and the azygous system. The plexus is linked to the abdominal and thoracic veins by the intervertebral foramina and through this connection transmit intra-abdominal and intra-thoracic pressure to the epidural space. The venous plexus is also connected to the iliac veins through the sacral venous plexus. Obstruction of the inferior vena cava, advanced pregnancy or intra abdominal tumor scan cause distension of the venous plexus leading to an increased risk of being traumatized during needle and/or catheter

placement in the epidural space. These veins are more prominent along the lateral wall of the vertebral canal usually they are out of reach of a correctly placed needle by midline approach. The dose and rate of local anaesthetic should also be reduced in any case of increased intraabdominal pressure / inferior vena cava obstruction as the resultant engorgement of the venous plexus would reduce the effective volume of the epidural space. The injected drug may therefore spread rapidly upwards or downwards along the epidural space.

Epidural arteries

The epidural arteries located in the lumbar region of the vertebral column are branches of the ilio-lumbar arteries. These arteries are found in the lateral region of the space and therefore accidental puncture is uncommon by midline approach.

Spinal arteries:

As already discussed, the spinal cord is supplied by one anterior spinal and two posterior spinal arteries. The spinal branches of the subclavian, aortic and iliac arteries cross the epidural space on the way to sub arachnoid space. The largest of them, the artery of Adamkiewicz supplies the anterior spinal artery at the lumbar level. This artery enters the epidural space between $T_8 – L_3$ levels and any damage to it would cause ischaemia of entire lumbar region of the cord. In general, anterior spinal artery is more susceptible due to it being unpaired.

Pharmacokinetics of Epidural Blockade

Epidural anaesthesia results from the interaction of local anaesthetics with nerve structures located within the epidural space. Local anaesthetics can reach the sites of action along various distribution pathways. Uptake into extra neural tissues

like epidural fat and systemic absorption compete with neural tissue distribution thereby affecting the clinical potency and duration of action. Therefore, epidural doses of local anaesthetics are much higher than spinal doses.

Specifically, drugs may

1. Exit the intervertebral foramin at or each the paraspinous muscle space,
2. Drugs may diffuse into epidural fat,
3. Drugs may diffuse into ligaments and finally,
4. Drugs may diffuse across the spinal meninges.

The only mechanism by which drugs redistribute from the epidural space to the spinal cord is diffusion through the spinal meninges and the cellular arachnoid mater is the principal meningeal barrier to diffusion accounting for 95% of the resistance to meningeal permeability.

Meningeal permeability is not the only determinant of a drug spinal cord bioavailability after epidural administration. Drugs can partition into various environments in the epidural space and be unavailable for transfer across the spinal meninges.

Lipid soluble drugs have a tendency to get sequestrated into epidural fatty tissue. The dura mater is an important site of drug clearance especially in humans where dura mater is a highly vascular structure. As lipid soluble molecules traverse capillaries more readily than do more hydrophilic molecules, lipid soluble drugs may be cleared by this mechanism more readily than less lipid soluble drugs.

Meninges contain multiple enzyme systems, which are capable of drug metabolism. In addition, the meninges express enzymes capable of metabolizing

neurotransmitters, including epinephrine, norepinephrine, acetylcholine and neuropeptides. After epidural administration, local anaesthetics need to cross the spinal meninges to reach their site of action

Epidurally administered drugs that reach the CSF, also can diffuse back across the meninges into the epidural space, but this happens only when the drug concentration in the epidural space falls below that in the CSF. Diffusion is dependent mainly on the drug's physicochemical properties, particularly, lipid solubility.

Physiological Effects Of Epidural Blockade

The physiological responses to epidural anaesthesia are mainly due to sympathetic blockade accompanied by sensory and motor blockade to various degrees. Some of the most important (but not all) physiological effects of epidural blockade can be discussed in relation to either sympathetic blockade of vasoconstrictor fibres (below T_4) and/or of cardiac sympathetic fibres. Major sympathetic blockade can be avoided by trying to keep the block level around or below T_{10}.Lower abdominal, urologic, gynaecological and lower limb surgeries scan be carried out satisfactorily with acceptable sympathetic blockade.

Zone of differential blockade:

Erlanger and Gasser showed that action of local anaesthetics on nerve fibres is by "differential conduction blockade". The nerve fibres are of three types viz A, B , C A minimum length of myelinated nerve fibres should come in contact with local anaesthetic for conduction blockade. In myelinated fibres, the blockade occurs at nodes of Ranvier and three consecutive nodes need to be blocked for impulse conduction to be completely interrupted.

All types of nerve fibres are affected by local anaesthetics. but within any one fibre type, there is tendency for small, slower conducting fibers to be more readily blocked than large, fast conducting fibres. Between fibre type showever, these rules do not hold good. Myelinated preganglionic B fibres which have a faster conduction time are about three times more sensitive to local anaesthetics than the slower non-myelinated post ganglionic C fibers.

Sensory Aα fibres appear to be more sensitive to blockade than motor A_β fibres, although of the same conduction velocity, this may be because sensory fibres conduct at a higher frequency. It has been suggested that this selectivity for sensory fibres exhibited by Bupivacaine and Ropivacaine is a function of frequency dependent block.

Sensory

In intradural block sympathetic fibres are blocked two or three segments higher than sensory fibres. In extradural block, the relationship is complex. Level of sympathetic block is the same as (or lower than) sensory with epidural blockade. Sympathetic block will be greater when more concentrated solutions are used or when adrenaline added, as this has similar effect.

Motor

In intradural block, the difference between sensory and motor block is slight(two segments). In extradural block, the difference in levels is greater, depending on nature of local anaesthetic solution.

Factors Influencing Height And Distribution Of Local Anaesthetic:

Patient characteristics:

- Age: Study done by Bromage shows a correlation between age and dose, an increase in dose from age 4-18 years followed by a gradual decrease from 19 year onwards.
- Height: A simple thumb rule is to use 1ml per segment for height of 150 cm and then add 0.1 ml per segment for each 5 cm over 150 cm.
- Weight: Under normal circumstances, there is not much correlation between spread of analgesia and the weight. However in morbidly obese patients a given dose of local anaesthetics can cause a higher than normal block due to compression of epidural space due to increased intra-abdominal pressure.
- Intra-abdominal pressure: epidural venous engorgement in pregnancy, obesity, tumours can cause a higher blockade with a given dose due to narrower epidural space
- Posture: In sitting position there is slight propensity of the drug to spread caudally and higher doses may be required.
- Gender

Technique of injection:

- Site of injection: Rapid onset and denser blockade is seen when the point of injection was nearer to nerve roots. Lumbar epidural injection has a better cephalad spread than caudal epidurals.

- Direction of bevel
- Rate of injection: A rapid injection of local anaesthetic produces a rapid but in complete and more extensive block. Injection rate of 0.3–

  0.75ml/sec results in most reliable block.

Characteristics of Anaesthetic Solution:

- Amount: Earlier epidural anaesthesia was considered to be equivalent to multiple paravertebral blocks and the tendency was to give a large volume of diluted drug. However studies by Bromage showed that increasing dosage linearly increases the degree of sensory blockade.
- Concentration: An increase in the drug concentration increases the density of motor blockade.
- Density
- Temperature
- Use of adjuvants

Effects Of Epidural Anaesthesia On Various Organ Systems:

Cardiovascular System:

The action of epidural anaesthesia on cardiovascular system depends on the level of block:

1. If the level of block is below T_4 there is dilation of resistance and capacitance vessels due to loss of sympathetic tone. This causes a fall in BP. However if there is a blockade of cardiac efferent sympathetic fibres

from $T_1$to T_4 there is a loss of chronotropic and inotropic drive resulting in a fall in cardiac output.

2. The activation arterial or Bainbridge reflex causing bradycardia -The lowering of blood pressure in the right atrium consequent to diminished venous return [Bainbridge (1874-1921) effect]

3. The operation of Mary's law causing tachycardia.

4. Depression of vascular smooth muscle and β adrenergic blockade of myocardium with fall in cardiac output.

Block not extending above T4 is not always associated with fall of blood pressure in fit young adults. However, elderly may suffer significant hypotension when moderate volumes are injected into the epidural space.

Slowing heart rate is caused if any of the anterior roots carrying sympathetic cardiac accelerator fibres are blocked (T_1– T_4). Activation of Bainbridge reflex may further contribute to bradycardia which is more frequent than tachycardia.

Theories of causation of fall in blood pressure

1. Diminished cardiac output consequent on reduction of venous return to heart due to failure of peripheral pump – calf muscles.

2. Dilatation of post arteriolar capillaries and small venules due to paralysis of vasoconstrictors, compensatory vasoconstriction takes place in areas not anaesthetized via carotid sinus reflexes. In high spinal blocks, majority of vasoconstrictor fibres including those to arm (T2-T10),are paralyzed, hence low blood pressure.

3. Paralysis of sympathetic nerve supply to heart T_1-T_4. Bradycardia may give rise to fall in cardiac output.

4. Paralysis of sympathetic nerve supply to adrenal glands splanchnic nerves, with consequent catecholamine depletion.

5. Absorption of drug into circulation. Seen more commonly with epidural blockade due to the larger volume of drug used.

6. Pre-existent hypovolemia, if present, may cause precipitous hypotension after central neuraxial blockade. Compression of great vessels within abdomen, by the pregnant uterus, abdominal tumours or abdominal packs may cause severe hypotension in presence of central neural blockade.

Respiratory System:

The phrenic nerve supplying diaphragm arises from the anterior roots of C_3, C_4, C_5 and should not be encroached upon during neuraxial blockade. Lumbar and even mid thoracic epidurals usually do not cause much effects on respiratory system. During epidural anaesthesia, breathing becomes quiet and tranquil. This is not only due to motor blockade, but also to differentiation with reduction of sensory input to respiratory center.

The ventilation perfusion during extradural block is not greatly altered and effects on respiratory functions are relatively small with no effect on FRC or V/Q ratio. The lung volumes and capacities (tidal volume, vital capacity) are basically unchanged during epidural anaesthesia. Abdominal muscle and intercostals muscle paralysis is compensated by diaphragm moving down. The pulmonary gas exchange is preserved.

The patient may stop breathing so that respiratory support by IPPV and, if necessary, the tracheal intubation maybe required. Causes may be:

- Inadequate medullary blood flow due to inadequate cardiac output-a serious situation demanding immediate cardiorespiratory support.
- Massive epidural spread.
- Accidental subdural injection
- Toxic effects of local analgesic drug.
- Injecting narcotic analgesic drugs

Gastrointestinal System:

Pre ganglionic sympathetic fibres from T5 to L1 are inhibitory to gut, there is no effect on oesophagus, the innervations of which is vagus. The small gut is contracted as the sympathetic inhibitory impulses are removed, the vagus being all powerful, Sphincters are relaxed and peristalsis is active although not more frequent. Pressure within the bowel lumen is increased.

Nausea and vomiting due to the hypotension may occur in upto 20% of patients and usually come on in waves-lasting a minute or so and then passing away spontaneously. Stimuli arising in the upper abdomen might not be blocked causing discomfort. Colonic blood supply and oxygen availability are increased, perhaps an important factor in the prevention of anastomotic breakdown following gut resection.

1. Theories of causation of nausea and vomiting:

a. Hypotension: corrected using fluid boluses and vasopressor drugs

b. Increased peristalsis

c. Traction on nerve endings and plexuses, especially via vagus (usually upper abdomen)

d. Presence of bile in stomach due to relaxation of pyloric and bile-duct sphincters

e. Narcotic analgesics (premedication)

f. Psychological factors

g. Hypoxia

Liver

There are no specific effects of significance. The degree of hypotension that compromises liver function is not known. Liver disease may interfere with the metabolism of local anaesthetic drugs.

Endocrine system

Surgical stress produces a variety of changes in endocrine system and metabolic function. There is an increased catabolism of proteins and oxygen consumption. Increased plasma concentrations of catecholamines, vasopressin, growth hormones, renin, angiotensin, glucose, Anti diuretic hormone (ADH) and Thyroid Stimulating Hormone (TSH) are noted and this is referred to as surgical stress response.

Neuraxial blocks in general suppress the increase of ADH. It also delays adrenal response to trauma, whereas operations under GA cause a rise in steroids.

In any case, either regional or general, there is no difference in the postoperative period once the effects of the block are discontinued. Spinal block suppresses the hyperglycemic response to surgery and stress and so is useful in diabetic patients but this does not extend into postoperative period. The response to insulin is augmented and anaesthetist should be aware of possibility of hypoglycemia.

Epidural block prevents lymphopenia and granulocytosis after operation, thus inhibiting the metabolic endocrine response to surgery and preventing immune depression.

Adductor canal- Saphenous nerve

Anatomy-

The saphenous nerve is a terminal sensory branch of the femoral nerve. It supplies innervation to the medial aspect of the leg down to the ankle and foot. It also sends infrapatellar branches to the knee joint.

A saphenous nerve block is useful as a supplement to sciatic nerve block for foot and ankle procedures that involve the medial aspect of the malleolus and the foot. The saphenous nerve block use as a supplement to multimodal analgesia protocols in patients having knee arthroplasty. Typically, a more proximal (mid-thigh) approach and a larger volume of local anesthetic is used for this "adductor canal nerve.

Indications: saphenous vein stripping or harvesting; supplementation for medial foot/ankle surgery in combination with a sciatic nerve block, and analgesia for knee surgery in combination with multimodal analgesia.

