"EVALUATION OF THE EFFECT OF DEXMEDETOMIDINE AS ADJUVANT TO ROPIVACAINE IN ULTRASOUND GUIDED POPLITEAL SCIATIC NERVE BLOCK IN BELOW KNEE SURGERY- A ONE YEAR HOSPITAL BASED DOUBLE BLIND RANDOMIZED CLINICAL TRIAL"

By

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With reference to the above, we wish to inform you that your proposed research project titled **"EVALUATION OF THE EFFECT OF DEXMEDETOMIDINE AS ADJUVANT TO ROPIVACAINE IN ULTRASOUND GUIDED POPLITEAL SCIATIC NERVE BLOCK IN BELOW KNEE SURGERY- A ONE YEAR HOSPITAL BASED DOUBLE BLIND RANDOMIZED CLINICAL TRIAL."** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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LIST OF ABBREVIATIONS

ASA	-	American society of Anesthesiologists
mL	-	Millilitre
mcg	-	Microgram
ng / dL	-	Nanogram per decilitre
bpm	-	Beats per minute
BP	-	Blood pressure
SBP	-	Systolic Blood pressure
DBP	-	Diastolic Blood pressure
MAP	-	Mean arterial pressure
Inj.	-	Injection
LA	-	Local anaesthetic
cm	-	Centimeter
mm	-	Millimetre
mA	-	Milliampere
msec	-	Millisecond
min	-	Minute
hr	-	Hours
kg	-	Kilogram
MHz	-	Mega hertz
TAP block	-	Transversus abdominis plane block
I.V	-	Intravenous
SD	-	Standard deviation
US	-	Ultrasound
USG	-	Ultrasonography
CNS	-	central nervous system
CSF	-	Cerebrospinal fluid
FDA	-	Food and Drug Administration

ABSTRACT

TITLE: " Evaluation of the effect of Dexmedetomidine as adjuvant to Ropivacaine in ultrasound guided Popliteal Sciatic nerve block in below knee surgery-A One year hospital based double blinded randomized clinical trial"

Background: For improving the characteristics of nerve blocks we use varieties of adjuvants. Dexmedetomidine is a highly selective alpha 2 adrenergic agonist proven to be better than clonidine as an adjuvant to local anaesthetics in nerve blocks.

Objectives: The purpose is to assess the impact of Dexmedetomidine added to Ropivacaine in ultrasound guided Popliteal Sciatic nerve block for below knee surgery. The onset, duration of sensory and motor block, effect on intraoperative hemodynamic parameters, time to 1st dose of analgesic and total analgesic requirement in 1st 24 hours' post-surgery are studied.

Methods: Study involved 60 adult patients of ASA I - III status undergoing below knee surgery under ultrasound guided popliteal sciatic nerve block. Randomized to two groups. Group A- received 15 mL of 0.75% Ropivacaine+0.5mL(50mcg) Dexmedetomidine and group B- received 15mL of 0.75% Ropivacaine +0.5mL Normal saline. Onset and duration of sensory and motor block, hemodynamic parameters and post op analgesics requirements were studied to evaluate the effect of Dexmedetomidine as adjuvant.

Results: The Dexmedetomidine group experienced a significantly faster onset of sensory block, averaging 9.23 ± 1.14 minutes, compared to 10.6 ± 1.89 minutes in the Control group. The duration of the sensory block was significantly longer in the Dexmedetomidine group, averaging 15.18 ± 2.16 hours, compared to 11.65 ± 1.96

hours in the Control group. Similarly, the Dexmedetomidine group showed a quicker onset of motor block at 11.67 ± 1.4 minutes versus 13.07 ± 2.02 minutes in the Control group, and the duration of the motor block was significantly extended, averaging 14.3 ± 2.2 hours compared to 10.52 ± 1.99 hours in the Control group. Dexmedetomidine group consistently showed lower average heart rates, and BP (SBP, DBP, MAP) compared to the Control group but not needing any intervention. The time to the first rescue analgesic was significantly longer in dexmedetomidine group with an average of 15.93 ± 2.33 hours compared to 13.33 ± 2.4 hours in the Control group and most of patients in the Dexmedetomidine group required only 1 gram of inj.paracetamol (70%) whereas majority in the control group required 2 grams of paracetamol injection (80%).

Conclusion: Dexmedetomidine as adjuvant to Ropivacaine lead to an earlier onset and prolonged duration of sensory and motor block in ultrasound guided Popliteal Sciatic nerve block for below knee surgery. When compared to control group, Dexmedetomidine group has a notable impact on reducing heart rate .It prolonged the time to the first postoperative request for rescue analgesia and also reduced the total requirement of postop analgesics.

Key words: Popliteal sciatic nerve block, ultrasound,Ropivacaine,dexmeditomedine

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INTRODUCTION

The popliteal sciatic nerve block is a regional anaesthesia technique used for surgeries below the knee. Anaesthetic drug is deposited via popliteal approach around sciatic nerve under ultrasound guidance.

Additional possible advantages of the popliteal nerve block include the prevention of systemic problems and intense analgesia both during and after surgery. It is also beneficial in medically compromised patients[1,2]

There is evidence from studies that ultrasound-guided blocks lead to ease in administration of block, precision in drug placement and faster onset which in turn provides safer and effective block with fewer consequences in comparison with non-ultrasound-based techniques, which depend on anatomical landmarks to place needle.[3] The volume needed to produce an adequate block may be minimized with ultrasound guidance by direct visualization of local anaesthetic spreads around the nerves. Precise administration of anaesthetics to the sciatic nerve under ultrasound guidance ensures a robust safety profile with less systemic adverse events.[4]

Although good postoperative pain control can be achieved with nerve blocks and longer acting anaesthetic like Ropivacaine, the duration of block alone is insufficient in preventing opioid usage after surgery. While continuous infusion and Perineural catheters are good solutions for extending the duration of analgesic effects, they can pose problems with patient care, catheter displacement, and the possibility of higher infection risk. Therefore, the practice of combining drugs with LA-such as steroids, clonidine, adrenaline, and opioids has been around for a long time and continues to be of great interest.[5]

Recently, the analgesic effects of Dexmedetomidine-a highly selective alpha-2 adrenergic receptor agonist has been investigated. It may eventually replace clonidine as its more selective. Local anaesthetics and Dexmedetomidine appear to work synergically to extend the duration of the local anesthetics effects and speed up patient recovery. Patients undergoing lower limb surgeries benefit from reduced pain during the perioperative period, leading to improved postoperative mobility and satisfaction.[6]

Many trials with Dexmedetomidine as additive in TAP block, Femoral block, Brachial plexus block etc. are there. The action of Ropivacaine along with Dexmedetomidine in Popliteal sciatic nerve block have not been studied especially in the Indian population.

So we have evaluated the effect of Dexmedetomidine as additive to Ropivacaine on onset and duration of sensory and motor block in popliteal sciatic nerve block. Also its effect on hemodynamic parameters and post op analgesia was studied.

AIM AND OBJECTIVES OF THE STUDY

AIM

- "To evaluate the effect of Dexmedetomidine as adjuvant to Ropivacaine in ultrasound guided Popliteal Sciatic nerve block in below knee surgery."

PRIMARY OJECTIVE:

- To evaluate the effect of Dexmedetomidine as adjuvant to Ropivacaine on onset and duration of sensory and motor block in ultrasound guided Popliteal Sciatic nerve block for below knee surgery.

SECONDARY OJECTIVES:

- To determine the effect on intraoperative hemodynamic parameters.
- To determine the time to 1st dose of analgesic in the postoperative period.
- To determine the total analgesic requirement in 1st 24 hours' post-surgery

REVIEW OF LITERATURE

William Halsted was the first to report administering cocaine to accessible peripheral nerves in order to execute dental blocks and achieve "conduction" anaesthesia in peripheral areas in 1884. He started by blocking the mandibular nerve. Along with themselves, Halsted and Hall also carried out a number of other peripheral nerve blocks on "volunteers" who were medical students (Cousins and Bridenbaugh 1988). Finding and blindly injecting a peripheral nerve percutaneously became the next obstacle in the development of regional anaesthesia.[7]

Orebaugh et al. (2007, 2009) observed that when anaesthesia residents conducted nerve block under supervision of USG rather than nerve stimulation, ultrasound guidance led to a reduction in procedure times, needle insertions, and unintentional vessel punctures .[3]

In 2010 J. Griffin et al reviewed various studies on use of ultrasound and its advantages over conventional techniques in regional anaesthesia. They opined that the introduction of ultrasound has made regional anaesthesia more safe, efficient and cost effective. The real time introduction of needle, visualization of anatomical structures and accurate deposition of local anesthetic has made ultrasound the gold standard for regional anaesthesia. The added advantages in post-operative pain and significant reduction in morbidity involved with blind techniques have made ultrasound guided nerve blocks popular.[4]

The use of Dexmedetomidine as an adjuvant to LAs for peripheral nerve blocks was studied in 2012 by D. Marhofer et al. 36 volunteers underwent ultrasound-guided ulnar nerve block with either 3 ml ropivacaine 0.75% alone, 3 mL ropivacaine

0.75% along 20 µg dexmedetomidine, or 3 mL ropivacaine 0.75% with systemic 20 µg dexmedetomidine. Researchers observed a significant prolongation of Ulnar nerve block (about 60%) when perineural dexmedetomidine was added to 0.75% ropivacaine, and approximately 10% prolongation when 20 µg of Dexmedetomidine was administered systematically.[6]

In a 2015 study by Suneet Kathuria et al., sixty patients receiving Brachial plexus block were allocated into groups of twenty. 30 mL ropivacaine (0.5%)was administered to all, 50 mcg of dexmedetomidine was added in to ropivacaine in second group, and Dexmedetomidine infusion (50 mcg) was given in last group. The second group was shown to have a substantially faster onset as well as a longer block duration than the other groups. [8]

In 2015, Medha Mohta et al. conducted a study on the analgesic effectiveness of dexmedetomidine paired with bupivacaine for paravertebral block in patients going through major breast cancer surgery. Patients in group one was given paravertebral block with 0.5% bupivacaine 0.3 mL/kg with 1 mL of normal saline; patients in second group got paravertebral block with 0.5% bupivacaine 0.3 mL/kg and dexmedetomidine 1 mcg/kg in an amount of 1mL.The incorporation of dexmedetomidine to bupivacaine in paravertebral block was found to result in a longer duration of the block's effects, extended postoperative analgesia, decreased postoperative opioid intake, and a reduced risk of nausea and vomiting[9]

The effects of various dosages of dexmedetomidine on the characteristics of lower limb nerve block in patients receiving arthroscopic knee surgery were investigated by Abdulatif et al. in 2016. In this investigation, a USG-guided femoral nerve block was carried out prior to general anaesthesia utilizing a control group of 25

mL of bupivacaine 0.5% mixed with normal saline and three test groups of 25 mcg, 50 mcg, or 75 mcg of dexmedetomidine. Dexmedetomidine dosage levels of 50 mcg and 75 mcg were linked to longer block durations, shorter onset times, and longer times until the patient requested rescue analgesia for the first time after surgery.[10]

In a study done in 2016 by Xiawei Hu et al, USG assisted Popliteal sciatic nerve, obturator and femoral block together was used as surgical anaesthesia for patients undergoing varicose vein stripping. Patients were randomized to receive either Dexmedetomidine (50 mcg) or saline (0.5mL) combined with 0.75% Ropivacaine (10 mL) plus 2% lidocaine(9.5mL). The Dexmedetomidine group experienced a faster onset and duration of sensory and motor blockage. Dexmedetomidine combined with 0.75% ropivacaine plus 2% lidocaine in Popliteal Sciatic nerve block, showed notable improvement in the effectiveness of regional anaesthesia.[11]

In a 2017 study by E. Koraki, C. et al. on the use of dexmedetomidine with 0.5% ropivacaine in axillary brachial plexus block using USG, one group received 0.5% Ropivacaine (15 mL) with Dexmedetomidine(1mL) while other group received saline instead of Dexmedetomidine. Results indicated significant increase in the duration of sensory, motor block and the duration of analgesia The onset time of the sensory block was also significantly faster with Dexmedetomidine as an adjuvant but onset of motor block showed no significant difference.[5]

The impact of adding dexmedetomidine in supraclavicular brachial plexus block for patients going through procedures on arm was examined in a 2017 study by Chinnappa J et al.60 patients made to equal random groups of 30. One got 0.5% Ropivacaine (30 milliliters) and one milliliter saline, and the other got one mcg/kg

dexmedetomidine along with Ropivacaine[12] .It observed analogous results like E.Koraki C et al.

In a 2017 study by Jakob H. Andersen et al., all participants underwent bilateral saphenous nerve blocks: on one side 20 mL Ropivacaine (0.5%) plus one mL dexmedetomidine, and on other side 20 mL Ropivacaine(0.5%) plus one mL saline. In the ones who got ropivacaine plus dexmedetomidine, the mean duration of block assessed by temperature sensation in the leg was 22 hours (95% confidence interval, 21–24) as opposed to 20 hours (95% confidence interval, 19–21) in the saline group. Therefore, the study came to the conclusion that, although not always to a clinically significant degree, dexmedetomidine extends saphenous nerve block action.[13]

Dexmedetomidine as additive to ropivacaine for Erector Spinae plane block was the subject of a study by Wang Yi-han et al. in 2020 on surgery of lumbar spine. The results showed that this combination reduces the need for remedial analgesia, prolongs the duration of sensory block, effectively controls postoperative acute pain, and enhances patient satisfaction and postoperative recovery quality. The Dexmedetomidine group experienced lower VAS pain scores at 12-48 hours ($P < 0.05$) .Following surgery, there was also a decrease in the use of opioids.[14]

In 2020, QiangWang et al investigated the impact of Dexmedetomidine as adjuvant in ropivacaine for Erector spinae plane block in patients who had esophageal cancer. The patients were randomized to receive ESPB using 28 mL of 0.5% ropivacaine, with 2 mL of normal saline or 0.5 µg/kg dexmedetomidine in 2 mL administered interfascially. Under ultrasound guidance, an erector spinae plane block was executed at the fifth thoracic level. They found that it prolonged the duration of

analgesia, Time to first analgesic request, and thus reducing the perioperative analgesic consumption without increasing additional incidence of adverse effects.[15]

Although there are several studies evaluating the effect of Dexmedetomidine as adjuvant to local anaesthetics in peripheral nerve blocks, very few trials have studied its effect when added to local anaesthetic agent for popliteal block. Hence this study was conducted to determine the effect of Dexmedetomidine as an adjuvant to Ropivacaine in popliteal sciatic nerve block.