Contraindications Absolute

- Active or latent (less than 1 year) knee sepsis
- Presence of active infection elsewhere in body
- Extensor mechanism dysfunction
- Medically unstable patient

Relative

- Neuropathic joint
- Poor overlying skin condition
- Morbid obesity
- Noncompliance due to major psychiatric disorder, alcohol, or drug abuse
- Insufficient bone stock for reconstruction
- Poor patient motivation or unrealistic expectation
- Severe peripheral vascular disease

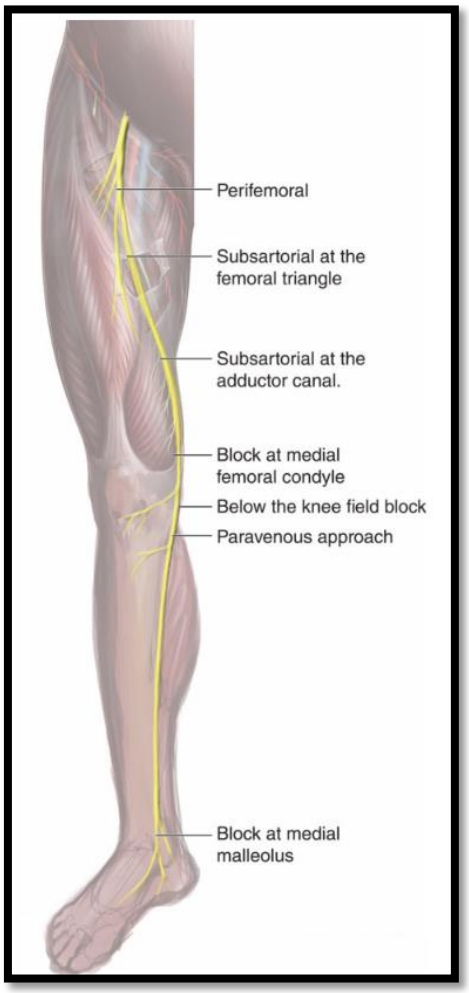
Adductor cannal block Block Under Ultra-Sound Guidance

The sartorius muscle descends in a lateral to the medial direction across the anterior thigh and forms a “roof ” over the adductor canal in the lower half of the thigh. The muscle appears as a trapezoid shape beneath the subcutaneous layer of adipose tissue.

The sides of the triangular canal are formed by the vastus medialis laterally and the

adductor longus or magnus medially (depending on how proximal or distal the scan is). The saphenous nerve is typically imaged by ultrasound as a small, round, hyperechoic structure anterior to the artery. The femoral vein accompanies the artery and saphenous nerve, which all can be identified at a depth of 2–3 cm

Figure 7: Various approaches to the saphenous nerve block



TECHNIQUE

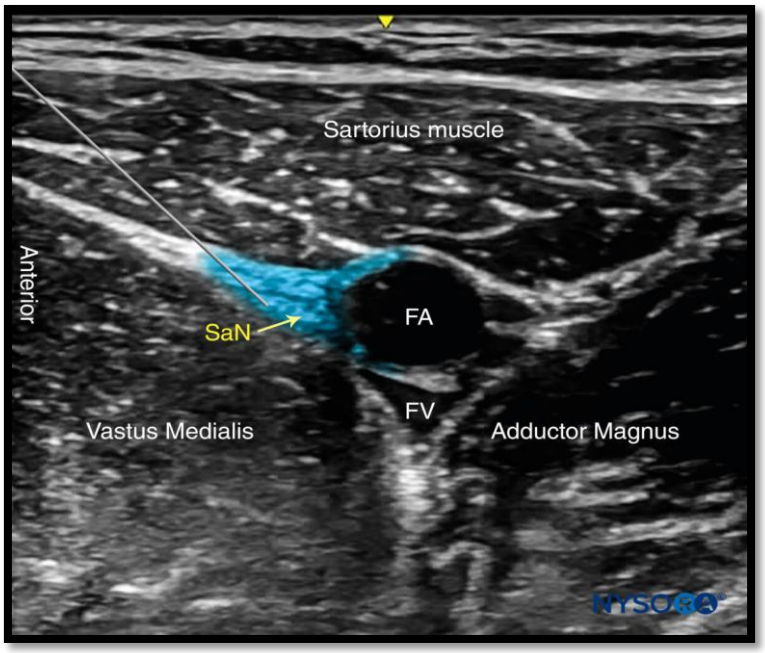
The skin is disinfected and the transducer is placed anteromedially, approximately at the junction between the middle and distal third of the thigh.

The saphenous nerve block should be performed at the most distal level where the artery still lies immediately deep to the sartorius muscle, thus minimizing the

amount of motor nerve block of the vastus medialis; an adductor canal nerve block is typically performed more proximally, around the mid-thigh level. The needle is inserted in-plane in a lateral-to-medial orientation and advanced toward the femoral artery.

Once the needle tip is visualized anterior to the artery and after careful aspiration, 1–2 mL of local anesthetic is injected to confirm the proper injection site. When injection of local anesthetic does not appear to result in its spread around the femoral artery, additional needle repositions and injections may be necessary.

Figure 8: USG – guided Adductor canal block



Knee arthroplasty

Knee arthroplasty is a reconstruction of the knee joint. It is more commonly referred to as a total knee replacement and is a very reliable procedure with predictable results. Total knee arthroplasty (TKA) is an excellent treatment option for individuals with symptomatic osteoarthritis in at least 2 of the 3 compartments of the knee and who have failed conservative treatment. Additionally, partial knee arthroplasty (PKA) is an excellent treatment option for individuals with symptomatic

osteoarthritis localized to 1 compartment of the knee and who have failed conservative treatment.35 The primary goal of either surgery is durable pain relief with the improvement of functional status.

Anatomy and Physiology

The knee is a synovial hinge joint with minimal rotational motion. It is comprised of the distal femur, proximal tibia, and the patella. There are 3 separate articulations and compartments: medial femorotibial, lateral femorotibial, and patellofemoral. The stability of the knee joint is provided by the congruity of the joint as well as by the collateral ligaments. The capsule surrounds the entire joint and extends proximally into the suprapatellar pouch. Articular cartilage covers the femoral condyles, tibial plateaus, trochlear groove, and patellar facets. Menisci are interposed in the medial and lateral compartments between the femur and tibia which act to protect the articular cartilage and support the knee

Ultrasonography[16-18]

Ultrasound waves are sound waves with a frequency greater than 20,000Hz. These frequencies are above the audible upper limit of human hearing. Medical ultrasound is the application of this ultrasound waves to visualize the internal organs of our human body. The frequencies used for this purpose, ranges from 3 to 20 MHz. In recent years, ultrasound is widely used in anaesthesia for obtaining vascular access and performing peripheral nerve blocks. Ultrasound guided techniques helps in increasing success rate and reduce its complications.

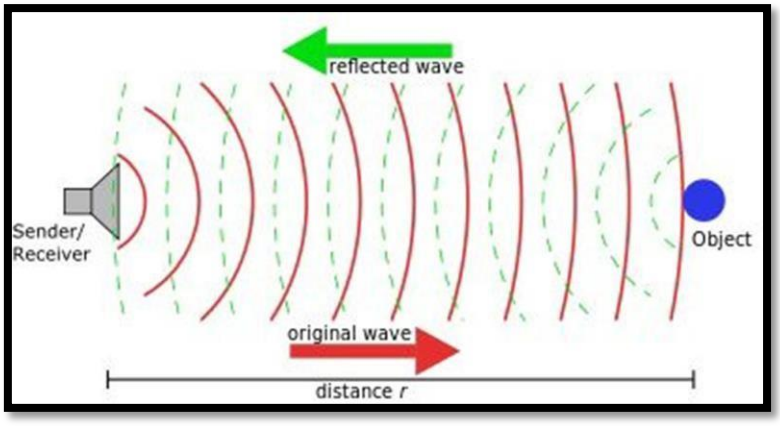
Ultrasound Pulse Generation

The ultrasound transducer contains multiple piezoelectric crystals which are interconnected electronically. When mechanical energy is applied to these crystals and some ceramics, they generate electrical energy. This phenomenon known as the "Piezoelectric Effect" was first described by the Curie brothers in 1880. They also described the "Reverse Piezoelectric effect", where in application of electricity to these crystals produced vibrations which generate ultrasound waves.

Ultrasound Wavelength and Frequency

The wavelength and frequency are inversely related. High frequency ultrasound waves (10 to 20 MHz) give images with a high axial resolution but are more attenuated as we go deeper. Therefore, these transducers are optimal to image the superficial structures. Low frequency ultrasound waves (2 to 8 MHz) penetrate deeper but provide low axial resolution and are used to image deeper structures.

Figure 9-Principles of Ultrasonography



Ultrasound Tissue Interaction:

As the ultrasound waves travel through tissues, they are partly transmitted to deeper structures, partly reflected back to the transducer as echoes, partly scattered, and partly transformed to heat.

Reflection

For image generation, the echoes returned after hitting a tissue interface is of interesttous. The amount of echo returned after hitting a tissue interfaceis determined by a tissue property called acoustic impedance. The intensity of a Reflected echo is proportional to the mismatch in acoustic impedances between two mediums.

Refraction

The change in the direction of the ultrasound waves after hitting an interface between two media with different velocities of sound transmission is refraction. This causes artefacts as the returning echoes are incorrectly located.

Scattering

Ultrasound waves which incident on the tissues at right angles are reflected back to the transducer. If the waves are not at right angle, then there turning echoes are scattered in all directions in a non-uniform manner

Absorption

Some of the ultrasound waves are absorbed by the tissue and are converted to heat.

Attenuation

As the ultrasound waves travel through tissue, the returning echoes will become weaker due to absorption, scattering and refraction.

Diffraction

The spreading out of the ultrasound waves as its moves further away from the source is diffraction.

Construction

The ultrasound probe has an array of individual transducers which acts as both a transmitter and a receiver. Each transducer emits a short burst of ultrasound and is quiescent until it detects the echoes returning. This is called “Pulsed Ultrasound”. The speed of ultrasound in our body tissues is fairly constant at a speed of 1540m/s. The time taken for an echo to return is used determine the distance between the tissue and the probe.

Across the plane of an image, the ultrasound image is swept to form two dimensional images one line at a time. These lines are then summated to produce a frame. The frames are repeated to produce a real-time image. The brightness of the image depends upon the amplitude of the returning echo from the anatomical interfaces.

Scanning Modes

A-mode (amplitude mode): This displays a single echo signal against time to measure depth.

B-mode (brightness mode): It is a two dimensional image produced using an array of transducers and a series of reflected echoes.

M-mode(motionmode) : is a specialized type of B-mode imaging where one particular line is ensonified repeatedly to examine a moving structure plotting out how the structure moves with time.

Ultrasound controls

Gain alters the brightness of the image by amplifying the received signal.

Time-Gain Compensation (TGC) differentially amplifies signals from different depths, allowing equal amplitudes from all depths to be displayed.

Focus adjusts the beam to be at its narrowest at the required depth to image the region of interest. It thereby improves lateral resolution

Depth can be adjusted to have the structure that is being examined to be in the centre of the screen.

Approaches and techniques

There are two basic approaches to ultrasound guidance. With the out-of- plane technique, the needle tip crosses the plane of imaging as an echogenic dot. With the in- plane approach, the entire tip and shaft of the advancing needle are visible.

Out-of-plane:

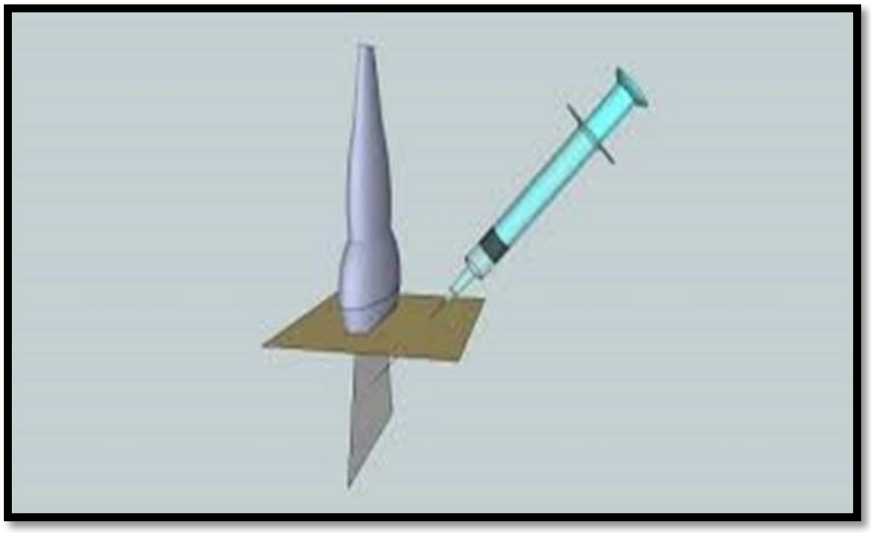
This technique involves insertion of needle at the midpoint of probe such that the needle cuts across the ultrasonic beam. The image obtained is a cross section of the needle shaft or tip. Path to target is shorter as compared to in-plane technique, but visibility of needle is not optimum, indirect markers like tissue movement or hydro dissection is needed to confirm placement.

1) Most similar to other approaches to regional block (nerve stimulation or palpation)
2) Shorter needle path than within-plane approaches
3) Along the nerve path(catheters)

Disadvantages:

Unimaged needle path, crossing the plane of imaging without recognition.