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BASIC SCIENCES

ANATOMY[16,17]

SCIATIC NERVE

Introduction

The **sciatic nerve** is the largest nerve in the human body and is derived from the sacral plexus. It originates from the anterior rami of the lower lumbar (L4-L5) and upper sacral spinal nerves (S1, S2, S3). It contains fibers from both the posterior and anterior divisions of these spinal nerves

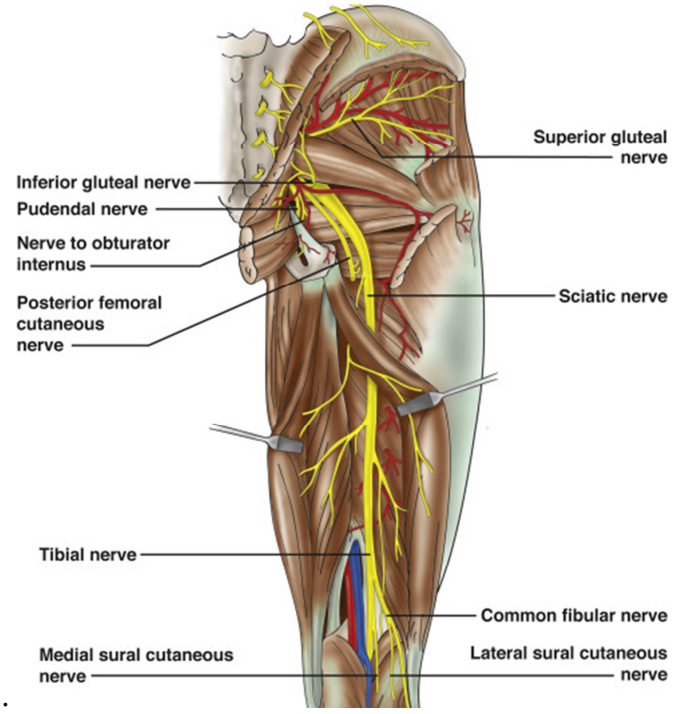


Figure 1: Sciatic nerve Anatomy

The sciatic nerve arises in the lumbosacral region. It descends through the posterior aspect of the thigh. Before entering the popliteal fossa, the nerve terminates by splitting into two large terminal branches: the **tibial nerve** and **common fibular (peroneal) nerve.**

The main function of the sciatic nerve is to provide sensory and motor supply to the skin and muscles of the thigh, leg and foot.

Origin	L4, L5, S1, S2, S3
Branches	Muscular branches of sciatic nerve, tibial nerve, common fibular (peroneal) nerve
Supply	Motor: Muscles of the posterior thigh, ischial portion of adductor magnus, muscles of the posterior, anterior and lateral compartments of the leg, foot muscles. Sensory: Lateral and posterior leg, dorsum and sole of the foot.

Origin and course

The sciatic nerve is a terminal branch of the sacral plexus. It is formed from both anterior and posterior divisions of the anterior (ventral) rami of spinal nerves L4 through S3. The anterior branches of these five spinal nerves meet and converge in the posterior pelvic region to form a single large nerve. The sciatic nerve then descends posteriorly and leaves the pelvis through the **greater sciatic foramen.** It passes inferior to the piriformis muscle, accompanied by the posterior femoral cutaneous nerve, pudendal nerve, internal pudendal artery and vein, inferior gluteal artery and vein.

The sciatic nerve then continues its course through the **posterior thigh**. It runs between the long head of the biceps femoris muscle and the adductor magnus muscle, and laterally to semitendinosus and semimembranosus muscles.

Branches

On its course through the posterior thigh, the sciatic nerve gives off several small **motor muscular branches** that innervate several muscles of the thigh (muscles of the posterior compartment, ischial portion of adductor magnus).

Sciatic nerve in popliteal fossa

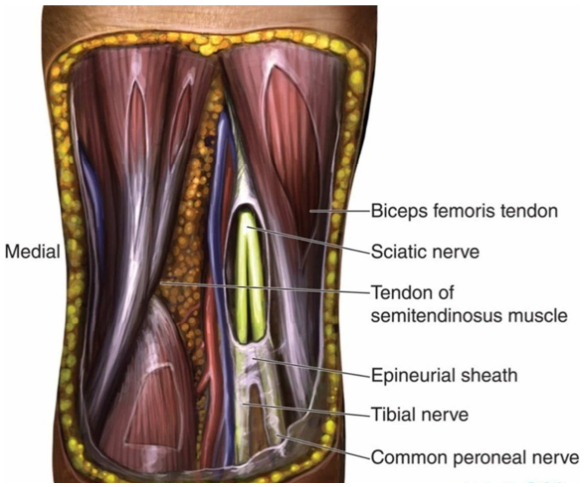


Figure 2: Sciatic nerve in popliteal fossa

At the apex of the popliteal fossa, the sciatic nerve terminates by dividing into two terminal branches:

- The tibial nerve
- The common fibular (peroneal) nerve

The **tibial nerve** continues the course of the sciatic nerve and descends down through the posterior aspect of the leg as far as the heel of the foot. More specifically, the tibial nerve passes through the center of the popliteal fossa and runs below the tendinous arch of the soleus muscle. It continues its course in a neurovascular bundle through the posterior leg compartment and passes through the **tarsal tunnel**. When it reaches the foot, the tibial nerve divides into two terminal branches: medial and lateral plantar nerves that innervate the majority of the foot muscles.

In contrast to the tibial nerve, the **common fibular (peroneal) nerve** courses laterally towards the head of the fibula. When it reaches the anterior compartment of the leg, the nerve divides underneath the fibularis longus muscle into the superficial fibular(peroneal)nerve and deep fibular (peroneal) nerve. The superficial branch supplies the lateral compartment of the leg, while the deep branch supplies the anterior compartment of the leg and medial aspect of the foot.

The **deep fibular (peroneal) nerve** descends between the fibula and the superior part of fibularis (peroneus) longus, runs deep to extensor digitorum longus and anterior to the interosseous membrane.

Innervation

The sciatic nerve is a mixed nerve that provides numerous branches for sensory and motor supply for the skin and muscles of the lower limb. Additionally, it provides articular branches for the innervation of the lower limb joints.

Motor innervation:

- The **sciatic nerve** provides a direct motor supply for the muscles of the posterior thigh via small muscular branches. These muscles include biceps femoris, semimembranosus, semitendinosus, and the ischial portion of the adductor magnus
- The **tibial nerve** provides motor supply for the muscles in the posterior portion of the leg and foot. These muscles include the gastrocnemius, soleus, plantaris, popliteus, flexor hallusis longus, flexor digitorum longus and tibialis posterior.
- The **common peroneal nerve** supplies the muscles of the anterior and lateral sections of the leg and foot. The muscles of the anterior aspect include: tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius. The lateral leg muscles include peroneus longus and peroneus brevis.

Sensory innervation:

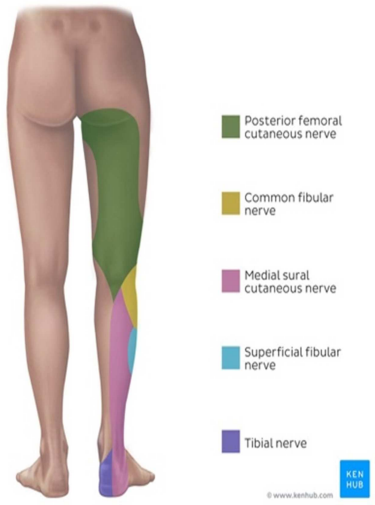


Figure 3: Sensory innervation of Sciatic nerve

The sciatic nerve gives off **sensory branches** that provide sensory supply through its terminal branches;

- The **tibial nerve** innervates the sole of the foot.
- The **branches of the common peroneal nerve** innervate the lateral aspect of the leg and dorsum of the foot, as well as the skin between the first two toes.
- The tibial nerve and common peroneal nerve make up the medial and lateral sural nerves. These nerves provide sensation to the calf and a small lateral portion of the foot.

POPLITEAL SCIATIC NERVE BLOCK[18,19]

Distal sciatic nerve block (popliteal fossa block) is a very clinically valuable technique that results in anaesthesia of the calf, tibia, fibula, ankle, and foot. This section describes the landmarks and nerve stimulator techniques to perform a popliteal sciatic nerve block.

Popliteal fossa block performed with long-acting local anaesthetics such as ropivacaine can provide 12–24 hours of analgesia after foot surgery. Analgesia with popliteal fossa blocks lasts significantly longer than with ankle blocks.

Distribution of Anaesthesia:

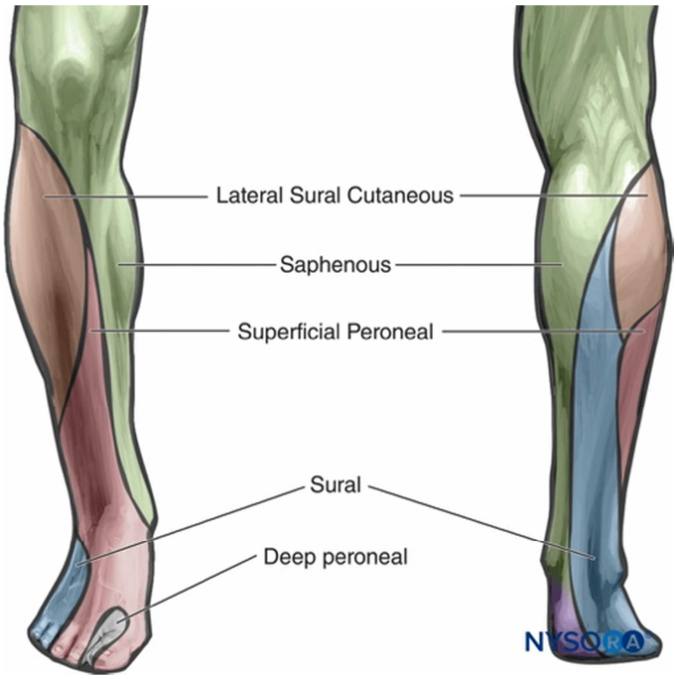


Figure 4: Sensory distribution of anaesthesia after popliteal block. (Popliteal block results in anaesthesia of all shaded areas except that of the saphenous nerve (femoral)).

Popliteal block results in anaesthesia of the entire distal two thirds of the lower extremity, with the exception of the medial aspect of the leg. Cutaneous innervation of the medial leg below the knee is provided by the saphenous nerve, a superficial terminal extension of the femoral nerve. Depending on the location of surgery, the addition of a saphenous nerve block may be required.

ULTRASONOGRAPHY[20]

Ultrasound, is sound waves in a frequency range of around 2 to 15 megahertz has a wide range of diagnostic and treatment purposes in the field of medicine. Ultrasonography works on the principle of the Piezoelectric effect. This effect

converts mechanical/kinetic energy into electrical energy by the deformation of crystals. The piezoelectric effect can also be reversed i.e., by electrical energy the crystals can be oscillated to form ultrasound waves (mechanical energy).

The ultrasound transducer has the function of producing the ultrasound by the above-said mechanism. This ultrasound produced travels through tissues and gets reflected. The returned echo waves after reaching the transducer get changed to electrical energy which is later processed and produce an image. The transducers work in a range of frequencies. Transducers with higher frequencies (5 – 7.5 MHz) are used in imaging superficial structures whereas the ones with lower frequencies (2.5 – 3.5 MHz) produce images of deeper structures.

It is on the surface that lies between tissues of varying density, that the ultrasound gets reflected. If the difference in densities is higher, the sound waves that get reflected are also high and the opposite also holds. Therefore, with a very high difference in densities (bones, air, calculi) the sound will be completely reflected. This produces acoustic shadowing. If the tissues are homogenous in their densities, then echo-free images are seen (blood, urine, ascites).

Transducer:

This is the handheld part of the ultrasound machine. It has the function of inter-converting the energies (electrical and mechanical) based on the piezoelectric effect. They contain lead zirconate titanate crystals commonly. They produce ultrasound waves in either linear(sequential) arrays or phased arrays.

It comprises 5 major components:

- Crystals: possessing piezoelectric property. Can be arranged in either a linear or curvilinear manner.
- Electrodes: positive and ground. For electrical connection
- Damping block: to dampen stray sound waves.
- Matching layer: one or multiple. For proper transmission of sound waves to the tissues.
- Housing.

Linear Transducer:

- The piezoelectric crystals – Linearly arranged.
- Produce rectangular ultrasound beam.
- Used for superficial imaging.
- Footprint – wide with a frequency of 2.5 – 12MHz at the center in the 2D imaging probe and frequency of 7.5 – 12 MHz at the center in the 3D imaging probe.

Figure 5: Linear USG probe

Applications:

- ❖ Peripheral nerve blocks
- ❖ Vascular examination, venous puncture (catheterization)
- ❖ Breast imaging
- ❖ Thyroid imaging
- ❖ Tendons and joints
- ❖ During laparoscopic procedures
- ❖ Measuring body fat thickness
- ❖ Ultrasonic velocity change imaging

Curvilinear Transducer:

- The Piezoelectric crystals – curvilinear arrangement.
- They produce a convex ultrasound beam.
- Used to image deeper tissues.
- As the depth of imaging increases, image resolution decreases.
- A footprint is wide with central frequency being, 2.5 – 7.5MHz for 2D imaging and 3.5 – 6.5MHz for 3D imaging.

Applications:

- ❖ Abdominal examinations,
- ❖ Transvaginal and trans rectal examinations,
- ❖ Diagnosis of organs.