Figure 10-Out of plane approach



In-plane(IP):

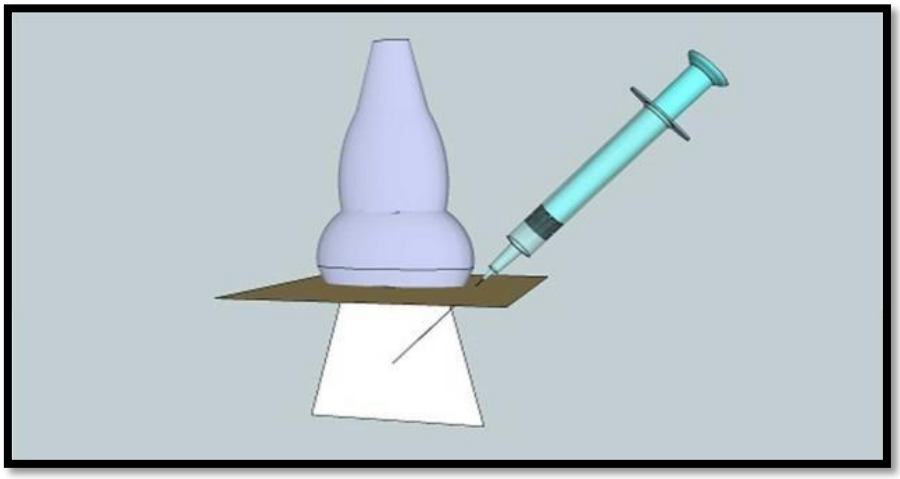
In this technique needle is inserted along the length of ultrasound probe. It aligns the entire length of the beam with the shaft of needle. The image displayed will depict the entire needle shaft and its tip thereby improving the precision of nerve blocks. But the needle visibility depends on angle of insertion and the needle traverses a longer path to reach the target area.

Most direct visualization.

Disadvantages:

1. Partial line-ups (creating a false sense of security when the needle tip is not correctly identified.
2. Some unimaged needle path occurs with IP approach, but typically less than with OOP approach.
3. Longer paths and therefore more structures to cross with the block needle.

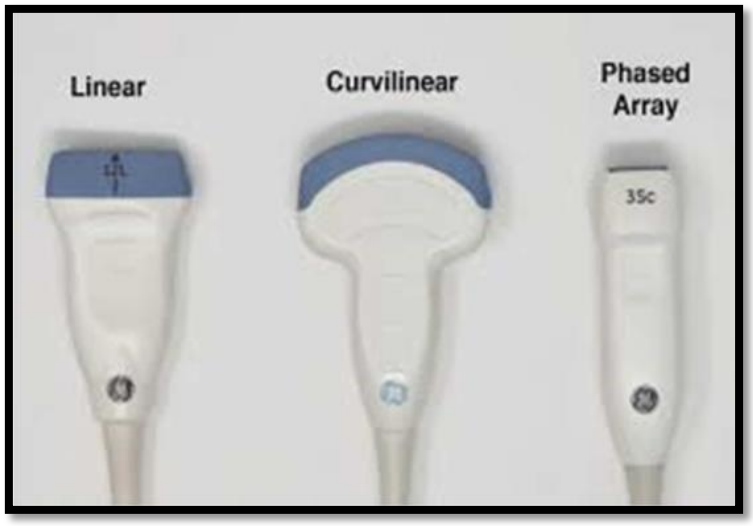
Figure 11-In plane approach of ultrasonography



Ultrasound probes

- Commonly used are three types- Linear high frequency (6 to 12 MHz) probes which has high resolution and lesser penetration and is ideal for visualizing superficial structures.
- Curvilinear low frequency probes(2-5MHz)which has lower solution, higher penetration and is ideal for deeper structures like intraabdominal organs.
- Phased Array Probe also has low frequency (2MHz – 7.5MHz) gives a large depth with a small acoustic window, ideal for chest ultrasound

Figure 12-Ultrasoundprobes



Imaging

Ultrasound image is produced by echoes received as the Ultrasonic beam interacts with the tissues it travels through. Acoustic impedance of a structure is the function of the elasticity and density of the particular tissue. Materials with higher acoustic impedance transmit sound faster, and do not allow for continued compression by the impending wave. The sound beam is attenuated while traversing various tissues within the body. The beam will be scattered somewhat when it encounters varying tissues on the way with different acoustic impedancesoritmay be reflected back from structures and returns back to the transducer. Refraction and absorption by tissues may also attenuate the waves. Those tissues that reflect the wave are termed echoic and those which do not reflect the wave are termed anechoic. Always use plenty of sterile ultrasound gel to remove the air interface between the skin and probe. Air does not allow the passage of the ultrasound beam even though it has low Acoustic impedance. Bone has high acoustic index so it appears to be white on the ultrasound image as it is hyper reflective to the beam. Blood and other fluids appear to be black on the image since they are anechoicin nature. Soft tissue appears as grey on the sonographic image as they have medium echogenicity.

The nerves appear round or oval in transverse view and are hypo-echoicorthey appear as honeycomb structures with septations inside them. Nerves are bordered by a hyper-echoic layer of connective tissue. Blood vessels will appear as circular hypoechoic to anechoiec structures with a well-defined hyper- echoic border which is the vessel wall. Veins are compressible with thinner walls where as arteries have thicker walls and appear pulsatile in nature. Muscles have fibrous-lamellar texture and appear as heterogeneous or homogeneous hypoechoic structures with hyper-echoic septa in between.

Basic principles of ultrasound guided nerve blocks.

- First involves the identification of anatomical structures like muscles, fascia, blood vessels and bones.
- Visualization of the nerve plexus or the fascial plane where drug should be deposited.
- Should be able to differentiate between normal and altered anatomy of the region scanned.
- Identify the correct plane for needle insertion to avoid trauma to vessels
- Strict a septic technique
- Real time visualization of needle when it is inserted inside.
- -Once the target is reached, inject a small volume of drug or saline and see the spread and confirm location, else reposition the needle
- Do frequent aspiration during injection of drug to rule out intravascular injection.
- Complete visualization of the spread of total volume of local anaesthetic drug injected.
- Always keep ready all resuscitation equipment, drugs and standard monitoring.

ROPIVACAINE

Introduction

Ropivacaine is a newer, longer acting local anaesthetic agent which belongs to the amino amide group. It was first synthesized by Ekenstam in 1957; however it was first introduced for clinical practice only since1996.Chemically it belongs to the same group as bupivacaine and mepivacaine (epipecoloxylidide local anaesthetic).

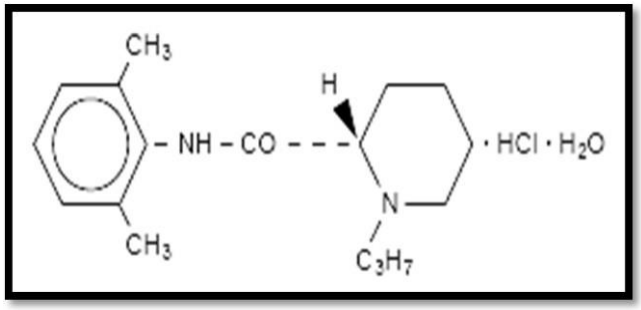
It was found that butyl derivatives of pipecoloxylidides (example bupivacaine) were more cardiotoxic than propyl derivatives, causing a significant number of cardiac arrests.[19]

Thus ropivacaine was developed as a pure S – enantiomeric form of pipecoloxylidides. Though ropivacaine has been available internationally forover three decades, it is a relative new entrant in the Indian market.

It is becoming increasingly popular among anaesthesiologists and has been used extensively in almost all modes of regional anaesthesia: infiltration, peripheral nerve blocks, spinal anaesthesia, epidural anaesthesia as well as caudal epidural blocks in paediatric patients.

Chemical Structure

Figure 13:Chemical structure of ropivacaine



Ropivacaine is an amino amide local anaesthetic agent, chemically described as S-(-)-1-propyl-2',6'-pipecoloxylidide hydrochloride monohydrate. The ***International Union of Pure and Applied Chemistry*** name is (S)-N-(2,6-dimethylphenyl) -1- propylpiperidine-2-carboxamide. It's molecular formula is $C_{17}H_{26}N_2O•HCl•H_2O$ and it has a molecular weight of 328.89.

Ropivacaine is a white crystalline powder. At 25°C ropivacaine hydrochloride has a solubility of 53.8 mg/mL in water and a distribution ratio between n-octanol and phosphate buffer at pH 7.4 of 14:1. The pKa of ropivacaine is 8.07which is very similar to that of bupivacaine (8.1) .

However, ropivacaine has a much lesser lipid solubility as compared to bupivacaine and mepivacaine. This can be explained on the basis of presence of apropyl (3 Carbon) side chain in ropivacaine as compared to a butyl (4 Carbon) side chain in the other two local anaesthetics. This lower lipid solubility ***Physical Properties*** of ropivacaine has a significant effect on the block characteristics of ropivacaine as discussed ahead.[20]

Mechanism of Action And Corelation With Structure

Ropivacaine reversibly inhibits the voltage gated sodium channels present on the nerve cell membranes thus preventing the influx of sodium ions into the cells. This:

I. Blocks generation and conductance of nerve impulses.

II. Slows propagation of nerve impulses

III. Reduces the rate of rise of action potential

Almost all local anaesthetic agents block the unmyelinated C and myelinated Aδfibres, which transmit pain impulses, at the same rate.

The rate of blockade of motor fibres (Aα and Aβ), however depends upon the physio chemical properties like pKa and lipid solubility of the individual drug. As ropivacaine is less lipid soluble than bupivacaine, the Aα and Aβ blockade is slower and hence motor blockade is less potent. Studies of lumbar epidural block in humans

have confirmed that equal volumes and concentrations of bupivacaine and ropivacaine produce similar degree of sensory block but the motor block produced by ropivacaine is slower in onset, lesser in intensity and shorter in duration.

Clinically the order of blockade of nerve fibres is autonomic, sensory and motor, while the regression of the block occurs in reverse order.

The nerve impulse transmission is lost in the following order:

The order of the loss of nerve function is

1. Pain
2. Temperature
3. Touch
4. Proprioception
5. Skeletal muscle tone.

Pharmacokinetics

Absorption:

The systemic concentration of ropivacaine depends on the total dose and concentration of drug given, the route of administration, the patient's haemodynamic state and the vascularity of the site of administration. When administered in the epidural space, ropivacaine has a biphasic absorption. The half-lives of the two phases (mean$\pm$ SD) are 14$\pm$7 minutes and 4.2 $\pm$0.9 hours respectively.

Distribution:

After intravascular infusion, ropivacaine has a steady state of distribution of 41 ± 7 litres. It is 94% protein bound, mainly to α_1-acid glycoprotein. In case of

continuous epidural infusion of ropivacaine the plasma concentration can rise due to increased protein binding and reduced clearance. Ropivacaine can easily cross the placenta.

Metabolism and excretion:

Ropivacaine is extensively metabolized by the liver, predominantly by the cytochrome P_{4501A} mediated aromatic hydroxylation to produce 3 – hydroxyl ropivacaine. After a single IV dose, approximately 37% of the total dose is excreted in the urine as both free and conjugated 3-hydroxy ropivacaine. An additional unquantified amount of 2 – hydroxyl – methyl ropivacaine has also been identified s a metabolite.

Ropivacaine metabolites are mainly excreted via kidney. After i.v. administration 86% of the dose is excreted in urine of which only 1% is in unchanged form. Following IV administration, ropivacaine has a mean ±SD total plasma clearance of 387±107mL/min, an unbound plasma clearance of 7.2±1.6L/min and a renal clearance of 1mL/min. The mean ± SD terminal half life is 1.8 ± 0.7h and4.2 ±1.0h after i.v. and epidural administration respectively.

Pharmacodynamics

Central Nervous System & Cardio Vascular System:

Ropivacaine has a higher threshold for both cardiac as well as neurotoxicity as compared to bupivacaine due to its lower lipid solubility and stereo - selective properties. This holds good for both isomers of ropivacaine which have been shown to be less cardio depressant than respective bupivacaine isomers in animal studies.

CNS toxicity occurs earlier than cardiac toxicity on iv infusion in healthy volunteers.

Potency:

Lipid solubility of a local anaesthetic correlates well with its potency and toxicity. Compounds which are more lipophilic penetrate the nerve cell membrane more readily. Thus, fewer molecules are required to produce the desired conduction blockade.

Others:

Continuous epidural infusion of 0.375 % and 0.188% ropivacaine has been shown to inhibit platelet aggregation in plasma.

Adverse Effects

Excessive plasma levels are due to over dosage, unintentional intravascular injection or slow metabolic degradation. The mean doses at which CNS symptoms of toxicity begin to occur in human beings are 4.3 and 0.6 mcg/mL of total and free plasma concentrations respectively. When prolonged blocks are used the risks of reaching atoxic plasma concentration or inducing local neural injury are increased.

Various possible side effects include

a) Injection site pain
b) **Cardiovascular system toxicity:** Vasovagal reaction, syncope, postural hypotension, non-specific ECG abnormalities which include wide QRS complexes, increased conduction time and reduced contractility.
c) **Gastrointestinal system toxicity:** Faecal incontinence, tenesmus, nausea, vomiting.

d) **Central nervous system toxicity:** Tremor, Horner's syndrome, dyskinesia, neuropathy, vertigo, convulsion and coma. Because of depressant effect of ropivacaine on medulla, excitatory stage of CNS might not occur.