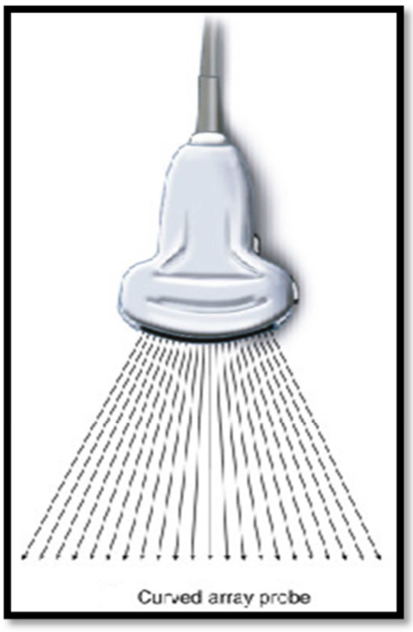


Figure 6: Curvilinear USG probe

ULTRASOUND GUIDED POPLITEAL SCIATIC NERVE BLOCK[19]

- Indications: Foot, ankle and Achilles tendon surgery
- Transducer position: Transverse over the popliteal fossa
- Goal: Local anaesthetic spread surrounding the sciatic nerve within the epineural sheath
- Local anaesthetic: 15–20 mL

USG GUIDANCE

US guidance reduces the volume required for a reliable nerve block because the injection can be halted once adequate spread is observed. The most common approaches to the popliteal sciatic nerve block are the lateral approach, with the patient in the supine or lateral position, and the posterior approach in the prone or

lateral position. While the patient position and needle path differ between the two approaches, the rest of the technique details are similar

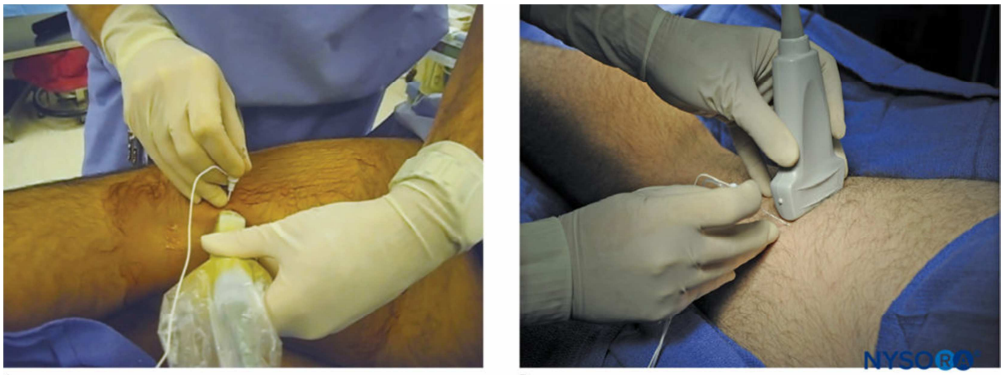
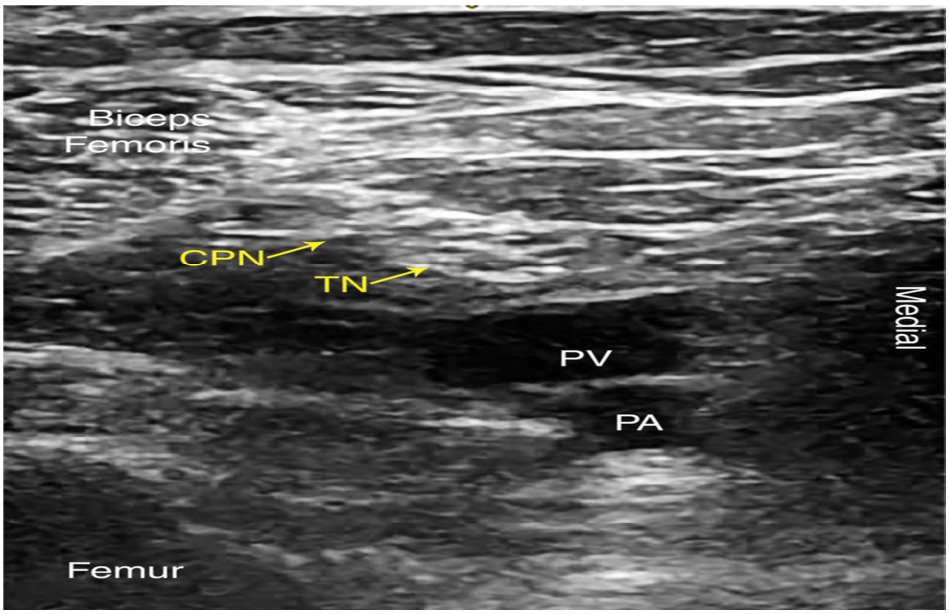


Figure 7: The posterior approach to the US-guided popliteal sciatic nerve block can be performed (A) with the patient in the lateral position, or (B) with the patient prone.[31]

The injection of local anaesthetic must occur within the sciatic nerve sheath that contains both components of the nerve. The injection is ideally accomplished at the position where both components of the nerve are within the sheath but slightly separated by adipose tissue, allowing for safe placement of the needle between them. Although the sciatic nerve block can be accomplished with an injection around either nerve component, injecting into the space between both is more common in clinical practice.

ULTRASOUND ANATOMY

Beginning with the transducer in the transverse position at the popliteal crease, the popliteal artery is identified, aided with colour Doppler US when necessary, at a depth of approximately 3–4 cm. The popliteal vein accompanies the artery at it is positioned just superficial (posterior) to it. On either side of the artery are the biceps femoris muscles (laterally) and the semimembranosus and semitendinosus muscles (medially). The tibial nerve is positioned superficial and lateral to the vein and is seen as a hyperechoic, oval or round structure with a honeycomb pattern (**Figure 8**). Asking the patient to dorsiflex and plantar flex the ankle makes the two sciatic nerve branches twist or move in relation to each other. Usually, tilting the transducer caudally is necessary to bring out the nerve from the neighbouring adipose tissue.



(Figure 8). Sonoanatomy of the sciatic nerve at the popliteal fossa. The two main divisions of the sciatic nerve, the tibial nerve (TN) and the common peroneal nerve (CPN), are seen immediately lateral and superficial to the popliteal vein (PV) and artery (PA). This image was taken at 5 cm above the popliteal fossa crease, where the TN and CPN have just started diverging.

Once the tibial nerve has been identified, the common peroneal nerve is visualized slightly more superficial and lateral to the tibial nerve. The transducer should be slid proximally until the tibial and peroneal nerves are visualized coming together to form the sciatic nerve before its division.

This junction usually occurs at a distance 5–10 cm from the popliteal crease but may occur very close to the crease or, less commonly, more proximally in the thigh.

As the transducer is moved proximally, the popliteal vessels move deeper and become more challenging to image. Adjustments in depth, gain, focus, and direction of the US beam should be made to keep the nerve visible at all times. At the popliteal fossa, the sciatic nerve typically is visualized at a depth of 2–4 cm.

Equipment

The equipment recommended for a popliteal sciatic nerve block includes the following:

- Ultrasound machine with a linear transducer (8–12 MHz), sterile sleeve, and gel
- Standard nerve block tray
- A 20-mL syringe containing local anaesthetic
- 50- to 100-mm, 21- to 23-gauge, short-bevel, insulated stimulating needle
- Sterile gloves

LANDMARKS AND PATIENT POSITIONING:

POSTERIOR APPROACH

This nerve block is performed with the patient in the prone or lateral position A small footrest is useful to facilitate identification of a motor response if nerve stimulation is used. A footrest also relaxes the hamstring tendons, making transducer placement and manipulation easier.

GOAL

The goal is to inject the local anaesthetic within the common connective tissue (Vloka's) sheath that envelops the TN and CPN. Alternatively, separate nerve blocks of TN and CPN can be performed.

TECHNIQUE

For the posterior approach, the needle is inserted in plane from lateral to medial or out of plane. If nerve stimulation is used (0.5 mA, 0.1 msec), the contact of the needle tip with either branch of the nerve usually is associated with a motor response of the calf or foot. Once the needle tip is placed within the common sciatic nerve sheath, 1–2 mL of local anaesthetic is injected to confirm the proper injection site. Such injection should result in a distribution of local anaesthetic within the sheath, and separation of the TN and CPN within Vloka's sheath

Choice of Local Anesthetic:

Popliteal block requires a larger volume of local anesthetic (20 mL) to achieve anesthesia of both divisions of the nerve. US guidance reduces the volume required for a reliable nerve block because the injection can be halted once adequate spread is observed. The choice of type, volume, and concentration of local anesthetic should be based on the patient's size and general condition and whether the block is planned for surgical anesthesia or pain management. The type and concentration of local anesthetics and the choice of additives to local anesthetic influence the onset and, particularly, the duration of the block.

TABLE 1. Local anesthetics choice for popliteal block.

	Onset (min)	Anesthesia (h)	nalgesia (
3% 2-Chloroprocaine	10–15	1	2
1.5% Mepivacaine	15–20	2–3	3–5
1.5% Mepivacaine epinephrine	15–20	2–5	3–8
2% Lidocaine epinephrine	10–20	2–5	3–8
0.5% Ropivacaine	15–30	4–8	5–12
0.75% Ropivacaine	10–15	5–10	6–24
0.5 (L) Bupivacaine	15–30	5–15	6–30

LOCAL ANAESTHETICS:[21]

Local anaesthetic drugs are water-soluble salts of lipid-soluble alkaloids. The structure of local anaesthetics consists of three components: a lipophilic aromatic group, an intermediary link and a hydrophilic amine group. The intermediary link categorises local anaesthetics into esters or amides

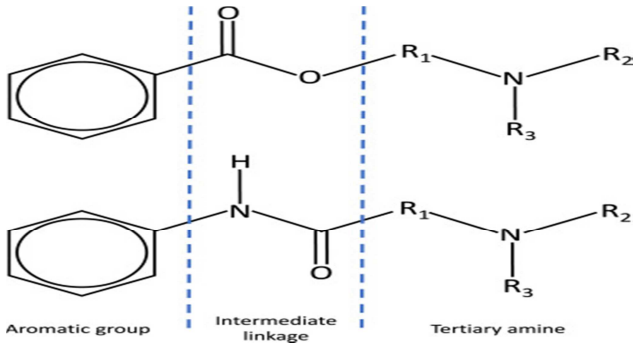


Figure 9: Molecular structure of local anesthetic

Mechanism of action of Local anesthetic:

Local anesthetic agents suppress action potentials in excitable tissues by blocking voltage-gated Na channels. In doing so, they inhibit action potentials in nociceptive fibers and so block the transmission of pain impulses.

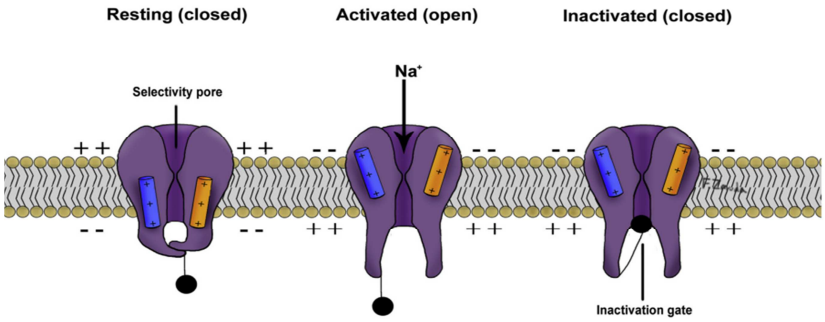


Figure 10: Mechanism of action of local anaesthetics

Lipophilic, unionized local anaesthetic molecules cross the phospholipid neuronal membrane. The molecules dissociate to reach a new equilibrium of ionised and un-ionized moieties, dependent on the intracellular pH and the pKa of the local anesthetic. The ionized form binds to open voltage-gated Na. channels in a reversible and concentration-dependent manner. The binding site for local anaesthetics is located in domain IV, loop S6 and is only accessible when the channel is open. The binding of local anesthetics to open Na channels increases with the frequency of nerve depolarization.

Bound local anaesthetic drug stabilizes the inactivated receptor state, preventing further neuronal transmission. Local anaesthetic nerve block is concentration-dependent. With increased concentrations of local anaesthetic, the peak of the action potential is reduced, the firing threshold increases, impulse conduction is attenuated, and the refractory period is lengthened. Increased concentrations inhibit all nerve conduction.

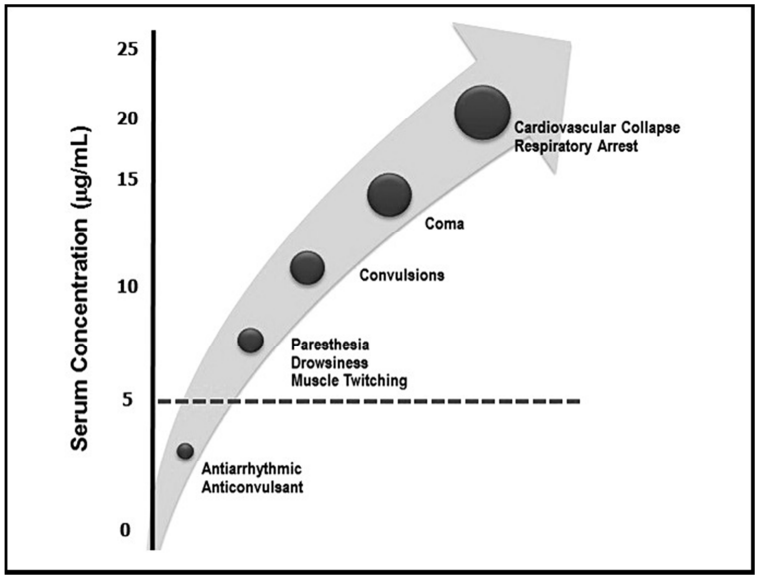


Figure 11: Symptoms of Local anesthetic systemic toxicity

Toxicity:

Local anesthetic systemic toxicity (LAST) is a life-threatening adverse event associated with the increasingly prevalent utilization of local anesthetic (LA) techniques throughout various health care settings, with an incidence currently estimated to be 0.03%, or 0.27 episodes per 1,000 peripheral nerve blocks.

ROPIVACAINE[22,23]

INTRODUCTION

Ropivacaine, a newer and longer-lasting local anesthetic belonging to the amino amide group, was synthesized by Ekenstam in 1957 but wasn't introduced for clinical use until 1996. Chemically akin to bupivacaine and mepivacaine, ropivacaine is a pipecoloxylidide local anesthetic.

Research revealed that butyl derivatives of pipecoloxylidides, such as bupivacaine, posed greater cardiotoxicity risks, leading to numerous cardiac arrests. In response, ropivacaine was developed as a pure S-enantiomeric form of pipecoloxylidides. While ropivacaine has been available internationally for over three decades, its introduction to the Indian market is relatively recent

Ropivacaine is gaining popularity among anesthesiologists and is extensively utilized in various regional anesthesia techniques, including infiltration, peripheral nerve blocks, spinal anesthesia, epidural anesthesia, and caudal epidural blocks in pediatric patients.

CHEMICAL STRUCTURE

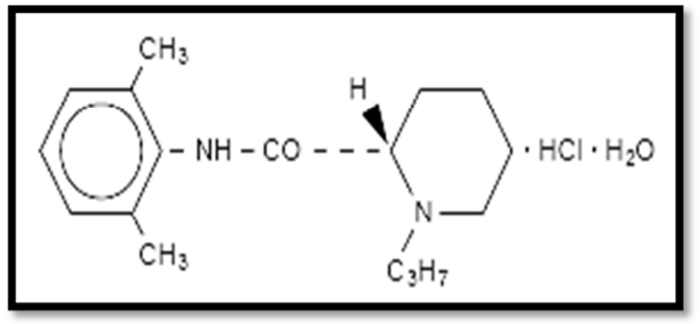


Figure 12: Chemical structure of ropivacaine

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. Its molecular formula is $C_{17}H_{26}N_2O•HCl•H_2O$ and it has a molecular weight of 328.89.

Physical properties

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 8.07 which 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 a propyl (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 of ropivacaine has a significant effect on the block characteristics of ropivacaine as discussed ahead.

Mechanism of action and correlation 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. Block 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 A δ fibres, 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 hemodynamic 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 as 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 I.V administration, ropivacaine has a mean ± SD total plasma clearance of 387 ± 107 mL/min, an unbound plasma clearance of 7.2 ± 1.6 L/min and a renal clearance of 1 mL/min. The mean ± SD terminal half-life is 1.8 ± 0.7 hr and 4.2 ±1.0 hr after I.V and epidural administration respectively.

Pharmacodynamics

Central Nervous System &Cardiovascular System:

Ropivacaine exhibits a higher threshold for both cardiac and neurotoxicity compared to bupivacaine, attributed to its lower lipid solubility and stereo-selective properties. This characteristic applies to both isomers of ropivacaine, which have demonstrated less cardio depressant effects than their respective bupivacaine counterparts in animal studies. In healthy volunteers, CNS toxicity manifests earlier than cardiac toxicity during intravenous infusion.

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 a toxic 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.

e. **Liver and Biliary system toxicity:** Jaundice

f. **Metabolic disorders:** Hypomagnesemia

Advantages over other Local anaesthetics

Ropivacaine offers a more distinct blockade, providing improved differentiation between sensory and motor functions. Consequently, it is favoured for labour analgesia and postoperative pain relief. In comparison to bupivacaine, ropivacaine induces a less intense motor blockade of shorter duration, facilitating earlier patient mobilization and discharge. This leads to reduced morbidity and treatment costs. Additionally, ropivacaine exhibits lower systemic toxicity and a more favourable cardiovascular profile than bupivacaine. Developed as a safer alternative to bupivacaine, ropivacaine maintains the desirable blocking properties of racemic bupivacaine.

Administration is by:

- Local infiltration (post-surgical analgesia)
- Peripheral nerve blocks
- Spinal anesthesia (injected into the CSF to produce anesthesia for orthopaedic surgery, abdominal surgery, or caesarean delivery)
- Epidural anesthesia/analgesia for labour pain
- Caudal block (anesthesia and analgesia below the umbilicus, usually for paediatric surgery

DEXMEDETOMIDINE:

Dexmedetomidine is a selective α2 agonist which has properties of sedation, analgesia, anxiolysis and sympatholytic effect. Since dexmedetomidine is having all these properties, it is used in perioperative period and in intensive care unit for sedation, analgesia. Few studies have shown that dexmedetomidine has anti-shivering properties.

Physical and Chemical Properties:

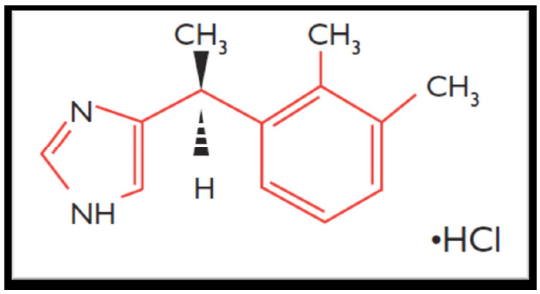


Figure 13: Chemical structure of Dexmedetomidine

Dexmedetomidine chemical formula Dexmedetomidine is a d-enantiomer of medetomidine which belongs to imidazole group of α2 agonists. It has high specificity for α2 receptors than α1 receptors (α2: α1 = 1600:1) when compared to clonidine (α2: α1 = 200:1). Dexmedetomidine is highly water soluble. pKa is about 7.1.

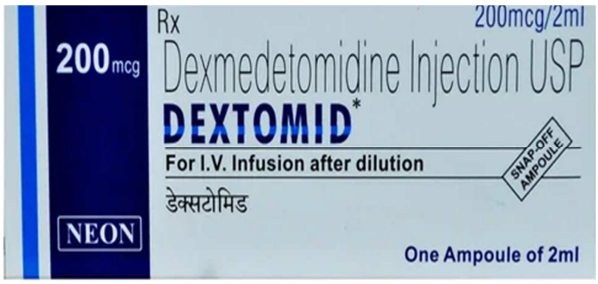


Figure 14: drug ampule of dexmedetomidine.

The presynaptic α adreno receptors which regulates the release of Noradrenaline was designated as α2 adrenoreceptors. These α2 adreno receptors are widely distributed in CNS and peripheral tissues, thereby controlling the modules of sympathetic nervous system. The α2 receptors subdivided into α2A , α2B, α2c etc.; . All these are required for normal regulation of presynaptic neurotransmitter release from the central noradrenergic area and from sympathetic nerves in heart.

Pharmacokinetics:

Dexmedetomidine is a dextro-isomer of medetomidine. It is administered by intravenous route. It is short acting with linear concentration dependent kinetics.

Dosing and administration:

The pharmacodynamic effects of the drug are achieved at a plasma concentration of 0.5-1.2ng/dl. The dose of the drug approved by FDA is a loading dose of 1mcg/kg body weight over a period of 10 minutes followed by a maintenance dose of continuous intravenous infusion of 0.2 to 0.7 mcg/kg/hr.

The rate flow of this infusion is titrated according to required level of sedation and by careful monitoring of hemodynamics of the patient. Dexmedetomidine is used as a good sedative agent in ophthalmic surgeries. In these surgeries the drug is administrated intravenously as a loading dose of 0.5 mcg/kg followed by a maintenance dose of 0.6 mcg/kg/hr.

Distribution:

Dexmedetomidine is significantly bound to plasma protein (94%). The pharmacokinetics of this drug is explained as a “two-compartment model”. After intravenous injection it is rapidly distributed with a distribution half-life of 6 minutes. The elimination half-life of the drug is 2 to 2.5 hours. The steady state volume of distribution is estimated as approximately 68L to72 L.

Metabolism and elimination:

The dexmedetomidine is mostly metabolized in the liver by the cytochrome P450 enzymes by glucuronidation and biotransformation. There are various metabolic pathways by which dexmedetomidine is metabolized

1. N-glucuronidation to inactive metabolites (41%).
2. N-methylation to produce 3-hydroxy N-methyl dexmedetomidine (21%)
3. Hydroxylation followed by conjugation.
4. Conjugation and N-methylation.
5. Hydroxylation followed by conjugation.

Most of the drug undergoes complete biotransformation which results in minor quantity of drug to be excreted in urine and feces as unconjugated form. The elimination half-life of the drug is approximately 2 hours with an average clearance of 45L/hour in adults. The pharmacokinetics of the drug are impaired in patients with hepatic and renal diseases. In hepatic diseases, there is an increase in half-life, volume of distribution and as well as decrease in clearance and protein binding of the drug. In renal diseases, there is a decrease in terminal elimination half-life, but no change in clearance and volume of distribution.

Table 2: PHARMACOKINETICS OF DEXMEDETOMIDINE

Molecular weight	236.7 Daltons
Lipid solubility	30
Distribution half life	6 minutes
Protein binding	94%
Volume of distribution	118 L
Elimination t1/2	120-180 minutes
Context sensitive half life	4- 250 minutes

Mechanism of action:

Dexmedetomidine is a potent α2 agonist. The α2 receptors are a part of both central nervous system and peripheral nervous system. These receptors are also present in liver, kidney and pancreas. The site of action of these receptors are presynaptic, post synaptic and extra synaptic regions among which presynaptic site is of major concern. So, depending on site of action of these receptors, various responses are evaluated. The presynaptic site is the region where modulation of release of Nor-adrenaline and ATP occurs. Since it is a potent α2 agonist; it mainly acts on presynaptic region and causes inhibition of release of nor-adrenaline. Its main action

is to inhibit the neuronal firing in the brain and spinal cord by action on the α2 receptors and thus leading to bradycardia, hypotension, sedation and analgesia.

Pharmacodynamics:

Dexmedetomidine after its initial dose administration exerts a brief biphasic pattern of cardiovascular response. This response is seen after a loading dose of 1microgram/kg of body weight. There is initial increase in blood pressure followed by reflex bradycardia. This initial increase in blood pressure is because of stimulation of α2b receptors which are present in smooth muscles. This is followed by decrease in blood pressure which is because of central sympathetic system outflow blockade.

Dexmedetomidine inhibits the release of noradrenaline, thus leading to further hypotension and bradycardia. Postoperative bradycardia is commonly noticed with dexmedetomidine injection. Cautious use of dexmedetomidine to patients with poor left ventricular function should be considered.

Dexmedetomidine sedative effects are achieved by action on α2 adreno receptor located on postsynaptic membrane. It inhibits the G-protein which leads to increase in conductance through the potassium channels. The sedative effect of dexmedetomidine is achieved by action on locus coeruleus located in brain stem.

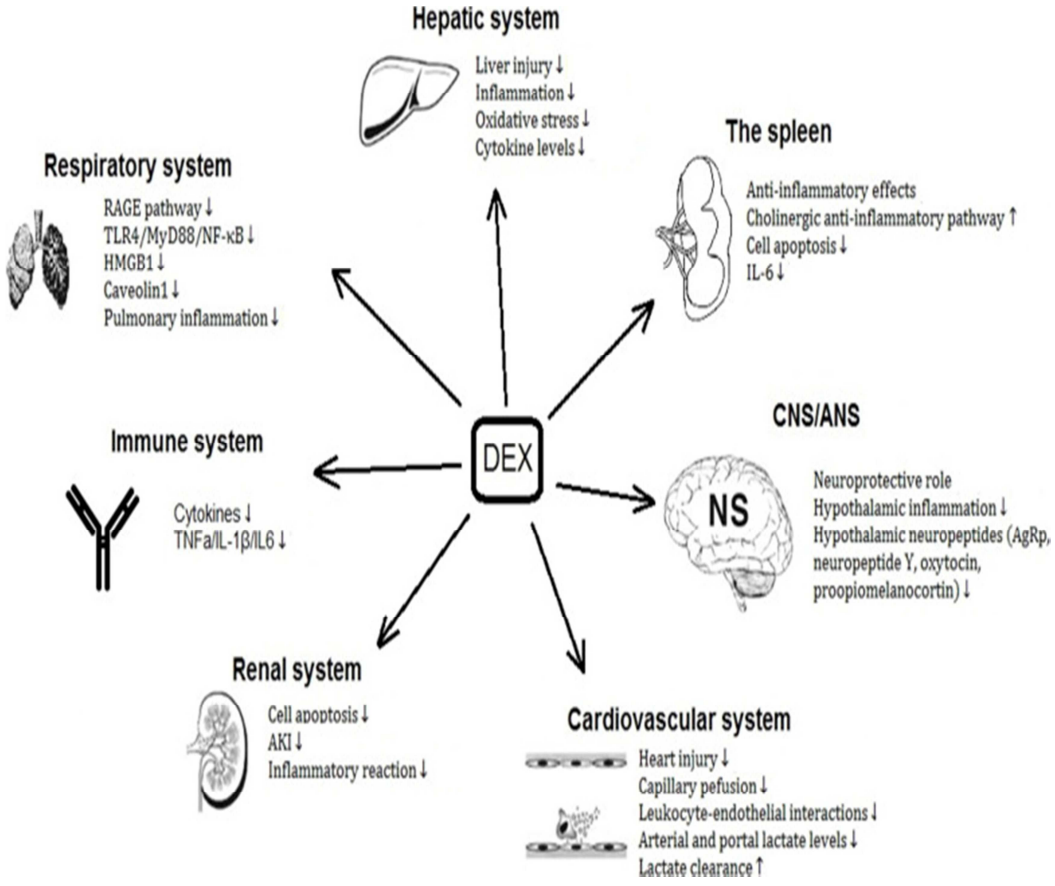


Figure 15: Dexmedetomidine clinical effects.

Uses:

The uses of the drug dexmedetomidine are as follows:

1. Sedation of intubated patients in ICU. (NOTE: it should be used more than 24 hours.)
2. Sedation of surgery patients without being intubated.
3. Reduction of use of opioids and other anesthetic agents
4. Anxiolytic.
5. Before performing laryngoscopy to reduce intubation response.

6. Anti-shivering property. (Dexmedetomidine acts as anti-shivering agent by binding to α2 receptors, thus mediate vasoconstriction and acts as anti-shivering agent. Dexmedetomidine reduces vasoconstriction and shivering threshold by acting on central thermoregulatory mechanisms rather than peripheral neurotransmitter.)
7. As an adjuvant to spinal anesthesia and peripheral nerve blocks.

Adverse effects:

1) Bradycardia
2) Sinus arrest.
3) Hypotension.
4) Transient hypertension.
5) Hypoxia
6) Nausea and vomiting.
7) Anemia
8) Atrial fibrillations
9) Fever, tachycardia.
10) Headache, agitation and nervousness (occurs if the drug is used for more than 24 hours.)

Contraindications:

1) Drug hypersensitivity.
2) Cardiac dysfunction (mainly left ventricular dysfunction mainly)
3) Advanced cardiac blocks.
4) Shock/ hypovolemia.

MATERIALS AND METHODS

The present study titled, **"EVALUATION OF THE EFFECT OF DEXMEDETOMIDINE AS ADJUVANT TO ROPIVACAINE IN ULTRASOUND GUIDED POPLITEAL SCIATIC NERVE BLOCK IN BELOW KNEE SURGERY-A ONE YEAR HOSPITAL BASED DOUBLE BLIND RANDOMISED CLINICAL TRIAL",** was conducted on patients between the aged between 18 to 70years, of any gender, under ASA I-III grade ,who were undergoing below-knee surgery at KLE's DR. Prabhakar Kore Charitable Hospital and Medical Research Centre, Nehru Nagar, Belagavi-10 between July 2022 and June 2023.

Source of Data: Patients with age between 18-70 years of either gender, belonging to American Society of Anaesthesiologists (ASA) grade I- III ,who were undergoing below knee surgery at KLE Dr. Prabhakar Kore Charitable hospital and medical research centre, Nehru Nagar, Belagavi -10 over a period of One year.

Study Design: A One Year Hospital Based Double blind Randomized clinical trial.