Liver and Biliary system toxicity: Jaundice

Metabolic disorders : Hypomagnesemia

Advantages Over Other Local Anaesthetics

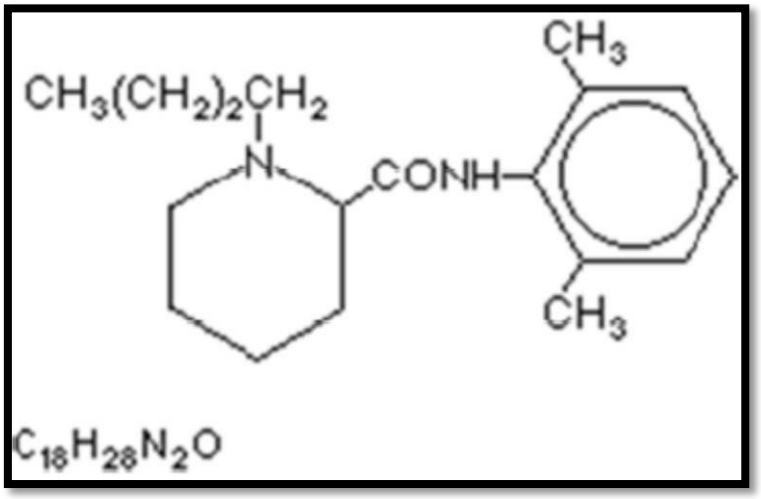
Ropivacaine produces a more differential blockade allowing better separation between sensory and motor block and is therefore a better choice for use in labour analgesia and post operative pain relief. When compared to bupivacaine it produces less dense motor blockade of shorter duration and hence permits earlier mobilization and discharge thus reducing both morbidity as well as cost of treatment. It has a lower systemic toxicity than bupivacaine and a better, cardio stable profile. Ropivacaine has been developed to offer a safer alternative to bupivacaine while retaining the desirable blocking properties of racemic bupivacaine.

BUPIVACAINE[21-22]

Bupivacaine is an amino amide class of local anaesthetic drug. It was first synthesized by Ekenstam in 1957 and its clinical use was started by LJTelivuoin1963. Since then, it has become one of the widely used local anaesthetic agents clinically.

Bupivacaine consists of a tertiary amine attached to a substituted aromatic ring by an amide linkage. The butyl group attached to the piperidine nitrogen makes bupivacaine more lipid soluble and potent. The molecular weight is 288. It is a chiral drug that exists as two enantiomeric forms – dextrorotary (R-) and levorotary (S-) forms. The pure levorotary form Levobupivacaine produce less cardiotoxicity compared to that of the racemic mixture.

Figure 14-Structure of Bupivacaine



PHARMACODYNAMICS

Bupivacaine permeates the nerve's axon membranes and accumulate with in the axoplasm. Binding to sites on voltage-gated Na+ channels prevent opening of the channels by inhibiting the conformational changes that underlie channel activation.

On comparison with lignocaine, it is four times more potent but the onset of action is slower. The duration of action is considerably longer. The sensory blockade caused by bupivacaine is much more than the motor blockade.

PHARMACOKINETICS

It is a weak base with a pKa of 8.1.Bupivacaine is highly protein bound(95%) and most important plasma protein binding site is alpha1 acid glycoprotein. At physiological pH of 7.4, 17% is non-ionised.

The onset and duration of action depend on the dose, concentration, route of administration and vascularity of the site of administration. The volume of distribution is 54 L. The elimination half-life is 210 minutes. The Clearance is 0.32 L/min. Bupivacaine undergoes biotransformation in liver by aromatic hydroxylation,

N- dealkylation, amide hydrolysis, and conjugation. The metabolites are excreted via the kidney. Less than 5% of the drug is excreted unchanged.

Dosage and preparations

Maximum dose of bupivacaine 2-3 mg/kg. Preparations available include 0.25%, 0.5% solutions in 10ml and 20ml vials, preservative free0.5% bupivacaine and 0.75% bupivacaine for intra thecal injections.

Uses

- Peripheral nerve block(0.25-0.5%)
- Epidural Anaesthesia(0.25-0.5%)
- Spinal Anaesthesia(0.5%,0.75%)
- Caudal Anaesthesia(0.25-0.5%)
- Infiltration Anaesthesia(0.25-0.5%)

Contraindications

- Known hypersensitivity to local anaesthetics
- Intravenous regional anaesthesia (IVRA)

Adverse effects

Local Anaesthesia Systemic Toxicity– Plasma concentration greater than 5mcg/ml due to overdosage, unintentional intravascular injection and slow metabolic degradation causes systemic toxicity.

Central Nervous System Toxicity

Non-specific signs of toxicity are metallic taste, circum oral numbness, diplopia, tinnitus, dizziness. Excitation is characterized by restlessness, anxiety, dizziness, tinnitus, blurred vision or tremors. Then, there is a depression of central nervous system causing drowsiness, unconsciousness and cardiac arrest.

Cardiovascular system effects

Part of the cardiac toxicity that occurs with high plasma concentrations of bupivacaine occurs because of the blockade of cardiac sodium channels. Accidental intravenous injection of bupivacaine causes cardiac dysrhythmias, atrio-ventricular block, ventricular tachycardia and ventricular fibrillation, bradycardia and asystole.

Pregnancy increases the sensitivity of cardio toxic effects of bupivacaine.

MATERIALS AND METHODS

Data Source:

The present study title "Comparative study of adductor canal block in addition to epidural infusion versus epidural infusion alone on post-operative analgesia in patients undergoing knee surgeries- a randomized control trial", was conducted under combined spinal epidural anesthesia at KLE's DR. Prabhakar Kore Hospital and Medical Research Center, Nehru Nagar, Belagavi during period of one year from 2023 to 2024".

Study Design : Randomized Controlled Trail.

Study Period : 1Year

Sample Size:

Lowest sample size formula, on mean and standard deviation basis was

$$n = \frac{(Z_1 - \alpha/2 + Z_1 - \beta)^2 (SD_1^{2+} SD_2^{2})}{(\bar{x}_1 - \bar{x}_2)^2}$$

where Z1-β is associated with test power and Z1-α/2 is associated with significance level. z1-α/2 = 1.96 at the 5% threshold of significance and z1-β = 0.84 at the 80% test power.

The VAS score was the parameter taken into account in the computation.

The first group's mean is 1X (1.10), and the second group's mean is 2X (0.68).

1st group's standard deviation (S1) is 0.70 & 2nd group's standard deviation (S2) is 0.50. 7.

The sample size produced with these values was 34.5.

The sample size was increased to 35 in order to round off.

Each of the two groups contained thirty-five subjects.

Inclusion Criteria:

- Patient's belongs to ASA class I, II & III
- Male & female patients among the ages of 30 & 80 who are scheduled for unilateral knee operations under a combination of spinal and epidural anesthesia
- Individuals who are able to give written consent.

Exclusion Criteria:

- Patient's with ASA class >III.
- Participators with Infected TKA, CVA history, Major neurology deficit, spine disease.
- Patients with peripheral neuropathy, allergies to trial drugs & contraindications to RA will be excluded from the study..

Ethical clearance : Approval for the study from the Institutional Ethical and research Committee & CTRI registration obtained prior to its commencement.

Informed Consent : the type of research and the intervention to be performed were explained to all subjects who met the selection criteria. Written informed consent was obtained from each subject prior to enrolment in the study.

Randomization: By computerized randomization, patients were classified into two groups.

GROUP A : Patient received Epidural infusion of 0.1% bupivacaine only**.**

GROUP B : Patient received Epidural infusion of 0.1% bupivacaine and Adductor Canal Block with 10ml of 0.25% ropivacaine."

METHODOLOGY

Standard investigations and a thorough pre-anesthesia evaluation were carried out prior to the day of suergery. An 18G IV access secured, after patient's nil-by-mouth status confirmation, and 10ml/kg of ringer lactate (RL) was used for preloading. The patient was shifted to operating room & universal equipment was attached to note heart rate, non-invasive BP, ECG, and pulse oximeter. Participators were randomized into one of the trial groups. Preoperatively, participants in Group B, received a USG-guided Adductor Canal Block (ACB) under strict aseptic precautions, a sum of 10 mL of 0.25% ropivacaine.

Then patients in both the groups Under strict aseptic precaution, in sitting position, Epidural space was identified at L2-L3 level using 18G Tuohy needle, catheter proceeded & fixed 5cm within epidural space SAB was performed at L3-L4 level utilizing 27G Whitacre needle, 3 ml of 0.5% bupivacaine (hyperbaric) was administered.

After 2.5 hours of spinal anesthesia, **0.1% bupivacaine 6ml bolus** was injected. and an **epidural infusion** of 0.1% bupivacaine was started at **5 mL/hr**. All patients received inj. PCM 1gm as the standard protocol

Post procedure, Participators were observed in PACU & pain evaluated by VAS score. Patients were managed by bolus of 5ml (0.1%) bupivacaine. if VAS >3 as rescue analgesia. If VAS score remained >3 even after the epidural bolus, inj

Tramadol 2mg/kg was administered. Participants were observed for vital signs throughout the procedure. VAS evaluation & rescue analgesics used were noted.

Statistical Analysis :

Software called SPSS 26.0 (IBM Corporation, USA) was utilized for the statistical analysis. The Kolmogorov-Smirnov test was utilized to detect whether the data was normal. Data can be displayed as a number (%) or as mean ± standard deviation. While demographics, VAS scores, rescue analgesia, and the length of analgesia from CESA till VAS > 3 were analysed using Pearson's chi-square test, the ANOVA test was employed to compare VAS pain scores in order to ascertain statistical signification. Statistical impact was described as p-value <0.05.

The following parameter were observed

1) The duration of analgesia was define as time of spinal anesthesia till patient complain of pain i.e VAS>3
2) The total number of epidural bolus doses
3) The total rescue analgesia required.

RESULTS

The present study entitle "comparative study of adductor canal block in addition to epidural infusion versus continuous epidural infusion on post-operative analgesia in patients undergoing knee surgeries- a randomized control trial" was conducted in 70 participants after patients dispersed into two groups.

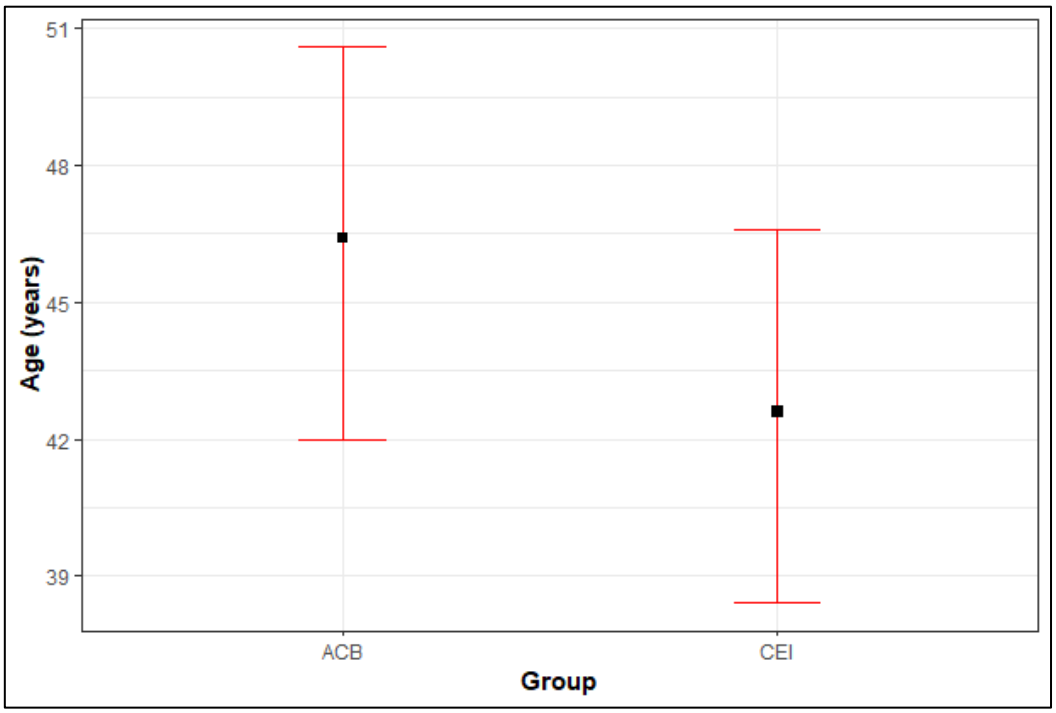
Results were acquired from 70 cases with 35 subjects in B group and 35 subjects in A group. Subsequent table shows differentiated demographic variables among groups.

Table 1: Differentiation in demographic variables over groups.

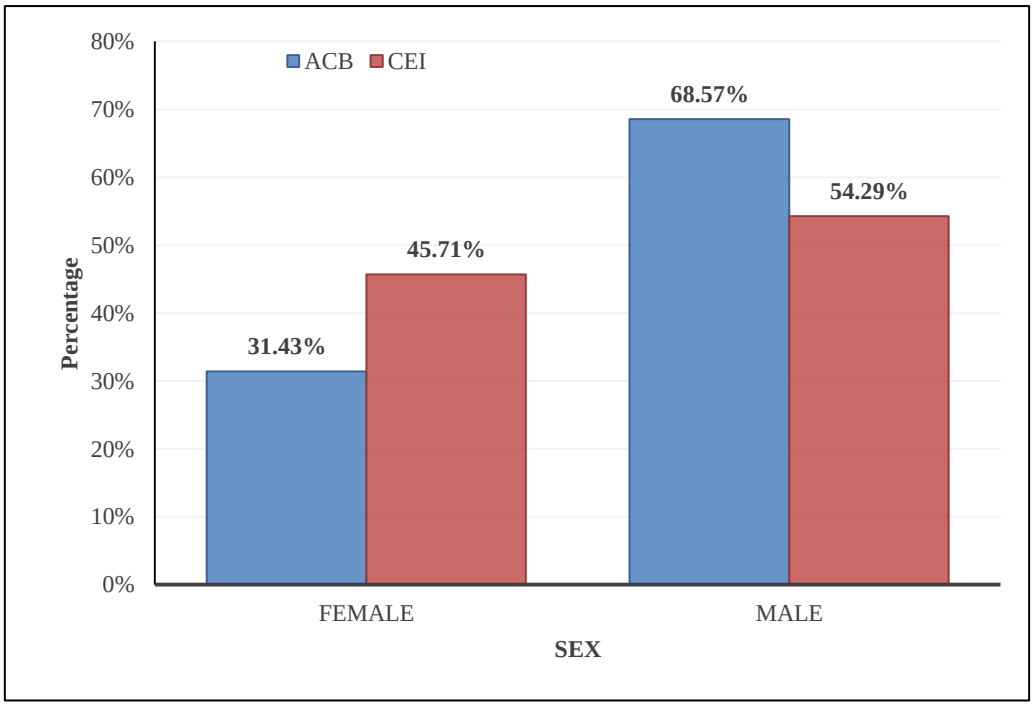
Variables	Sub Category	B	A	P value
Age (years)	M ± SD	46.4 ± 13.18	42.62 ± 12.71	0.2092^{MW}
	Mdn (Min, Max)	47 (18, 65)	45 (21, 61)	
Sex	Female	11 (31.43%)	16 (45.71%)	0.2195^{C}
	Male	24 (68.57%)	19 (54.29%)	

Participants mean age in the B group was 46.4 ± 13.18 years, while in the A group, it was 42.62 ± 12.71 years. The median ages for the B and A groups were 47 years (range: 18–65) and 45 years (range: 21–61), with no appreciable significance amidst groups (p-value = 0.2092). Regarding sex distribution, females comprised 31.43% of the B group and 45.71% of the A group, while males constituted 68.57% and 54.29%, respectively. The overall sex distribution was not significantly different amidst groups (p-value = 0.2195).

Graph 1: Mean plot of age over groups.



Graph 2: Sex Dispersal over groups.



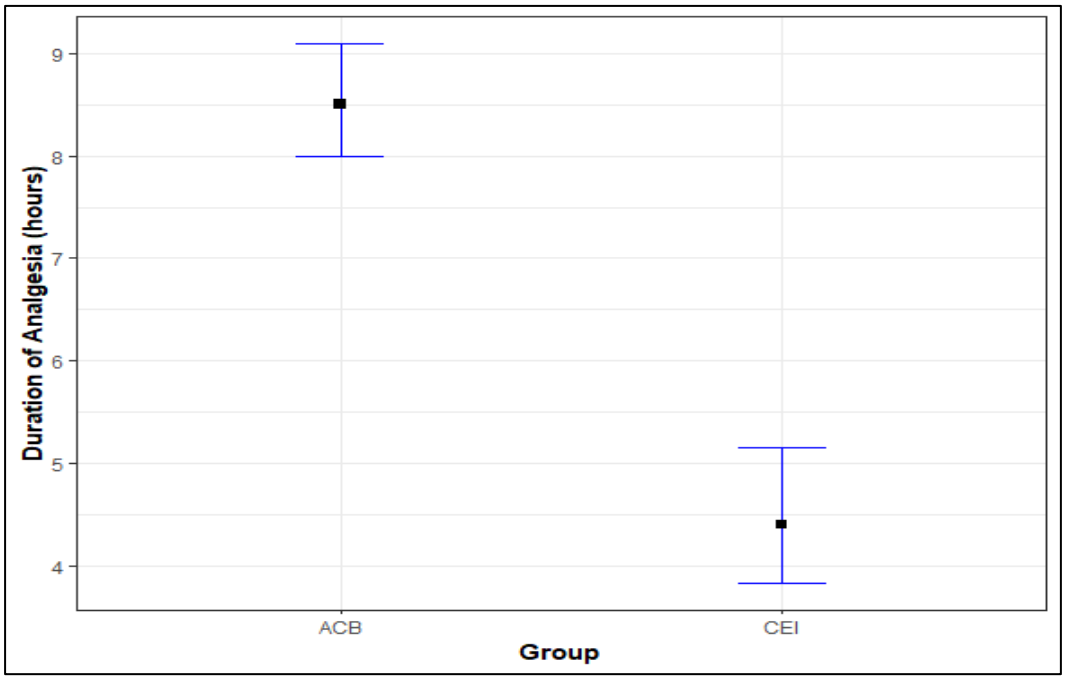
Below table depicts the comparison of duration of analgesia amidst groups.

Table 2: Comparison of duration of analgesia from spinal anaesthesia till VAS >3over groups.

Variables	Sub Category	B	A	P value
Duration of analgesia (hours)	M ± SD Mdn (Min, Max)	8.5 ± 1.71 8.03 (6.05, 14.42)	4.4 ± 2.07 4.05 (2.02, 14)	**< 0.001^{MW*}**

The mean action duration of analgesia in B group was 8.5 ± 1.71 hrs, with a median of 8.03 hours (ranging from 6.05 to 14.42 hrs). In A group, mean duration of analgesia was significantly shorter at 4.4 ± 2.07 hrs, with a median of 4.05 hrs (ranging from 2.02 to 14 hours). Differentiation amidst groups was statistically observable (p-value < 0.001).

Graph 3: Mean plot of duration of analgesia over groups.



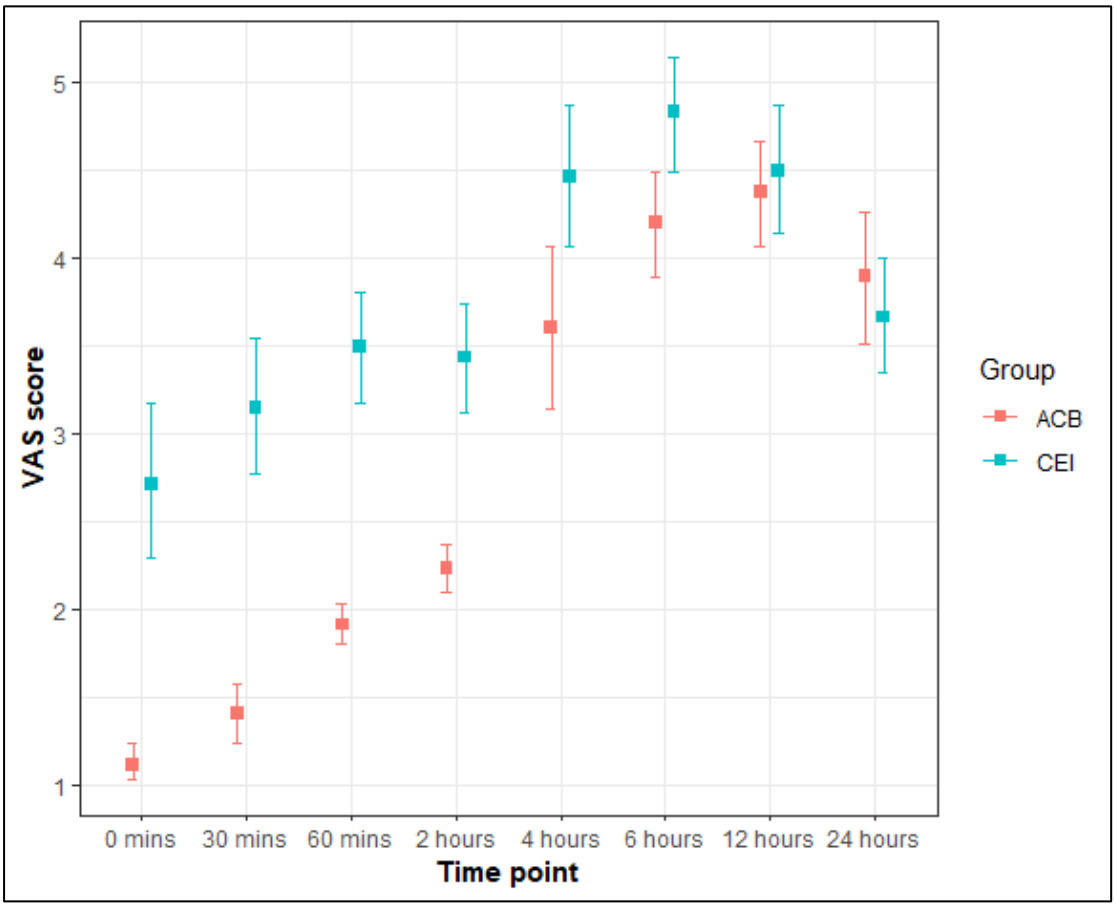
The following table gives the comparison of VAS score over groups.

Table 3: Comparison of VAS score over groups.

Time points	B	A	P value
0 min	1.11 ± 0.32 1 (1, 2)	2.71 ± 1.36 2 (1, 7)	**< 0.001^{MW}***
30 mins	1.4 ± 0.5 1 (1, 2)	3.14 ± 1.17 3 (1, 6)	**< 0.001^{MW}***
60 mins	1.91 ± 0.37 2 (1, 3)	3.49 ± 0.95 4 (2, 5)	**< 0.001^{MW}***
2 hours	2.23 ± 0.43 2 (2, 3)	3.43 ± 0.95 3 (2, 5)	**< 0.001^{MW}***
4 hours	3.6 ± 1.38 4 (2, 6)	4.46 ± 1.24 4 (2, 7)	**0.0142^{MW}***
6 hours	4.2 ± 0.93 4 (2, 6)	4.83 ± 0.98 5 (2, 6)	**0.0032^{MW}***
12 hours	4.37 ± 0.91 5 (1, 6)	4.49 ± 1.07 4 (2, 7)	0.9501^{MW}
24 hours	3.89 ± 1.13 4 (2, 6)	3.66 ± 1 4 (2, 7)	0.3194^{MW}

At 0 min post op, the B group had a notably lesser VAS score contrast to A group (p-value < 0.001). This trend continued at 30 mins, 60 mins, 2 hrs, 4 hrs & 6 hrs, whereas B group consistently reported lower pain scores than the A group, with all notable significance (p-values < 0.05). At 12 hours, VAS score was lessen in B group contrast to A group, but the differentiation was not notable (p-value = 0.9501). By 24 hrs, B group shown increased VAS score than A group, although no remarkable statistics obtained (p-value = 0.3194).

Graph 4: Mean plot of VAS score over time & group.



The subsequent table shows the contrast of total analgesics utilized over groups.

Table 4: Comparison of total analgesics used over groups.

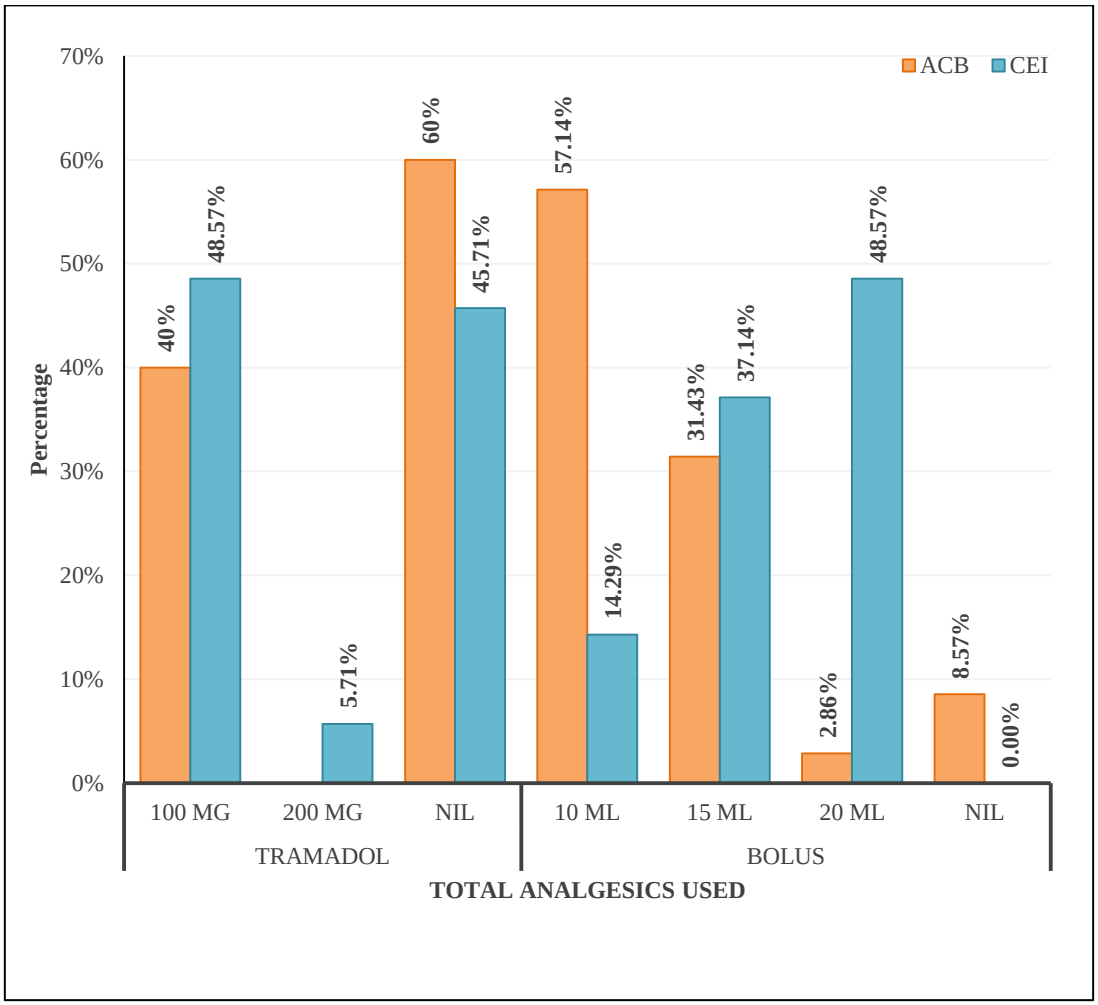
Analgesics	Sub Category	B	A	p-value
Tramadol	100 mg	14 (40%)	17 (48.57%)	0.2594^{MC}
	200 mg	0	2 (571%)	
	Nil	21 (60%)	16 (45.71%)	
Bolus	10 ml	20 (57.14%)	5 (14.29%)	$\mathbf{< 0.001^{MC}*}$
	15ml	11 (31.43%)	13 (37.14%)	
	20 ml	1 (2.86%)	17 (48.57%)	
	Nil	3 (8.57%)	0	

In terms of bolus administration, 10 ml was used by 20 (57.14%) subjects in the B group and 5 (14.29%) subjects in the A group. For the 15 ml bolus, 11 (31.43%) subjects in the B group and 13 (37.14%) subjects in the A group received it. The 20 ml bolus was administered to 1 (2.86%) subject in the B group and 17 (48.57%) subjects in the A group. Lastly, 3 (8.57%) subjects in B group and none in A group did not received a bolus. This shown notable significance (p-value < 0.001).