Study Period: One year

Sample Size: Total sample size:60

Formula for minimum size of sample is

$$\mathbf{n} = \frac{(z_\alpha + z_\beta)^2 (s_1^2 + s_2^2)}{(\bar{X}_1 - \bar{X}_2)^2}$$

considering mean and standard deviation

Ref:by taking values from the reference article by Hu et al in 2017[14], "Dexmedetomidine Added to Local Anesthetic Mixture of Lidocaine and Ropivacaine Enhances Onset and Prolongs Duration of a Popliteal Approach to Sciatic Nerve Blockade"

Where:

- S1 = 206
- S2 = 114
- $z\alpha = 1.96$ (for 5% significance level)
- $z\beta = 0.84$ (for 80% power)
- $\Delta = 935 - 758 = 177$ (difference in means)

Plugging in these values:

$n = [(206^2 + 114^2) * (1.96 + 0.84)^2] / [177^2] \approx 14$

So, the initial sample size obtained based on the given information was 14. To enhance the study's confirmatory power, the sample size was increased to 60 with each group now containing 30 cases.

Sampling technique and randomization:

Patients were randomly assigned to one of the two groups according to a computer-generated randomization table once they had satisfied the inclusion and exclusion criteria and provided their informed consent.

Group A (Dexmed group)- received 0.75%Ropivacaine (15 mL) +0.5mL(50mcg) Dexmedetomidine.

Group B (control group)- received 0.75% Ropivacaine(15mL) +0.5mL saline.

Inclusion Criteria

- Patients with ASA grade I - III.
- Patients with Age 18- 70 yrs.
- Patients undergoing below knee surgeries
- Patients who provide consent

Exclusion Criteria

- Patients with ASA grade IV or more
- Unable to give consent.
- Allergy to study drug
- Any contraindication for regional anaesthesia
- Pregnant patients
- Patients with an infection at the injection location

METHODOLOGY:

Study protocol:

Sixty patients who satisfied the exclusion and inclusion criteria undergoing below-knee surgery were selected for our study, once the Institution Review Board approval, the ethical committee clearance, CTRI registration and written informed consent were obtained. A computer-generated randomization table was used to randomly assign each patient to either groups to receive study drugs.

Group A (Dexmed)- 0.75% Ropivacaine(15mL)+0.5mL(50mcg) Dexmedetomidine.

Group B(control)- 0.75% Ropivacaine(15 mL) +0.5mL Normal saline

Neither the patient nor the observer who followed up the patient knew which drug the patient has received during the study to eliminate bias.

A through pre-anesthetic evaluation and routine investigations like complete blood count, FBS, PT/INR and viral serology were done. X-ray chest &ECG taken when patient's age more than 40years

All patients were advised to be nil by mouth for at least six hours prior to surgery.

Using iv cannula size 18 or 20 G IV fluid was started after cannulation. Monitoring devices according to ASA standards were attached to the patient before induction of anaesthesia. These included blood pressure(non-invasive), pulse oximeter and ECG. Heart rate, Blood pressure parameters and Saturation were noted. Oxygen supplied via nasal prongs throughout the procedure at 5L/min.

Under aseptic precautions, a preliminary scan was performed using Sonosite Ultrasound machine kept across the limb that has to be blocked. A linear probe was utilized for locating Sciatic nerve with patient in prone position. Tibial Nerve and Common peroneal nerve was seen separately- lying superficially and posteriorly to popliteal artery when the popliteal fossa was scanned. By moving the probe upwards these nerves converging to form the Sciatic nerve was seen. Using in-plane needling technique ,23 G Quincke's spinal needle was advanced at this point. Using ultra-sonographic direct imaging and after negative aspiration, the LA mixture was deposited around the Sciatic nerve close to its emergence as Tibial and common peroneal nerve.

An experienced anaesthesiologist performed all the blocks.

BLOCK EVALUATION: Block evaluation was done every 5 mins for 20 mins after local anaesthetic injection. Sensory block checked on dermatomes of Tibial and common peroneal nerve

Onset of Nerve blockade was assessed by cold swab testing

A cold swab test with 3-point scale was used to grade the sensory block[24]:

0-anaesthesia (Patients perceives neither touch nor cold)

1-analgesia ("patient perceives only touch, not cold")

2-no block (perceives both touch and cold)

Grade-0 was considered successful sensory block

Motor block evaluation was done using Modified Bromage scale[25] assessing movement of Toes and ankle(plantar or dorsiflexion).Grade ≤ 2 was taken as successful nerve block.

Grades

4- No block in relevant muscles of foot

3- Muscle strength decreased, can move foot against resistance

2- can lift foot against gravity, but not against resistance

1- trembling of muscles of foot

0- No movement

The patients considered as block failure and excluded from the study when adequate surgical anaesthesia is not achieved after 20 minutes of administration of block.

At regular intervals of 10 minutes, vitals were monitored and recorded. Any

Fall in Blood pressure greater than 20% from the baseline was considered significant and managed with administration of Inj.Mephentermine or Ephedrine.Fall in heart rate more than 20% from baseline was managed by Inj.Atropine(0.06mg)

Visual Analog scale(VAS) Score was used for post op pain assessment. When VAS ≥ 3, Patient received 15mg/kg Intravenous Paracetamol-which was repeated if required with a maximum dose of 4 g per day.

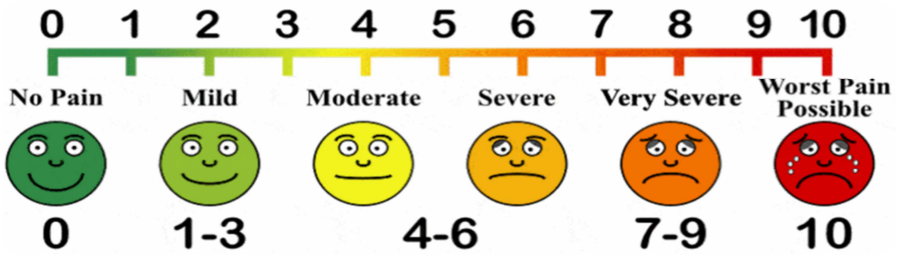


Figure 16:Visual Analog Scale

Time to first dose of analgesic and its total requirement within 24 hours of surgery was recorded. Any adverse effect of the study drug, if any was also recorded. Every patient's vital parameters (HR,SBP,DBP,MAP) were documented at baseline and every 10 minutes till end of surgery.

STATISTICAL ANALYSIS:

METHODS:

Data is analysed using statistical software R version 4.4.0. and Microsoft Excel. Categorical variables given in the form of frequency tables. Continuous variables given in Mean ± SD / Median (Min, Max) form. Chi square test is used to check the association of categorical variables with groups. Normality of variable is checked by Shapiro Wilk test and QQ plot. If data follows normal distribution, parametric tests will be used. Otherwise, non-parametric tests will be used. Two sample t test is used to compare the means of variables over groups. Mann Whitney U test is used to compared the distribution of variables over groups. Friedman test is used to compare the distribution of variables over time. P-value less than or equal to 0.05 indicates statistical significance.

RESULTS:

Data shows measurements on 60 subjects which are split into groups of 30 subjects each forming two groups.

Group A (Dexmed)- 0.75% Ropivacaine(15mL) +0.5mL(50mcg) Dexmedetomidine.

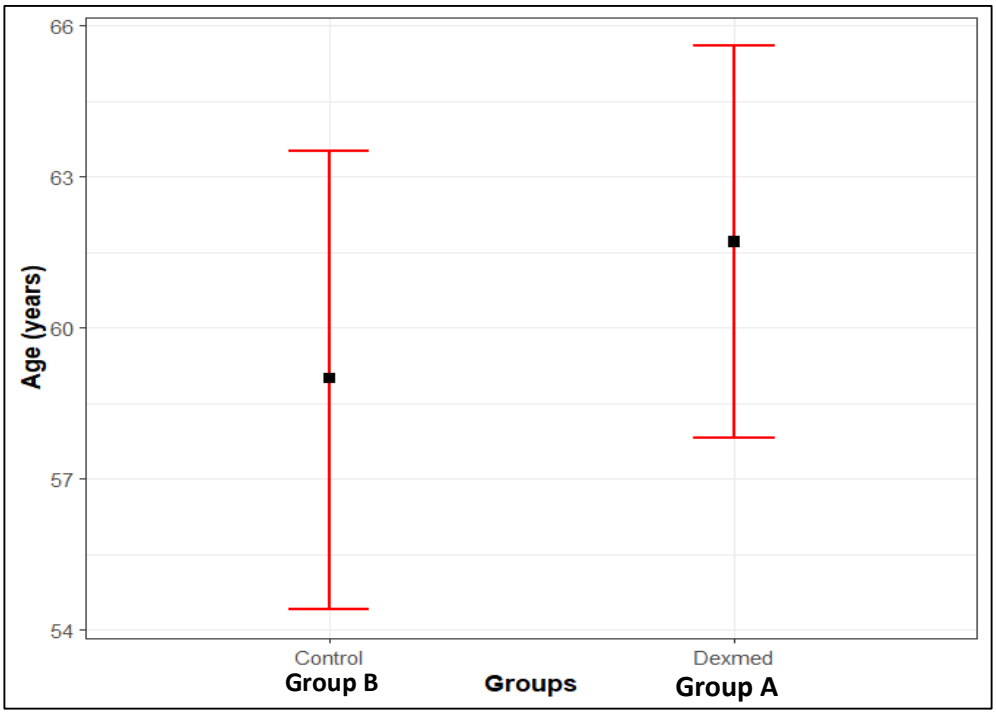
Group B (control) - 0.75% Ropivacaine (15 mL) +0.5mL Normal saline

The following table gives the demographic details comparison over groups.

Table 3: Age comparison

Variables		**Groups**		**p-value**
		Group A (Dexmedetomidine)	**Group B** (Control)	
Age (years)	Mean ± SD	61.7 ± 10.35	58.97 ± 12.17	0.3526
	Median (Min, Max)	62.5 (40, 83)	60 (32, 83)	

The age of participants in the Control group had a mean of 58.97 years with a standard deviation of 12.17, and a median age of 60 years ranging from 32 to 83 years. In the Dexmed group, the mean age was slightly higher at 61.7 years with a standard deviation of 10.35, and a median of 62.5 years ranging from 40 to 83 years. From two sample t test, it is observed that there is no significant difference in age over groups (p-value = 0.3526).

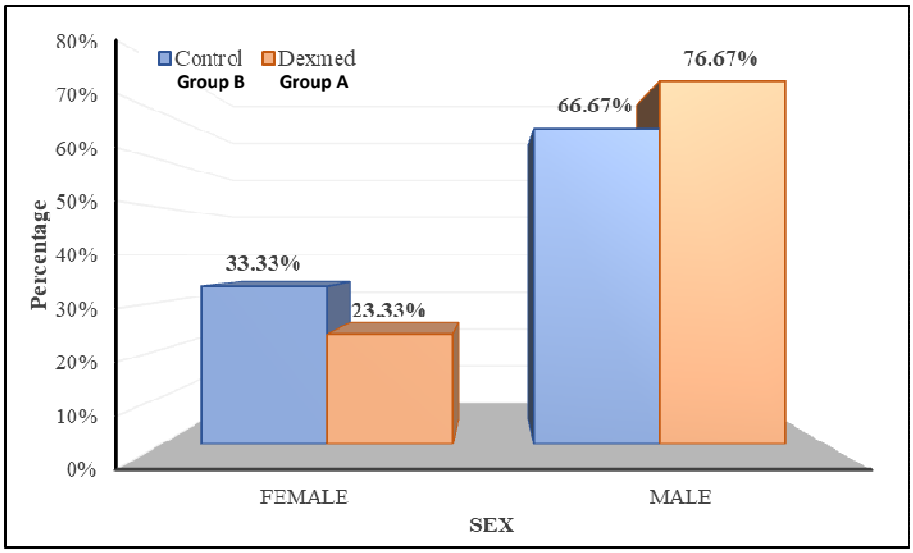


Graph 1: Age comparison

Table 4: Sex distribution

Variables	Sub Category	Groups		p-value
		Group A (Dexmedetomidine)	Group B (Control)	
Sex	Female	7 (23.33%)	10 (33.33%)	0.3901
	Male	23 (76.67%)	20 (66.67%)	

Regarding sex distribution, the Control group consisted of 10 (33.33%) females and 20 (66.67%) males, while the Dexmed group had 7 (23.33%) females and 23 (76.67%) males. From Chi square test, it is observed that, there is no significant difference in the distribution of sex over groups (p-value = 0.3901).

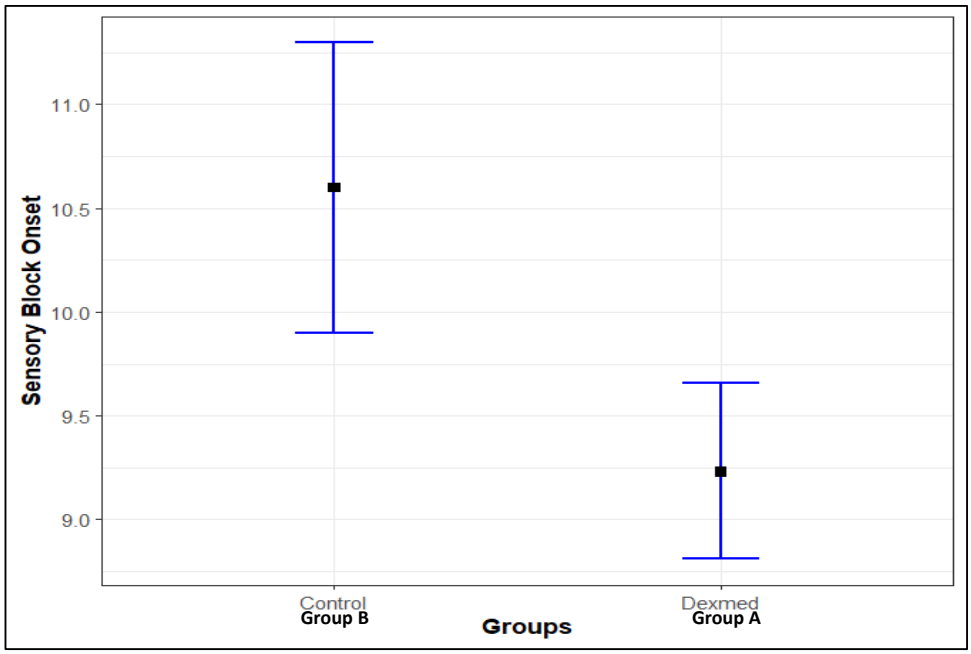


Graph 2: Sex distribution

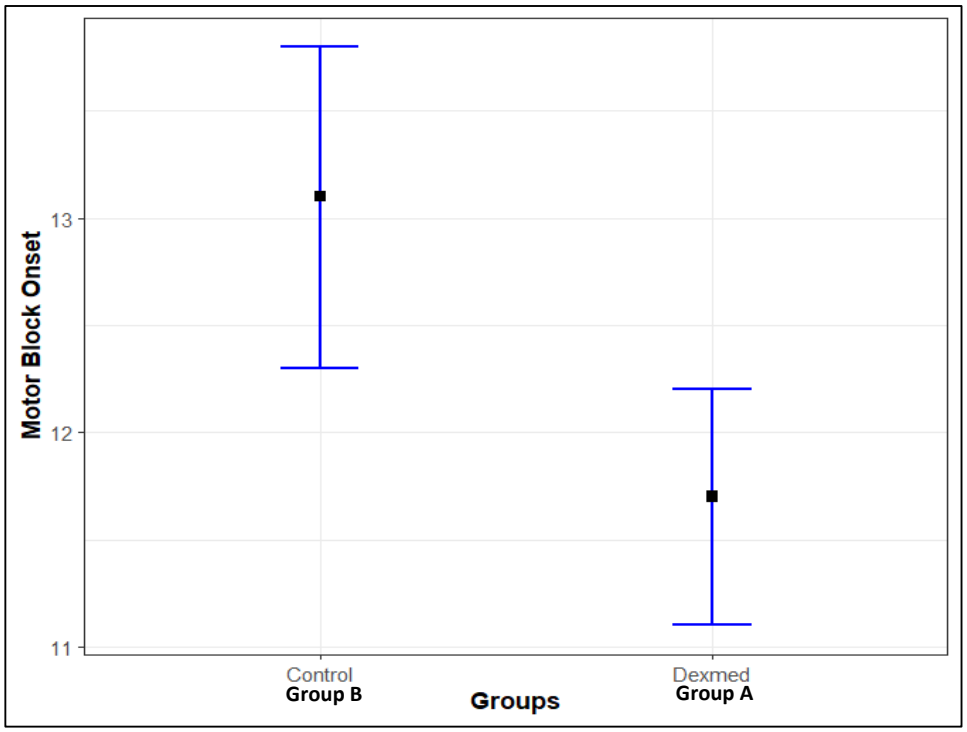
Table 5: Onset -sensory and motor blockade