The total dose of tramadol administered in both groups were estimated:

Tramadol usage, 100 mg was administered to 14 (40%) subjects in the B group and 17 (48.57%) subjects in the A group. The 200 mg dosage was given to 2 (5.71%) subjects in the A group, while none received it in the B group. Additionally, 21 (60%) subjects in the B group and 16 (45.71%) subjects in the A group did not received Tramadol. The differentiation amidst groups was not significant (p-value = 0.2594).

Graph 5: Distribution of total analgesic used amidst group



DISCUSSION

Knee surgeries is frequently associated with substantial postoperative discomfort, with approximately 30% of patients reported tolerable pain & 60% experienced severe pain. Adequate pain management was critical not only for optimizing immediate recovery but also for minimizing chronic post procedural pain, it is strongly correlated with severity of initial postoperative pain. By investigating the most effective analgesic strategies, we aim to enhance both temporary and permanent clinical betterment for knee surgeries patients.

This RCT conducted for one year period among 70 patients between the age 30 to 80 years, posted for knee surgeries. In random manner, participants divided into two groups, 35 in each, using a computer generated randomization. Group A received epidural 0.1% bupivacaine infusion at 5 mL/hr rate & Group B received Adductor canal block with 0.25% ropivacaine 10 ml & epidural infusion 0.1% bupivacaine at 5ml/hour.

The median age in B set was 47 years, contrast to 45 years in the A group, with no significant difference observed. As for sex distribution, 31.43% of patients in the B group were female, while 45.71% of the A group were female. Conversely, 68.57% of the B group were male, and 54.29% of the A group were male. No notable differentiation in gender dispersal observed among both sets ($p = 0.2195$).

Comparison of duration of analgesia from spinal anesthesia till VAS >3 over groups**:**

Analgesia mean duration action in B group class was 8.5 ± 1.71 hrs, with a median of 8.03 hours (ranging from 6.05 to 14.42 hours). In the A group, the mean duration was significantly shorter at 4.4 ± 2.07 hours, with a median of 4.05 hrs

(ranging from 2.02 to 14 hours). The differentiation among groups was statistically notable (P value < 0.001).

VAS at rest:

It was observed, The group B shown remarkable lesser pain scores (VAS) contrast to group A among variable time points (0-6 hours) postoperatively ($p < 0.05$), indicating improved pain management, but this disparity was not statistically remarkable around 12 hrs ($p = 0.9501$) and reversed at 24 hours, with the B group reporting higher VAS scores ($p = 0.3194$).

Remon Nadhy & authors, compared continuous epidural analgesia, USG guidance Continuous Femoral Nerve Block & USG guided Continuous Adductor Canal Block for post procedure pain control preceding TKR. VAS score used to differentiate three groups, in terms of postsurgical pain control & evaluated in PACU at 1hr,6hrs, 12hrs, 24hrs, 36hrs & 48 hrs. Epidural group revealed betterment in pain tolerance & lessen VAS contrast to, combined femoral & adductor blockade groups. This contrasts with the findings from the previous study, where ACB group consistently reported losser pain scores than CEI group in early postoperative hours. However, by 24 hrs post surgically, the trend reversed, ACB group reported enhanced VAS scores than CEI group [17].

"Burhan Asiq & coworkers compared ACB & FNB for postsurgical analgesia preceding arthroscopic knee surgery. The study found no notable significance in Visual Analogue Scale among two groups at various post procedure time points. This result is in contrast to earlier studies, which proved ACB participators experienced consistently lesser pain scores compared to FNB participators in initial postoperative hours[18].

A previous study that compared the effectiveness of a combination of ACB & Popliteal Artery Capsule Block (PACB) versus epidural analgesia indicated that the block group provided equally effective postoperative analgesia as the epidural group at both 8th & 24th hrs. No remarkable differentiation were obtained in VAS scores at 8th hour and 24th hour.[16]

Donghai Li & authors evaluated the efficacy of ACB versus FNB following TKR, it shown ACB provided superior pain relief compared to FNB in early postsurgical period within 24 hours at rest. This finding is consistent with earlier studies, which similarly demonstrated that ACB offered efficient pain control in initial post procedural hours compared to alternative methods like EA.[10]

In a meta-analysis by Aman Arya et al., titled Femoral Nerve Block Improves Analgesia Outcomes after Total Knee Arthroplasty, it was found that single-shot femoral nerve block (SSFNB) was equivalent in effectiveness to both SSFNB with sciatic nerve block (SSFNB Sciatic) and continuous femoral nerve block (CFNB) in terms of pain management at rest, observed at both 24 and 48 hours postoperatively.*

A comparative study by El Tallawy examined surgical wound catheters, FNB & ACB for post procedural pain relief following TKR. The study observed that significant reductions in both static and dynamic pain levels in ACB cases, followed by FNB cases, and lastly the surgical wound catheter (SWC) group, from 3 hrs to 72 hrs post operation ($P < 0.05$).[20]

In contrast to above study, our research, ACB was administered preoperatively, immediately following combined spinal-epidural anaesthesia (CSEA), resulting in complete pain relief at 0 hours postoperatively, which significantly contributed to patient satisfaction upon transfer to the recovery room.

This study revealed a observable differentiation in timing of rescue analgesia among two groups. Cases in A class required rescue analgesia notably earlier, whereas those in B class, who gained preoperative blocks, experienced a delayed need for rescue analgesia, extending up to 8-12 hours postoperatively.

Findings from this study suggest that the addition of ACB to epidural analgesia significantly enhances postoperative pain control. The B group, which received both epidural analgesia and ACB, demonstrated consistently lower Visual Analog Scale (VAS) scores compared to the A group at multiple time intervals postoperatively. The most significant difference was observed within the first six hours postoperatively, indicating that ACB contributes to superior pain relief during the critical early recovery phase.

ACB can be an additional or synergestic technique to epidural analgesia . Hence can effectively be use for post-operative pain relief.

Opioid requirement total rescue analgesia:

Regarding analgesic consumption, fewer patients in the B group required higher doses of rescue analgesics such as tramadol. Additionally, the mean duration of analgesic effect was significantly longer in the B group (8.5 ± 1.71 hours) compared to the A group (4.4 ± 2.07 hours), highlighting the prolonged efficacy of ACB in extending pain relief. Furthermore, bolus administration patterns indicated that patients in the B group required lower volumes of bolus doses, reinforcing the effectiveness of ACB in reducing additional analgesic requirements.

In a study by Burhan Asiq et al., the time to the 1st requirement of analgesia noted between 4-11 hrs, with mean of 6.90 ± 3.5 hrs in ACB cases & 4 -12 hrs with a

mean of 7.25 ± 2.9 hrs in FNB cases. Total tramadol consumption was nearly identical among two groups, with no remarkable significance ($p > 0.05$). As well ACB cases consumed 80 ± 30 mg of tramadol, while the FNB group consumed 85 ± 32 mg**.** This is consistent with the present study, which demonstrates similar tramadol usage in both groups.

In the study by Shung Tai et al., patients receiving IV morphine required more rescue analgesia compared to those receiving epidural or IA morphine, whereas in the present research trial, addition of ACB with CEA provided better post-operative pain management, and also similar effects were found with Tramadol in both groups.[21]

In a study by Khalid A. Alsheikh, a significantly higher proportion of subjects in CEA needed extra intravenous pain killers compared to the ACB. The analgesic used in CEA subjects was morphine, whereas none of the ACB patients required a second-line analgesic.[22] In the present study higher bolus dose of 5 mL of 0.1% bupivacaine was administered in Group A in comparison to Group B and no significant differentiation in Tramadol usage.

In a study by El Tallawy, no significant differences were observed in total intraoperative fentanyl consumption between the groups. However, the total morphine consumption via PCA in 1st 24 hours post- operation showed a significant increase in the SWC group, followed by the FNB group, with the ACB group exhibiting the lowest morphine consumption.[20]

Strengths:

- The current study compares two analgesic techniques (ACB, CEI), providing a comprehensive analysis of the effectiveness of either of the approaches for postoperative pain management in knee surgery. This allows for an apt application of the most effective pain relief strategies.

- The present clinical trial highlights the superior pain control provided by ACB in the early postoperative period (first 12 hours), with significantly lower analgesic requirements. This is crucial for improving patient comfort and facilitating early mobilization, which can positively impact recovery.

- The study measures and compares analgesic use at various postoperative time points (0, 30, 60 minutes, 2 hours), providing a physiological understanding of analgesic consumption patterns over time allowing a more precise evaluation of each intervention's effectiveness.

- The current trial’s novel approach of administering ACB preoperatively, immediately following combined spinal-epidural anaesthesia (CSEA), demonstrated significant benefits in reducing pain right after surgery and enhancing patient satisfaction. This technique could serve as an improvement to standard practices.

- The study provides data on analgesic use up to 24 hours, theorizing as to how pain management needs evolution over the first day after surgery, which can guide protocol for postoperative care planning.

Limitations:

- The present research study has been conducted at a single institution, which limits the generalizability of the findings. Different hospital settings or patient populations may yield varying results.
- Pain tolerance and response to analgesics can vary significantly among patients, and this study may not fully account for this variability. Factors such as age, comorbidities, and psychological factors (e.g., anxiety) could influence pain perception and analgesic requirements.
- The study primarily focused on immediate postsurgical period, with data collected upto 24 hrs post procedure. It does not provide information on the longer-term efficacy of the pain management strategies or the potential for chronic pain development beyond the 24-hour mark.
- Pain assessments rely on self-reported measures (VAS scores), which can be subjective. Variations in how patients perceive or report pain might introduce bias into the study findings.
- The study does not delve deeply into potential side effects or complications associated with each analgesic method (e.g., motor block, hypotension, nausea), which are important to consider when evaluating the overall efficacy and safety of the pain management strategies.
- While the current trial assesses commonly used analgesics like PCM and Tramadol, it does not explore other medications or adjuncts (opioids, NSAIDs, or nerve blocks) that may further influence pain management outcomes.

- Other factors such as surgeon technique, patient adherence to pain management protocols, and rehabilitation practices could influence postoperative pain levels, but these were not controlled for in the study which could introduce confounding variables that might affect the overall conclusions.

CONCLUSION

The conclusion from the present clinical trial is that the addition of an adductor canal block to epidural infusion provided superior postoperative analgesia compared to epidural infusion alone with lesser VAS score and reduced rescue analgesia requirement in patients undergoing knee surgeries.

SUMMARY

The present study titled "Comparative study of adductor canal block in addition to epidural infusion versus epidural infusion alone on post-operative analgesia in patients undergoing knee surgeries- a randomized control trial" was conducted at KLE's Dr. Prabhakar Kore Hospital, Belagavi, from 2023 to 2024, involving patients aged 35-80 years, of ASA physical status I-III, undergoing elective knee surgeries under combined spinal epidural anesthesia. A total of 70 patients were randomly divided into two groups: Group A received epidural infusion of 0.1% bupivacaine, and Group B received combined epidural infusion and ultrasound-guided adductor canal block (ACB) with 0.25% ropivacaine. Preoperatively, all patients underwent a standard anaesthetic evaluation, and intravenous access was secured with 10 mL/kg Ringer's lactate. Epidural space was identified at L2-L3, and a subarachnoid block was given at L3-L4 with 3 mL of 0.5% hyperbaric bupivacaine. After 2.5 hours, a bolus of 6 mL 0.1% bupivacaine was administered, followed by an infusion at 2 mL/hr. Pain was assessed postoperatively using the Visual Analog Scale (VAS), with rescue analgesics administered if VAS scores exceeded.