Variables	Groups		p-value
	Group A (Dexmedetomidine)	**Group B** (Control)	
Sensory Block Onset(min)	9.23 ± 1.14 9 (7, 12)	10.6 ± 1.89 10 (7, 15)	**0.0016**
Motor Block Onset(min)	11.67 ± 1.4 12 (10, 15)	13.07 ± 2.02 13 (10, 17)	**0.0063**

The Dexmed group showed a significantly faster onset of sensory block with an average of 9.23 ± 1.14 minutes compared to 10.6 ± 1.89 minutes in the Control group (p-value = 0.0016). Similarly, the Dexmed group experienced a quicker onset of motor block at 11.67 ± 1.4 minutes compared to 13.07 ± 2.02 minutes in the Control group (p-value = 0.0063).



Graph 3: Sensory block onset

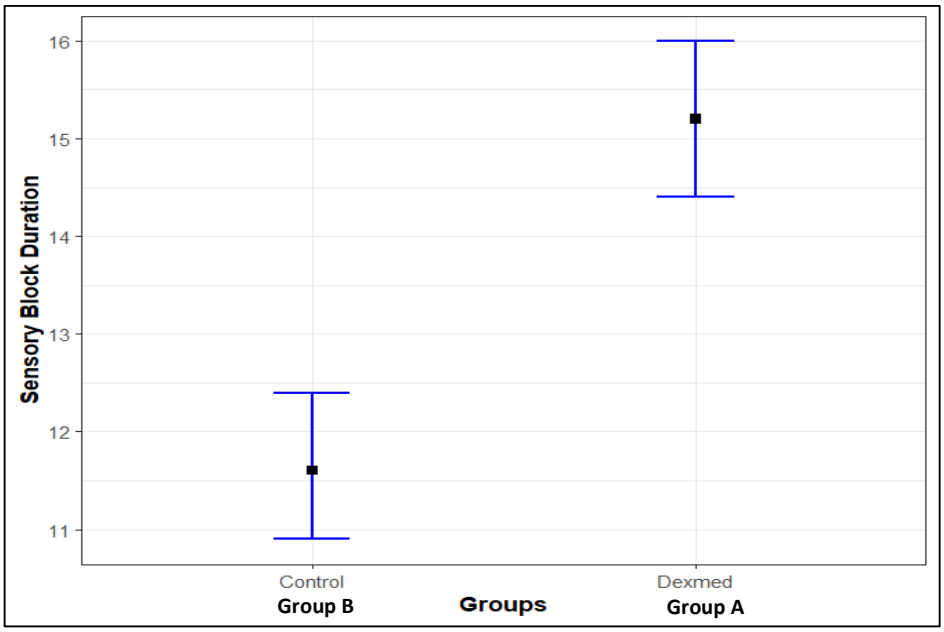


Graph 4: Motor block onset

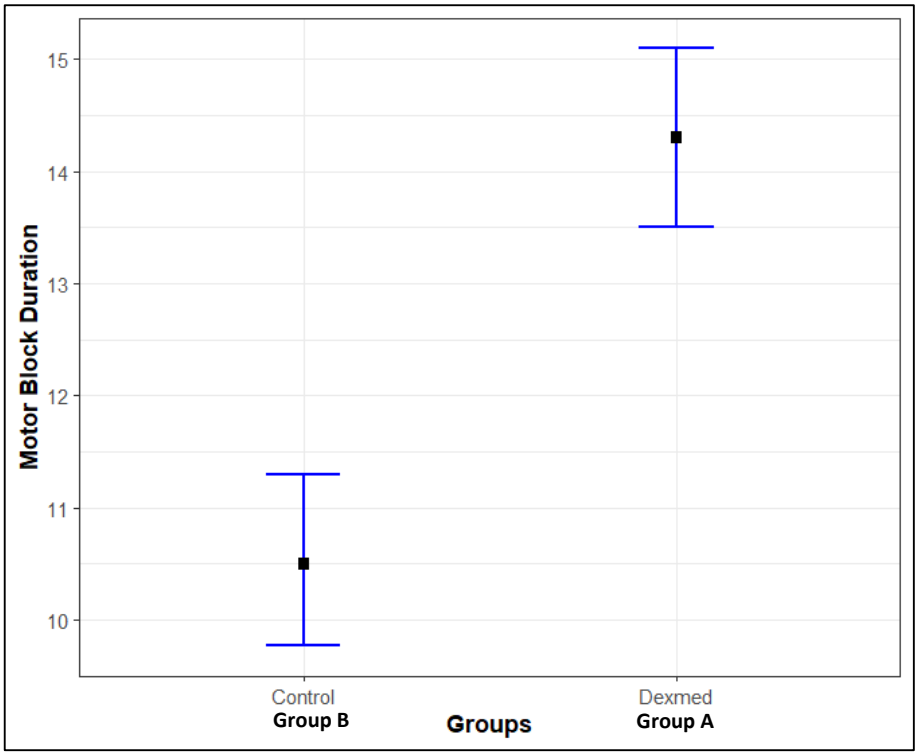
Table 6: Block duration

Variables	Groups		p-value
	Group A (Dexmedetomidine)	**Group B** (Control)	
Sensory Block Duration(hours)	15.18 ± 2.16 15 (10, 19.5)	11.65 ± 1.96 11.5 (8, 16)	**< 0.001**
Motor Block Duration(hours)	14.3 ± 2.2 14 (9, 19)	10.52 ± 1.99 10 (7.5, 15)	**< 0.001**

The duration of the sensory block was significantly longer in the Dexmed group, averaging 15.18 ± 2.16 hours, compared to 11.65 ± 1.96 hours in the Control group (p-value < 0.001). The duration of the motor block was also significantly longer in the Dexmed group, with an average duration of 14.3 ± 2.2 hours, compared to 10.52 ± 1.99 hours in the Control group (p-value < 0.001). These results indicate that Dexmed has a significant effect on both the onset and duration of sensory and motor blocks, leading to faster onset and longer duration of block compared to the Control group.



Graph 5: Sensory block duration



Graph 6: Motor block duration

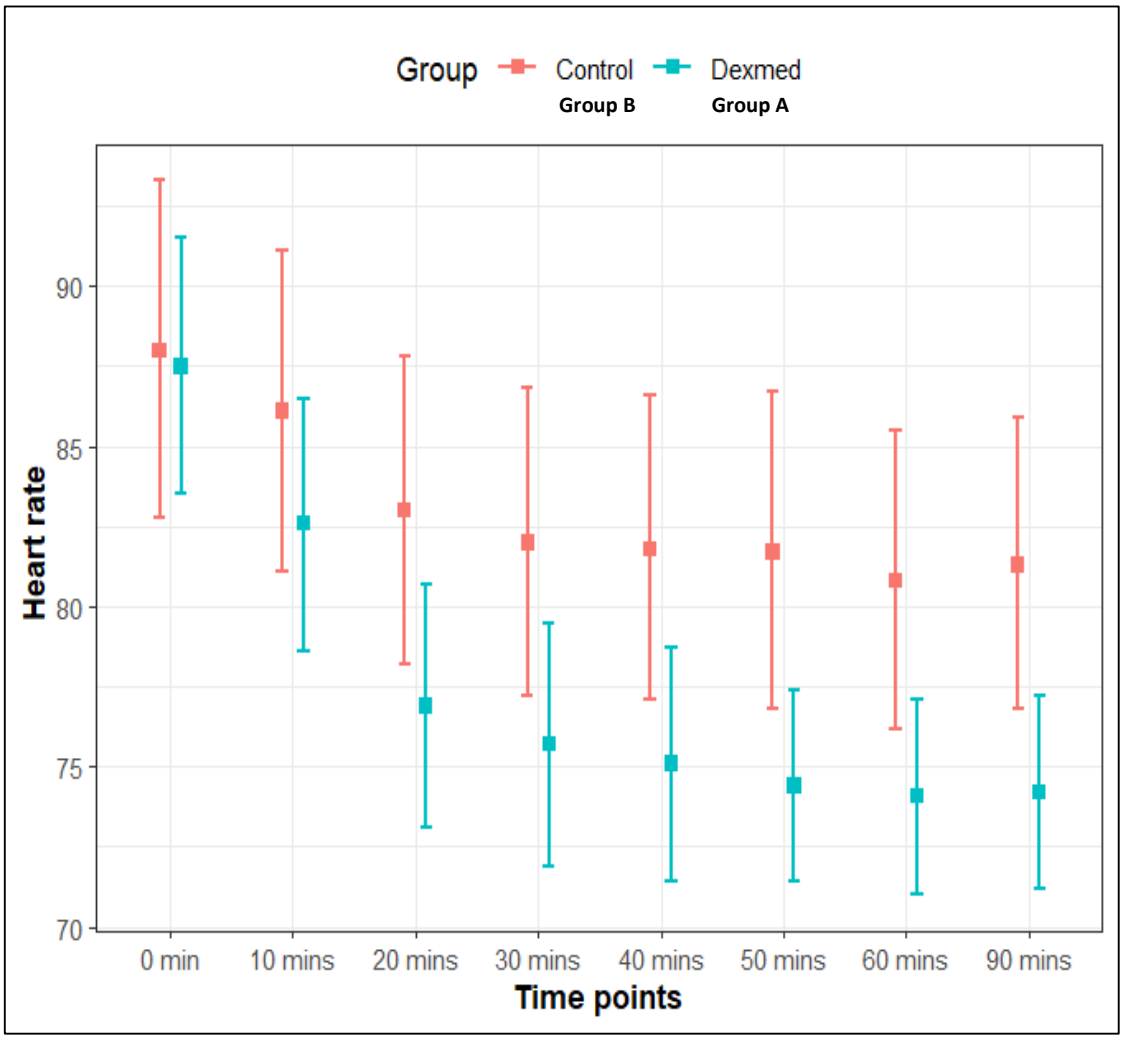
Table 7: Heart rate (bpm)

Heart rate	Groups		p-value
	Group A (Dexmedetomidine)	**Group B** (Control)	
0 min	87.47 ± 10.73 88 (60, 118)	88.03 ± 14.05 90 (64, 120)	0.4709
10 mins	82.57 ± 10.58 80 (58, 106)	86.1 ± 13.41 88 (63, 118)	0.2619
20 mins	76.93 ± 10.18 75 (58, 102)	83 ± 12.74 84 (59, 116)	0.0982
30 mins	75.73 ± 10.17 75 (54, 100)	82 ± 12.92 84 (61, 113)	**0.0412**
40 mins	75.07 ± 9.79 73 (50, 102)	81.83 ± 12.64 82.5 (60, 115)	**0.0240**
50 mins	74.43 ± 8 74 (54, 98)	81.73 ± 13.26 81.5 (62, 119)	**0.0120**
60 mins	74.07 ± 8.25 73 (56, 96)	80.83 ± 12.39 81 (62, 110)	**0.0156**
90 mins	74.2 ± 8.13 72 (56, 98)	81.33 ± 12.17 82 (61, 102)	**0.0098**
p-value	**< 0.001**	**< 0.001**	-

Initially, at 0 minutes, the average heart rate showed similarity (p-value = 0.4709. Over time, the Dexmed group consistently showed lower average heart rates compared to the Control group. Significant differences in heart rate between the groups were observed starting from 30 minutes onward, with p-values indicating

statistical significance at 30 minutes (p-value = 0.0412), 40 minutes (p-value = 0.0240), 50 minutes (p-value = 0.0120), 60 minutes (p-value = 0.0156), and 90 minutes (p-value = 0.0098).

Friedman's test showed a statistically significant change in heart rate over time within each group (p-value < 0.001).



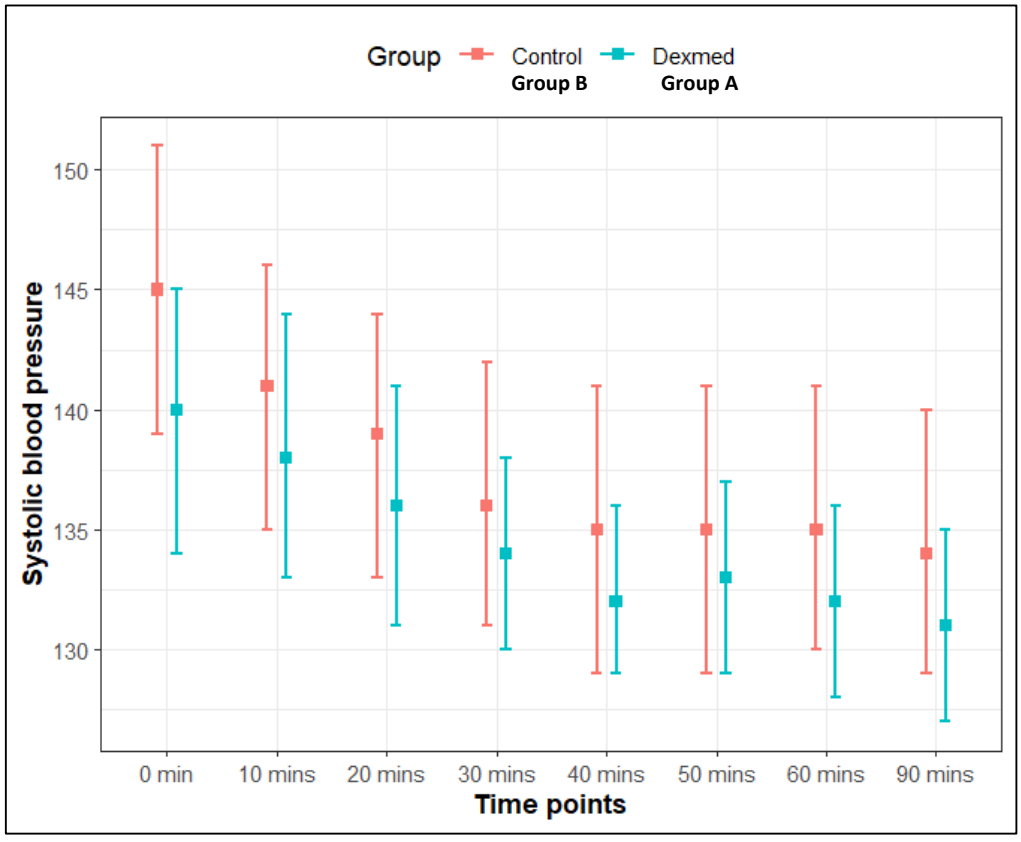
Graph 7: Heart rate

Table 8: Systolic Blood Pressure(mmHg)

SBP	Groups		p-value
	Group A (Dexmedetomidine)	**Group B** (Control)	
0 min	139.77 ± 14.9 139 (102, 170)	145.4 ± 16.14 146 (122, 175)	0.1655
10 mins	138.47 ± 14.52 137 (101, 168)	140.63 ± 14.38 140 (116, 174)	0.5141
20 mins	136.1 ± 12.61 136 (98, 158)	138.77 ± 14.26 138 (111, 172)	0.4628
30 mins	133.67 ± 10.87 136 (102, 151)	136.47 ± 14.78 136 (107, 172)	0.4067
40 mins	132.47 ± 10.4 133 (102, 150)	135.34 ± 15.48 133 (110, 177)	0.5942
50 mins	133.2 ± 10.59 132.5 (102, 150)	135.24 ± 15.16 134 (107, 173)	0.5501
60 mins	131.9 ± 10.31 130 (103, 150)	135.07 ± 14.59 134 (108, 174)	0.3358
90 mins	131.27 ± 10.75 131 (102, 150)	134.2 ± 15.24 133 (107, 173)	0.4531
p-value	**< 0.001**	**< 0.001**	-

At 0 minutes, the average SBP was 145.4 ± 16.14 mmHg in the Control group and 139.77 ± 14.9 mmHg in the Dexmed group, with no significant difference (p-value = 0.1655). Throughout the time points measured (10, 20, 30, 40, 50, 60, and 90 minutes), the SBP in the Dexmed group remained consistently lower than in the Control group, but the differences were not statistically significant at any single time point.

Friedman's test showed a statistically significant change in SBP over time within each group (p-value < 0.001).



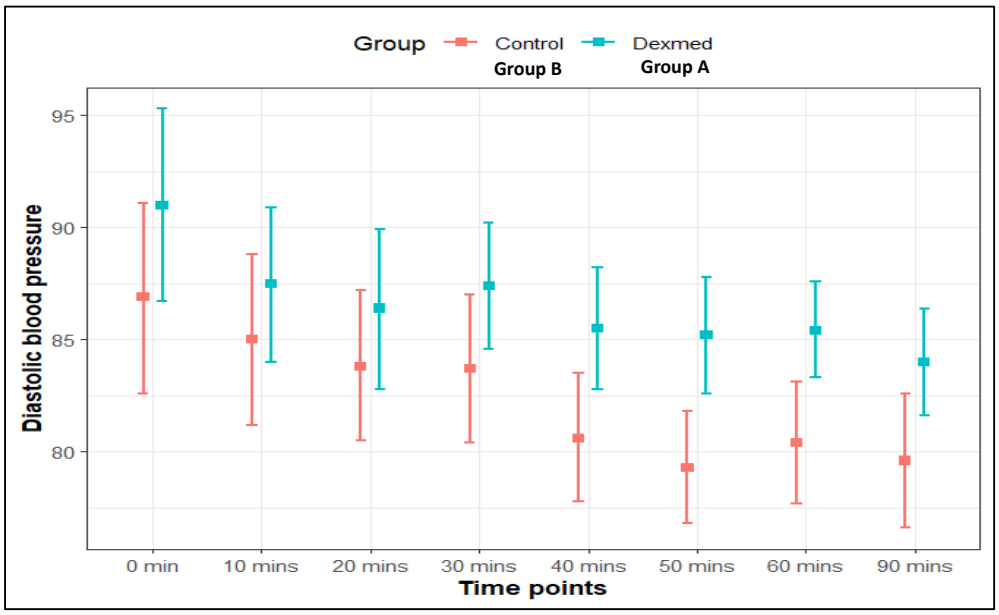
Graph 8: Systolic BP

Table 9: Diastolic BP(mmHg)

DBP	Groups		p-value
	Group A (Dexmedetomidine)	**Group B** (Control)	
0 min	86.87 ± 11.47 85.5 (66, 110)	90.97 ± 11.55 98 (70, 102)	0.1270
10 mins	84.97 ± 10.14 85 (66, 110)	87.47 ± 9.21 86.5 (66, 102)	0.3217
20 mins	83.83 ± 8.97 83 (65, 102)	86.37 ± 9.52 86 (65, 102)	0.2932
30 mins	83.67 ± 8.81 83.5 (61, 98)	87.37 ± 7.46 87 (64, 98)	0.0962
40 mins	80.62 ± 7.5 82 (56, 92)	85.53 ± 7.2 86 (70, 100)	0.0811
50 mins	79.34 ± 6.58 82 (61, 90)	85.2 ± 7 84 (70, 98)	0.0716
60 mins	80.37 ± 7.23 81 (68, 96)	85.43 ± 5.68 86 (68, 96)	0.0836
90 mins	79.6 ± 7.94 81 (61, 94)	84 ± 6.47 86 (70, 95)	0.0921
p-value	**< 0.001**	**< 0.001^{F*}**	-

Initially, at 0 minutes, the average DBP was 86.87 ± 11.47 mmHg in the Control group and 90.97 ± 11.55 mmHg in the Dexmed group, with no significant difference (p-value = 0.1270). Over the course of the study, significant differences in DBP between the groups emerged at several time points: 40 minutes (p-value = 0.0111), 50 minutes (p-value = 0.0016), 60 minutes (p-value = 0.0036), and 90 minutes (p-value = 0.0221), with the Dexmed group generally showing higher DBP values compared to the Control group.

Friedman's test showed a statistically significant change in DBP over time within each group (p-value < 0.001).



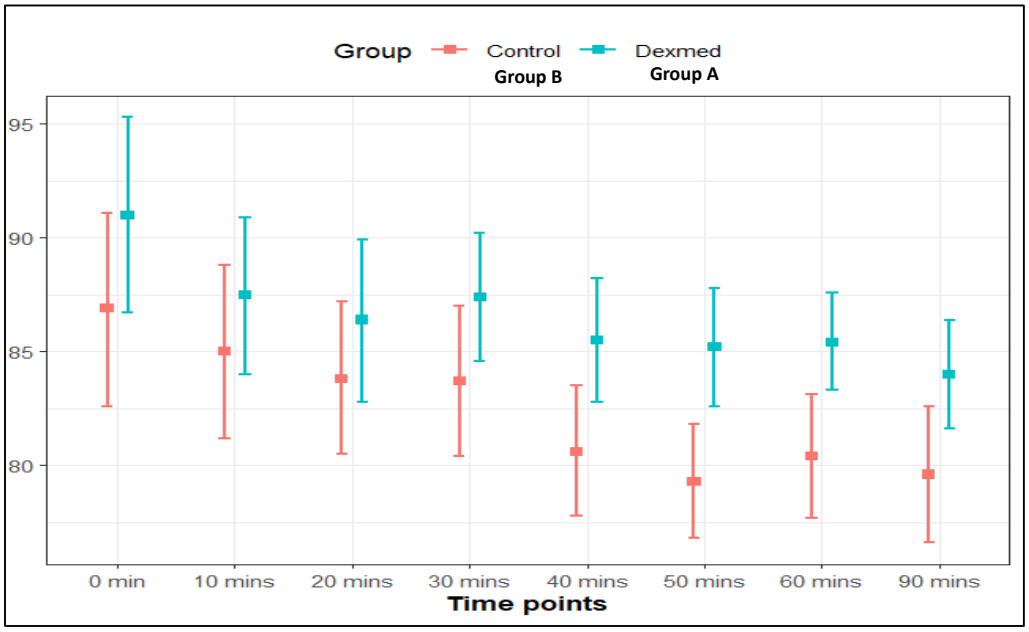
Graph 9: Diastolic BP

Table 10: MAP(mmHg)

MAP	**Groups**		
	Group A (Dexmedetomidine)	**Group B** (Control)	**p-value**
0 min	100.47 ± 11.44 100 (83, 120)	104.55 ± 12.13 111.5 (84, 125)	0.2357
10 min	92.45 ± 9.81 93 (77, 116)	93.17 ± 12.48 97 (70, 115)	0.5425
20 min	91.8 ± 9.78 90 (70, 109)	94.05 ± 8.43 94 (81, 114)	0.6372
30 min	91.72 ± 8.51 93 (77, 107)	93.33 ± 7.72 95 (81, 112)	0.2378
40 min	92.77 ± 8.16 94 (78, 107)	95.75 ± 4.68 98 (88, 103)	0.4632
50 min	89.97 ± 6.64 94 (75, 98)	97.14 ± 3.72 96 (90, 104)	0.6521
60 min	92.64 ± 7.31 93 (76, 102)	95.05 ± 4.74 93.5 (91, 105)	0.1739
90 min	96.22 ± 5.61 92 (82, 101)	95 ± 0 95 (95, 95)	0.5343
p-value	**< 0.001^{F}***	**< 0.001^{F}***	

Initially, at 0 minutes, the average DBP was 104.55 ± 12.13mmHg in the Control group and 100.47 ± 11.44 mmHg in the Dexmed group, with no significant difference (p-value = 0.2357). Over the course of the study, Throughout the time points measured (10, 20, 30, 40, 50, 60, and 90 minutes), the MAP in the Dexmed group remained consistently lower than in the Control group, but the differences were not statistically significant at any single time point.

Friedman's test showed a statistically significant change in DBP over time within each group (p-value < 0.001).

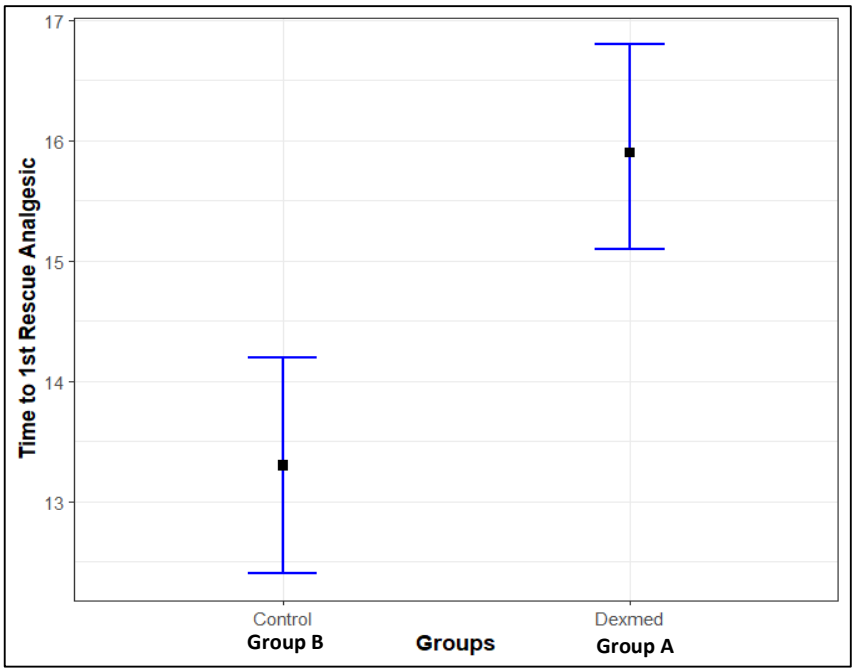


Graph 10 Mean Arterial Pressure

Table 11: Time to 1st rescue Analgesic Requirement

Variables	Sub Category	Groups		p-value
		Group A (Dexmedetomidine)	**Group B** (Control)	
Time to 1st Rescue Analgesic(hours)	Mean ± SD	15.93 ± 2.33	13.33 ± 2.4	**< 0.001**
	Median (Min, Max)	16 (11, 20)	13 (10, 18)	

The time to the first rescue analgesic was significantly longer in the Dexmed group, with an average of 15.93 ± 2.33 compared to 13.33 ± 2.4 in the Control group (p-value < 0.001).