Demographic data showed no significant differences between the two groups in terms of age ($p = 0.2092$) and sex ($p = 0.2195$). Pain assessment using the Visual Analog Scale (VAS) showed that the B group consistently reported lower pain scores than the A group at 0, 30, 60 minutes, 2 hours, 4 hours, and 6 hours postoperatively, with all differences being statistically significant ($p < 0.001$). However, no significant differences were observed at 12 hours ($p = 0.9501$) or 24 hours ($p = 0.3194$). The total analgesic usage was similar between the groups in terms of Tramadol dosage, but bolus administration differed significantly, with the B group receiving more 10 mL

boluses ($p < 0.001$). The mean duration of analgesia from spinal anesthesia to VAS >3 was significantly longer in the B group (8.5 ± 1.71 hours) compared to the A group (4.4 ± 2.07 hours) ($p < 0.001$).

In conclusion, this clinical trial showed that combining an adductor canal block with epidural infusion provided better postoperative pain relief than epidural infusion alone in knee surgery patients. The B group, which received both treatments, had lower pain scores and longer-lasting analgesia, especially in the first 6 hours. While Tramadol usage was similar in both groups, the A group needed more frequent bolus doses, indicating enhanced pain control. These results highlight the effectiveness of adding adductor canal block for improved and prolonged analgesia.

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ANNEXURE – I - INFORMED CONSENT FORM

"COMPARATIVE STUDY OF ADDUCTOR CANAL BLOCK IN ADDITION TO EPIDURAL INFUSION VERSUS CONTINUOUS EPIDURAL INFUSION ON POST-OPERATIVE ANALGESIA IN PATIENTS UNDERGOING KNEE SURGERIES- A RANDOMIZED CONTROL TRIAL"

Name of Student/Principal Investigator:

Name of Guide/Co Investigators:

Introduction: You are being invited for participation in the study "**COMPARATIVE STUDY OF ADDUCTOR CANAL BLOCK IN ADDITION TO EPIDURAL INFUSION VERSUS CONTINUOUS EPIDURAL INFUSION ON POST-OPERATIVE ANALGESIA IN PATIENTS UNDERGOING KNEE SURGERIES- A RANDOMIZED CONTROL TRIAL".** Knee surgeries have become very common in the present world. The pain that is dealt by the patients after the surgical procedures is far high. To treat the patient in coping up with the pain is utmost necessary. Hence, the respective study has aimed to know the better modality in relieving the pain of the patient after knee surgeries.

Explanation of procedure: If you agree to enrol in my study, I will ask you Present, past and family history. Then, you will be clinically examined in detail. You will be allocated into one of the two groups randomly using computer generated software. Group ACB-will undergo Adductor canal block. Group EI will undergo Epidural infusion alone.

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue

participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will not get any benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the **principal investigator.**

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: If you have any question or complaints with regard to your right as study participant you may contact Dr. Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waiving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**COMPARATIVE STUDY OF ADDUCTOR CANAL BLOCK IN ADDITION TO EPIDURAL INFUSION VERSUS CONTINUOUS EPIDURAL INFUSION ON POST-OPERATIVE ANALGESIA IN PATIENTS UNDERGOING KNEE SURGERIES- A RANDOMIZED CONTROL TRIAL**" My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE II - PROFORMA

"COMPARATIVE STUDY OF ADDUCTOR CANAL BLOCK IN ADDITION TO EPIDURAL INFUSION VERSUS CONTINUOUS EPIDURAL INFUSION ON POST-OPERATIVE ANALGESIA IN PATIENTS UNDERGOING KNEE SURGERIES- A RANDOMIZED CONTROL TRIAL".

Name:

Age:

Gender:

Weight:

Height :

Date of Examination:

Address:

Occupation :

Pre examination evaluation:

Past History:

- HTN DM IHD Arrhythmia Valvular heart diseases .
- H/o previous surgery/(s):

General physical examination:

- Weight (Kg) :
- Temperature (^{0}F) :
- Pallor :
- Cyanosis :
- Pedal edema :
- Clubbing :
- PR :
- BP :
- RR :

Systemic examination:

- RS:
- CNS:
- CVS :
- GIT:

Airway Assessment-

- Teeth-
- Jaw Movement-
- MPG-
- Spine

Investigation

- Hb:
- Platelet:

Preoperative physical status ASA Grade I II III IV V

Diagnosis:

- CESA AT-
- ADDUCTOR CNAL BLOCK AT-

In Post Operative period

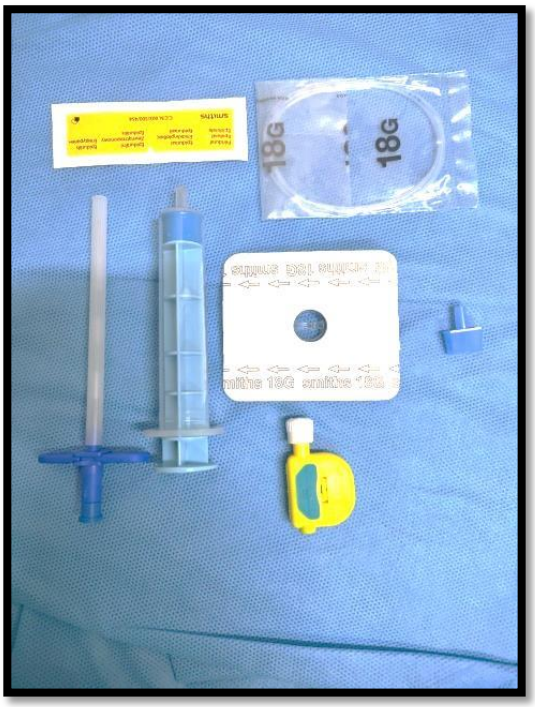
Frist rescue analgesia time-

	Visual Analog Score	Amount of Rescue Analgesics used-bolus	Tramadol use	Duration of analgesia	epidural infusion
0 Minute					
30 Minutes					
60 Minutes					
2 Hours					
4 Hours					
6 Hours					
12 Hours					
24 Hours					

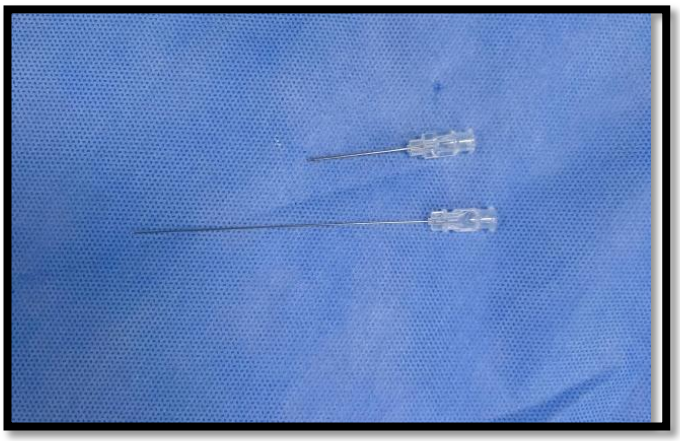
Signature of the anaesthesiologist :

Signature of the principal investigator

ANNEXURE –III- PHOTO GRAPHS

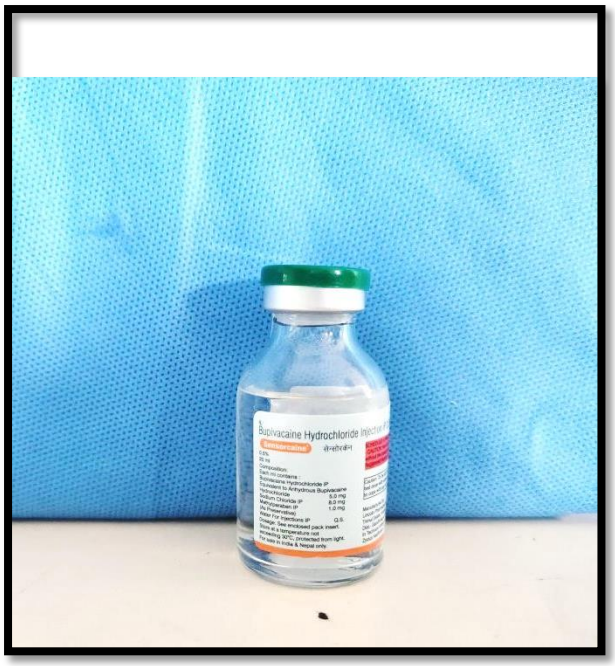


Photograph 1: Epidural kit

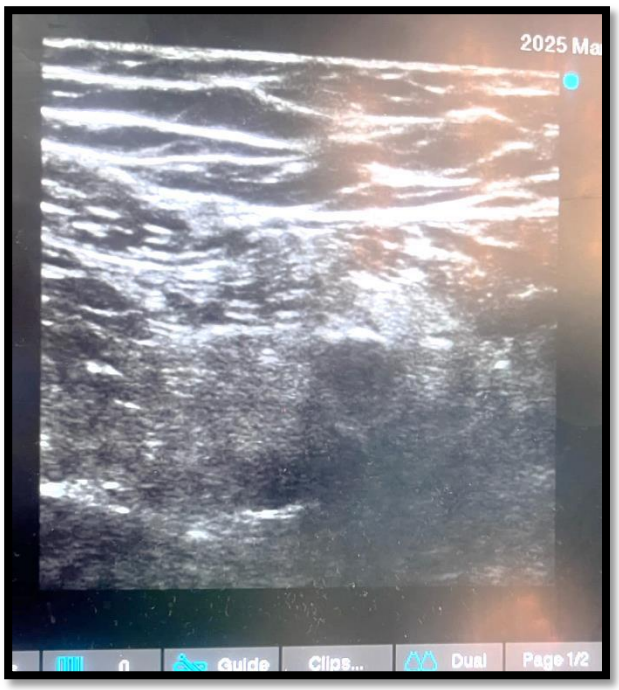


Photograph 2: 27 G whitacre needle

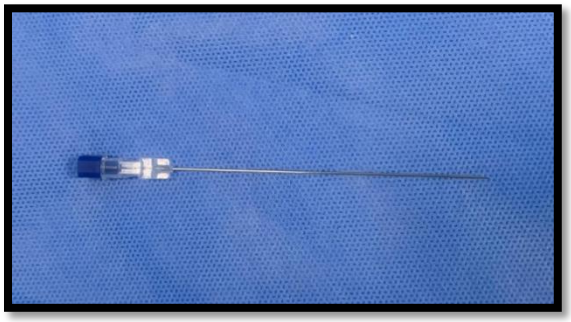
Photograph 3: Injection Ropivacaine



Photograph 4: Injection Bupivacaine



Photograph 5: USG showing Femoral Artery



Photograph 6: 23G Spinal Needle

					STUDY GRP-ADDUCTOR CANAL BLOCK																														
sr no.	Age	Sex	IP No.	group S/C	INDUCTION TIME			TIMEING OF 1ST RESCUE ANALGESIA	Duration of analgesia					Visual Analog Score-post-op						Analgesics Used						each group				Rate Of Epidural Infusion						
					CESA	procedure in hr	ACB	IN HOURS / BOLUS		30mins	60mins	2hour	4hours	6hours	12hours	24hours	0mins	30mins	60mins	2hours	4hours	6hours	12hours	24hours	TRAMADOL		BOLUS	BOLUS	0mins	30mins	60mins	2hours	4hours	6hours	12hours	24hours
1	63	M	10031943	ACB	9AM-12pm	3hr	9.20AM	5PM	8	1	1	2	3	3	4	1	6	nil	nil	nil	nil	nil	BOLUS 5ML	BOLUS 5ML	NIL	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
2	35	M	10053954	ACB	1PM-3pm	2hr	1.20PM	12AM	11	1	1	1	3	4	4	5	5		NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
3	18	M	10001323	ACB	12-4.30PM	4hr	12.20PM	11.45AM	11.45	1	1	1	2	2	5	5	5	NIL	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
4	34	M	10034462	ACB	1.40-4.30PM	3hr	2PM	10PM	8.2	1	1	2	2	2	3	3	5	NIL	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS 5ML	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
5	35	M	10037880	ACB	6.05-9PM	3hr	6.25PM	1AM	6.55	1	1	2	2	3	4	5	4	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS 5ML	NIL	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
6	62	F	10092690	ACB	9AM-12.45pm	3hr30min	9.20AM	7PM	9.55	1	2	2	3	4	5	5	4	NIL	NIL	NIL	NIL		BOLUS 5ML	BOLUS 5ML +TRAMADOL 100ML		TRAMADOL 100MG	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
7	30	M	10057582	ACB	10.45AM-1pm	2hr25min2222	11AM	6PM	7.15	1	1	2	2	2	4	5	3	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	NIL	NIL	5	NIL	NIL	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
8	35	F	10055982	ACB	1.PM-3.30PM	2hr 3omin	1.20PM	11PM	10	1	1	2	2	2	5	3	4	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
9	50	M	10071312	ACB	9AM-12.20pm	3hr 20min	9.20AM	5PM	8	2	2	2	3	4	5	4	3	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML+TRAMADOL	1OOMG	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
10	60	M	10208486	ACB	11.30AM-1pm	1hr 30min	11.45AM	9.30PM	10	1	2	2	2	5	6	4	4	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	NIL	5	5	NIL	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
11	65	M	10032633	ACB	9.30AM-1pm	3hr 30min	9.40AM	5.30PM	8	2	2	2	2	5	5	5	4	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
12	52	F	10112018	ACB	8.30AM-12pm	3hr 30min	8.45AM	4PM	7.3	1	2	2	2	4	5	6	3	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML +TRAMADOL 100ML	NIL	100MG	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
13	60	F	10077406	ACB	8.30AM-12.30pm	4hr	8.45AM	4.40PM	8.1	1	2	2	2	4	6	4	5	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS 5ML +TRAMADOL 100ML	NIL	BOLUS 5ML	100MG	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
14	59	M	10021537	ACB	2.30-5PM	2hr 30min	2.45PM	9PM	6.3	1	2	2	2	5	4	5	3	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
15	59	m	10045678	ACB	12.10PM TO 4.30PM	4hr 30min	12.20PM	9.45PM	9.35	1	2	2	2	4	3	4	5	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS 5ML	NIL	BOLUS 5ML + TRAMADOL	100MG	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
16	44	M	10021759	ACB	3.45-7.30	3hr 20min	4.30PM	12AM	8.15	1	2	2	2	6	4	5	2	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
17	55	F	10046300	ACB	8.30AM-12.30pm	4hr	8.45AM	5.30PM	9	1	1	2	2	3	5	4	6	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML+TRAMADOL	100MG	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
18	32	M	10045672	ACB	11-1.55PM	2hr 30min	11.20AM	6PM	7	1	1	2	3	4	4	5	4	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
19	65	M	10046236	ACB	9.30-12.45PM	3hr 15min	9.45AM	5PM	7.3	1	1	3	3	5	4	4	5	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
20	45	M	10046464	ACB	3.05-6PM	3hr	3.20PM	11PM	8	1	1	1	2	2	2	4	3	NIL	NIL	nil	NIL	nil	NIL	BOLUS 5ML	NIL	NIL	5	NIL	NIL	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
21	32	M	10046480	ACB	3.05-6PM	3hr	3.20PM	1.00AM	9	1	1	2	2	2	2	4	4	NIL	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS 5ML	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
22	50	M	10045707	ACB	8.45-1.30PM	3hr	9AM	6PM	9.15	1	1	2	2	4	3	3	4	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	NIL	BOLUS 5ML	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
23	40	F	10043029	ACB	2.45-6.30PM	3hr 30miin	3PM	10.30PM	7.445	1	1	2	2	4	3	4	3	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS5ML	BOLUS 5ML	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
24	36	F	10046307	ACB	2.30-5PM	2hr 30min	2.45PM	9PM	6.3	1	2	2	2	5	4	5	3	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
25	57	M	10046490	ACB	3.45-6.30PM	2hr 30min	4.30PM	12AM	8.15	1	1	2	2	6	4	5	2	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML +TRAMADOL 100ML	NIL	100MG	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
26	54	M	10046414	ACB	11-1.55PM	2hr 55min	11.20AM	6PM	7	1	1	2	3	6	4	5	4	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
27	45	F	10054907	ACB	6.05-9PM	3hr	6.25PM	1AM	6.55	1	1	2	2	2	4	5	4	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML + TRAMADOL	100MG	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
28	64	F	10045596	ACB	9AM-11.45pm	2hr 30min	9.20AM	7PM	10	1	2	2	3	4	5	5	4	NIL	NIL	NIL	NIL		BOLUS 5ML	BOLUS5M+TRAMADOL	NIL	100MG	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
29	56	M	10044098	ACB	3.0-5.30	2hr 30min	3.25PM	10.45PM	7.45	2	2	2	2	2	4	5	2	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS5ML	NIL	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
30	44	F	10043117	ACB	8.30AM-12.30pm	3hr	8.45AM	5.30PM	9.3	1	1	2	2	2	5	4	6	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS5ML +TRAMADOL	NIL	BOLUS	100MG	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
31	55	M	10046574	ACB	8.45-1.30PM	4hr 30min	9AM	5.45PM	9	1	1	1	2	2	5	4	5	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS5ML + TRAMADOL	NIL	100MH	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
32	27	M	10046577	ACB	10-1.30PM	3hr 30min	10.20AM	7PM	9	1	1	2	2	5	4	5	3	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	BOLUS	100MG	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
33	47	M	10045986	ACB	9.00-11.30AM	2hr 3omin	9.15AM	6PM	9	1	1	2	2	2	5	4	4	NIL	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
34	23	M	10040108	ACB	9.30-1.45AM	4hr 345min	9.45AM	3.45PM	6.15	1	2	2	2	5	4	4	3	NIL	NIL	NIL	BOLUS 5ML	BOLUS 5ML+ TRAMADOL	NIL	BOLUS5ML	BOLUS5ML	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
35	36	F	10045136	ACB	3.0-5.30 PM	**2hr 30min**	3.30PM	10.45PM	7.45	2	2	2	2	2	4	5	2	NIL	NIL	NIL	NIL	BOLUS 5ML	BOLUS	NIL	BOLUS+TRAMADOL	100MG	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
1	54	F	10046156	CEI	CESA- 9-	2hr 45min		11.45PM	14	5	5	4	3	6	5	7	3	BOLUS 5ML	NIL	BOLUS5ML	NIL	BOLUS 5ML	NIL	NIL	BOLUS+TRAMADOL	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML

2	45	F	10046254	CEI	CESA- 12PM-2PM	2hr 45min		2.05PM	2.05	4	3	2	5	4	3	5	4	BOLUS 5ML	NIL	NIL	NIL	BOLUS	NIL	BOLUS 5ML+ TRAMADOL	NIL	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
3	54	F	10112018	CEI	3.30min	2hr30min		4.30PM	3.3	4	4	3	3	4	5	4	4	BOLUS 5ML	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS5ML	NIL	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
4	28	M	10112592	CEI	CESA-9.30AM-1PM	3hr 3omin		2.45PM	5.45	2	3	4	4	3	5	4	3	NIL	NIL	BOLUS 5ML	NIL	NIL	BOLUS 5ML	NIL	BOLUS5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
5	49	F	10111607	CEI	CESA-9.30AM-1.15PM	3hr 45min		2.40PM	5.4	3	4	4	4	5	3	3	3	NIL	BOLUS 5ML	BOLUS 5ML	NIL	BOLUS5ML	NIL	BOLUS5ML+ TRAMADOL		100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
6	32	M	10112161	CEI	CESA- 9.15AM-12.15PM	3hr		2.30PM	5.15	2	2	2	3	4	2	5	3	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
7	28	M	10021574	CEI	CESA-12.20-4.00PM	4hr		6PM	5.3	1	2	2	5	4	6	3	4		NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
8	60	M	10025899	CEI	CESA-8.30AM-12pm	3hr 30min		12.30PM	4.3	1	3	3	4	5	3	6	4	NIL	BOLUS 5ML	NIL	NIL	BOLUS	NIL	BOLUS+TRAMADOL	BOLUS	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
9	23	M	10038214	CEI	CESA- 3.30-6.30	3hr		7.00PM	3.3	2	3	3	4	5	6	6	4	NIL	BOLUS 5ML	NIL	NIL	BOLUS	NIL	BOLUS	BOLUS	NIL	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
10	34	M	10020560	CEI	CESA- 9.30AM-12.15PM	3hr		3PM	5.3	2	2	5	5	4	5	4	3	NIL	NIL	BOLUS 5ML	BOLUS5ML	NIL	BOLUS5ML +TRAMADOL	NIL	BOLUS	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
11	50	F	10007294	CEI	CESA- 11.50AM 2.50PM	2hr		3.15PM	3.25	2	3	2	3	3	6	4	4	NIL	BOLUS 5ML	NIL	NIL	BOLUS5ML	NIL	NIL	BOLUS 5ML	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
12	61	F	10001163	CEI	CESA- 2.50PM - 6PM	3hr		6.15PM	3.25	5	4	5	3	3	6	3	4	BOLUS 5ML	NIL	NIL	BOLUS	NIL	BOLUS + TRAMADOL	NIL	BOLUS	100MG	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
13	21	F	10111973	CEI	CESA- 8.40AM-12.15PM	3hr 30min		1PM	4.2	2	2	4	2	3	5	4	3	NIL	NIL	BOLUS 5ML	NIL	NIL	NIL	BOLUS 5ML	NIL	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
14	26	M	10072003	CEI	CESA- 9.30AM-12.15PM	3hr		4PM	6.3	2	2	3	3	5	4	6	4	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML+ TRAMADOL	NIL	100MG	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
15	49	F	10871461	CEI	CESA- 8.40AM-1.10PM	4hr 30min		1.30PM	4.5	2	5	4	4	4	5	4	5	NIL	BOLUS 5ML	BOLUS 5ML	NIL	NIL	BOLUS+TRAMADOL	NIL	BOLUS	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
16	22	M	10094947	CEI	CESA-9.30AM-1PM	3hr 30min		1pm	3.3	3	2	4	3	6	5	4	3	BOLUS 5ML	NIL	NIL	BOLUS5ML	NIL	BOLUS 5ML+TRAMADOL	NIL	BOLUS 5ML+TRAMADOL	200MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
17	44	M	10109432	CEI	CESA- 6pm-8.45PM	2hr 45min		9.15pm	3.15	2	3	2	3	6	5	5	2	NIL	BOLUS 5ML	NIL	NIL	BOLUS 5ML	BOLUS 5ML+TRAMADOL	NIL	BOLUS5ML	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
18	60	F		CEI	CESA-11.30 TO 3PM	3hr 30min		3.30PM	5	2	3	4	4	6	5	4	4	NIL	BOLUS 5ML	NIL	NIL	BOLUS5ML	BOLUS	NIL	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
19	36	M		CEI	CESA- 9.30-12.30PM	3hr		12.30PM	3	3	4	4	5	6	4	5	3	BOLUS 5ML	NIL	NIL	BOLUS 5ML	NIL	BOLUS5ML +TRAMADOL	NIL	BOLUS 5ML	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
20	46	M		CEI	CESA-12.30-2.40PM	2hr 10min		2.40PM	2.1	3	3	3	3	4	6	4	2	BOLUS 5ML	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML +TRAMADOL 100ML	NIL	1OOMG	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
21	56	F		CEI	CESA- 12.35-3.55PM	3hr 20min		4.PM	3.25	4	3	3	2	2	6	6	7	BOLUS 5ML	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
22	49	M		CEI	CESA- 8.30-12.40	3hr 10min		4PM	8	1	1	2	2	4	5	4	4	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS5ML+ TRAMADOL	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
23	52	M		CEI	CESA- 8.40AM-1.10PM	3hr 30min		1.30PM	4.5	2	4	5	3	3	5	4	5	NIL	BOLUS 5ML	BOLUS 5ML	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	NIL	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
24	45	F		CEI	CESA-8.30-12PM	3hr 30min		12.05PM	4.5	5	4	4	3	6	4	5	3	BOLUS 5ML	BOLUS 5ML	NIL	NIL	NIL	NIL	BOLUS+TRAMADOL	BOLUS 5ML	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
25	56	F		CEI	CESA-9.30-12.30PM	2hr		2PM	4.3	2	2	3	4	3	5	4	3	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	BOLUS 5ML	NIL	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
26	36	F		CEI	CESA- 3.30-6.30	3hr		7.00PM	3.3	2	3	4	4	5	6	6	4	NIL	BOLUS 5ML	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML	BOLUS 5ML	NIL	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
27	32	F		CEI	CESA- 9.30AM-12.15PM	2hr 45min		3PM	5.3	2	2	5	5	4	5	4	3		NIL	BOLUS 5ML	BOLUS 5ML	NIL	BOLUS5ML +TRAMADOL	NIL	BOLUS	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
28	23	F		CEI	CESA- 11.50PM 2.50PM	2hr		3.15PM	3.4	3	5	3	2	7	4	5	4	NIL	BOLUS 5ML	NIL	NIL	BOLUS 5ML	NIL	BOLUS5ML + TRAMADOL	NIL	100MG	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
29	45	M		CEI	CESA- 2.50PM - 6PM	3hr		6.15PM	5.25	7	6	5	2	3	6	2	5	BOLUS 5ML	BOLUS 5ML	NIL	NIL	NIL	BOLUS 5ML + TRAMADOL	NIL	BOLUS 5ML	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
30	34	M		CEI	CESA- 8.40AM-12.15PM	2hr 30min		1PM	4.2	2	2	4	4	3	5	4	3	NIL	NIL	BOLUS 5ML	NIL	NIL	NIL	BOLUS 5ML	BOLUS 5ML	NIL	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
31	56	M		CEI	CESA- 9.30AM-12.15PM	3hr		4PM	6.3	2	2	3	3	5	4	6	4	NIL	NIL	NIL	NIL	BOLUS 5ML	NIL	BOLUS+TRAMADOL	NIL	100MG	5	5	10	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
32	60	M		CEI	CESA- 8.40AM-1.10PM	4hr 30min		1.30PM	4.5	2	2	3	4	4	5	4	5	NIL	NIL	BOLUS 5ML	BOLUS	BOLUS 5ML +TRAMADOL	NIL	NIL	BOLUS 5ML +TRAMADOL 100ML	200MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
33	56	M		CEI	CESA-9.30AM-1PM	3hr 30min		1pm	3.3	5	5	3	2	5	5	4	3	BOLUS 5ML	BOLUS 5ML	NIL	BOLUS 5ML	NIL	NIL	BOLUS5ML	NIL	NIL	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML

34	45	M		CEI	CESA- 6pm-8.45PM	2hr 45min		9.15pm	3.15	2	4	4	3	6	5	5	2	NIL	BOLUS 5ML	NIL	NIL	BOLUS 5ML	NIL	BOLUS 5ML +TRAMADOL 100ML	NIL	100GM	5	10	15	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML
35	34	M		CEI	CESA-11.30 TO 3PM	3hr 30min		3.30PM	4	2	3	4	4	6	5	4	4	NIL	BOLUS 5ML	NIL	NIL	BOLUS 5ML	BOLUS 5ML	NIL	BOLUS 5ML +TRAMADOL 100ML	100MG	5	15	20	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML	5ML