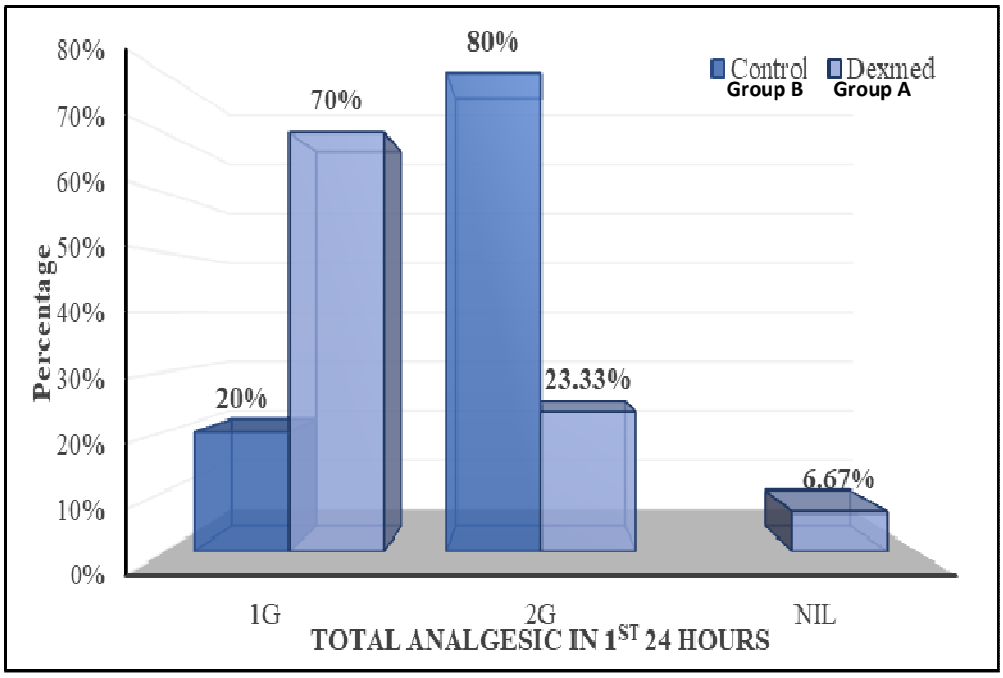


Graph 11: Time to 1st rescue analgesic

Table 12: Total Analgesic Requirement

Variables		Groups		p-value
		Group A (Dexmedetomidine)	**Group B** (Control)	
Total Analgesic in 1st 24 hours	nil	2 (6.67%)	0	
	1g Inj.Paracetamol	21 (70%)	6 (20%)	**< 0.001**
	2g Inj.Paracetamol	7 (23.33%)	24 (80%)	

Regarding the total analgesic consumption in the first 24 hours, a significantly higher proportion of patients in the Dexmed group required only 1 gram of analgesic (70%) compared to the Control group (20%), while a larger proportion of patients in the Control group required 2 grams of analgesic (80%) compared to the Dexmed group (23.33%). Additionally, 6.67% of patients in the Dexmed group required no analgesic at all, whereas all patients in the Control group required analgesics. These differences were statistically significant (p-value < 0.001).



Graph 12: Total analgesic in 1st 24 hours

Overall, the results indicate that patients in the Dexmed group had a longer duration before needing their first rescue analgesic and required less total analgesic in the first 24 hours compared to the Control group.

DISCUSSION

In current clinical practice, Peripheral nerve block is emerging with its potential benefits of providing deep analgesia both intraoperatively and post-operatively avoiding any systemic complications. Hence useful in medically compromised patients.

Popliteal sciatic nerve block is a frequently utilised regional anaesthesia in below knee surgeries. Ultrasound usage has helped in enhanced block quality with lesser complications compared to other techniques of nerve localization which are non-ultrasound based, where needle placement is guided by anatomical landmarks[4].

Long-acting local anaesthetics like Ropivacaine have shown to extend action of nerve blocks, thus reducing the need for postoperative opioids. α2 receptor agonists improved block efficacy further when added to local anaesthetics. Studies indicate that the duration of both sensory and motor blockade in peripheral nerve block is prolonged with clonidine.[26]

Dexmedetomidine is a newly introduced selective alpha 2 receptor agonist with action similar to clonidine.

Many studies are published on Dexmedetomidine in TAP block, brachial plexus block, femoral nerve block, etc. but the popliteal sciatic nerve block is still unexplored compared to the rest.

Hence this research was done to determine the effect of Dexmedetomidine with Ropivacaine in ultrasound assisted popliteal sciatic nerve block. we also studied

the effect on intraoperative hemodynamic parameters and postoperative analgesic requirement.

Abdullatif et al found that 50 mcg and 75 mcg of Dexmedetomidine when added to 25 mL of bupivacaine hastens the onset and prolongs the duration of the nerve block. He also found that 25 mcg does not produce any significant alteration in the characteristics of the peripheral nerve block[10].Hence we decided to add 50 mcg of Dexmedetomidine to the local anaesthetic (15 mL of Ropivacaine) in the study group in our study.

The sixty individuals in ASA grade I-III who participated in our study ranged in age from 18years to 70years, were of either gender and were randomized into two groups. Statistically the age and sex of the groups were found not significant thus eliminating selection bias.

Neither patient nor the observer who followed up the patient knew which drug the patient has received during the study eliminating observer bias.

The present study results indicate that the Dexmedetomidine group experienced a significantly faster onset of sensory block, averaging 9.23 ± 1.14 minutes, compared to 10.6 ± 1.89 minutes in the control group. The duration of the sensory block was significantly longer in the Dexmedetomidine group, averaging 15.18 ± 2.16 hours, compared to 11.65 ± 1.96 hours in the other.

Similarly, with Dexmedetomidine it showed a quicker motor block onset at 11.67 ± 1.4 minutes versus 13.07 ± 2.02 mins in Control group, and the motor block duration got significantly extended, averaging 14.3 ± 2.2 hours compared to 10.52 ± 1.99 hours in the Control group. These findings demonstrate that Dexmedetomidine

significantly enhances both the duration and onset of sensory and motor blocks, resulting in longer-lasting, faster anaesthesia on comparison with the Control group.

In another recent study by Pallavi et al, one group of patients received 19.5 millilitres of 0.75% Ropivacaine and 0.5 millilitres of saline for sciatic nerve block via popliteal approach, while the other group received 19.5 millilitres of 0.75% Ropivacaine plus 0.5 millilitres (50 mg) of Dexmedetomidine. The study showed that the average time until sensory block started in the Dexmedetomidine group was 5.78±2.7minutes, while in the other group it was 8.64±2.4 minutes. Onset of motor block was 8.78±1.7 minutes in dexmedetomidine group and 10.42±3.6 minutes in the other group. Duration of sensory and motor block was also seen to be prolonged with dexmedetomidine. Our study's results were comparable to the results of the above study. [27]

The mechanism by which dexmedetomidine as an adjuvant to local anaesthetics enhances their effect is multifactorial. At peripheral level, it activates α_2 adrenoceptors in peripheral vessels, constricting blood vessels around injection site, delaying absorption of local anaesthetics, and prolonging block time. At spinal cord level, its said to reduce the release and reuptake of excitatory neurotransmitters, such as glutamate and substance P, by binding to α_2 receptors in the spinal dorsal horn after systemic absorption. Hyperpolarized interneurons inhibit the ascending spinal pathway related to nociceptive sensation, producing analgesia. Similarly it acts on α_{2A} and α_{2C} adrenergic receptors in the brainstem to achieve analgesic effect from the central level by inhibiting the descending noradrenergic pathway in the medulla or reducing sympathetic nerve signals.[28,29]

Earlier studies have suggested that addition of Dexmedetomidine in peripheral nerve block caused decreased usage of analgesics after surgery.

QiangWang et al studied Erector spinae plane block in patients going through thoracotomy for oesophageal cancer using Dexmedetomidine and ropivacaine. This showed that in Dexmedetomidine group, the duration of analgesia (505.1 ± 113.9min) was longer than the normal saline group (323.2 ± 75.4min) ($P < 0.001$) thus reduced opioid consumption after open thoracotomy.[15]

Our study results indicate time till use of first analgesic is increased with Dexmedetomidine, averaging 15.93 ± 2.33 hours on comparison with 13.33 ± 2.4 hours in the Control group. Furthermore, in terms of total analgesic consumption, most patients in the Dexmedetomidine group required only 1 gram of inj. Paracetamol (70%) whereas majority in the control group required 2 grams of paracetamol injection (80%) within the first 24 hours. Additionally, 6.67% patients of Dexmedetomidine group required no analgesic at all, whereas all patients of the Control group needed analgesics. The findings of our study were thus similar to that of Qiang Wang et al.

There were few studies in which correlation of hemodynamic changes like heart rate and blood pressure due to addition of Dexmedetomidine was analysed. Results of our study indicate that while initial average heart rates were similar among Dexmedetomidine group and the Control group, significant changes emerged over time. Starting from 30 minutes onward, the Dexmedetomidine group consistently exhibited lower average heart rates compared to the Control group, with p-values indicating statistical significance at multiple time points (30, 40, 50, 60, and 90 minutes). These findings suggest that Dexmedetomidine has an impact on reducing

heart rate over time in comparison with the Control group. But at any point of time in the study, the fall in heart rate was not clinically significant to warrant any intervention.

Dexmedetomidine group consistently showed lower SBP, DBP and MAP than in the Control group throughout the time points measured (10, 20, 30, 40, 50, 60, and 90 minutes), but the differences were not statistically significant at any single time point. These findings suggest that although Dexmedetomidine influences SBP, DBP and MAP over time, it was not significant statistically or clinically significant to warrant any intervention. Within the group HR, SBP, DBP, MAP showed a downward trend possibly due to better patient comfort following pain relief after the block. The fall too did not call for any intervention. No notable Sedation occurred with the study drug at any time during the study period.

Abdulatif et al. studied the effect on pharmacodynamics of femoral nerve block with varying amounts of Perineural Dexmedetomidine using 25 mcg,50 mcg and 75mcg in 25 ml 0.5% Bupivacaine and observed that there was a quicker onset time, more duration of block and longer period until first postoperative request for emergency analgesics compared to the control group with normal saline when 50 mcg and 75 mcg of Dexmedetomidine was added. Our results are consistent with the results of the above study.[10]

Our data is in agreement with previous studies showing the enhanced nerve blockade in means of faster onset, prolonged duration and decreased amount of post op rescue analgesics after Dexmedetomidine is added to ropivacaine for nerve blocks.

One disadvantage seen is that addition of dexmedetomidine also prolongs the motor block, which though beneficial during prolonged surgeries can contribute to patient anxiety in the post-operative period especially when the surgery is of short duration.

LIMITATIONS

- Our study was conducted in 60 patients. This relatively small sample size is a limitation and further multicentric studies with a larger sample size may be done to get confirmative results.
- In our study, we evaluated the effect of a fixed dose of dexmedetomidine added as an adjuvant to ropivacaine, rather than studying different doses of dexmedetomidine.

FUTURE SCOPES

- Study with larger sample size to be conducted to get confirmative results.
- Evaluation of a smaller dose which may produce good postop analgesia without much prolongation of motor block.
- Clinical trials comparing the effect of Dexmedetomidine as an adjuvant to two different local anesthetics can also be undertaken.
- Clinical trials using different doses of Dexmedetomidine needs to be done for achieving optimal balance between block duration, haemodynamic side-effects and sedation in clinical practice

CONCLUSION

- Dexmedetomidine as adjuvant to Ropivacaine lead to an earlier onset and prolonged duration of sensory and motor block in ultrasound guided Popliteal Sciatic nerve block for below knee surgery.
- When compared to control group, Dexmedetomidine group has a mild impact on reducing heart rate without any significant effect on the blood pressure
- It prolonged the time to the first postoperative request for rescue analgesia and also reduced the total requirement of analgesics in the first 24 hours in the postoperative period.

SUMMARY

The study titled "EVALUATION OF THE EFFECT OF DEXMEDETOMIDINE AS ADJUVANT TO ROPIVACAINE IN ULTRASOUND GUIDED POPLITEAL SCIATIC NERVE BLOCK IN BELOW KNEE SURGERY– A ONE YEAR HOSPITAL BASED DOUBLE BLINDED RANDOMIZED CLINICAL TRIAL". was conducted.

60 individuals belonging to ASA I -III who met our inclusion and exclusion criteria, undergoing below knee surgeries under ultrasound guided popliteal sciatic nerve block were enrolled into two randomized groups. Group A- received 15 mL of 0.75% Ropivacaine+0.5mL(50mcg) Dexmedetomidine and group B- received 15mL of 0.75% Ropivacaine +0.5mL Normal saline. Onset and duration of sensory and motor block, hemodynamic parameters were studied to evaluate the effect of Dexmedetomidine as adjuvant to Ropivacaine in popliteal block.

Onset and duration of sensory and motor blockade, time to first dose of rescue analgesia and Total analgesic requirement in first 24 hours was noted. Haemodynamic parameters were monitored and recorded at regular intervals of 10 minutes.

The observations of our present study showed that addition of Dexmedetomidine as adjuvant lead to an earlier onset and prolonged duration of sensory and motor block in ultrasound guided Popliteal Sciatic nerve block for below knee surgery. When compared to other group, Dexmedetomidine group also had effect on reducing heart rate. But the effect on BP and sedation score was not significant. It prolonged the time to the first postoperative request for rescue analgesia and also reduced the total requirement of postop analgesics in the first 24 hours.

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ANNEXURE I -INFORMED CONSENT FORM

KAHERs JNMC BELAGAVI

INFORMED CONSENT FORM

"EVALUATION OF THE EFFECT OF DEXMEDETOMIDINE AS ADJUVANT TO ROPIVACAINE IN ULTRASOUND GUIDED POPLITEAL SCIATIC NERVE BLOCK IN BELOW KNEE SURGERY-A ONE YEAR HOSPITAL BASED DOUBLE BLIND RANDOMIZED CLINICAL TRIAL"

Name of Student/Principal Investigator:

Name of Guide:

Objectives: To evaluate the effect of Dexmedetomidine as adjuvant to Ropivacaine in ultrasound guided Popliteal Sciatic nerve block in below knee surgery.

-To determine the effect on intraoperative hemodynamic parameters.

-To determine the time to 1st dose of analgesics in the postoperative period.

-To determine the total analgesic requirement in 1st 24 hours post surgery

Introduction: Mr./Mrs._____ we are requesting you to enroll yourself in study titled **"EVALUATION OF THE EFFECT OF DEXMEDETOMIDINE AS ADJUVANT TO ROPIVACAINE IN ULTRASOUND GUIDED POPLITEAL SCIATIC NERVE BLOCK IN BELOW KNEE SURGERY– A ONE YEAR HOSPITAL BASED DOUBLE BLINDED RANDOMIZED CLINICAL TRIAL".** conducted by---------, Post Graduate in M.D. Anaesthesiology under the guidance of --------Professor, Department of Anaesthesiology, J.N. Medical College, Belagavi under KLE University, Belagavi.

Respected Sir/Madam, We request you to participate in our study as you are eligible for the proposed study. During the study you will be asked some questions regarding the present complaints that you are having.

Your participation in this research is voluntary. Your decision whether or not to participate in the study will not affect your relationship with J.N.Medical College. If you decide to participate you are free to withdraw at any time.

Explanation of procedure: If you agree to enroll in my study, I will ask you the present and past medical history and family history. Then you will be clinically examined in detail. On the day of surgery, Popliteal Sciatic nerve block will be done by a senior anaesthesiologist with the administration of the study drugs in the intraoperative period and the hemodynamic parameters will be measured.

Withdrawal from participation in the study: Participation in this study is 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/will not have nor get any benefits by participating in this study. The data gathered will help the 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 from identifying 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.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes 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:"Name,ph.no,mail" 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 waving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "EVALUATION OF THE EFFECT OF DEXMEDETOMIDINE AS ADJUVANT TO ROPIVACAINE IN ULTRASOUND GUIDED POPLITEAL SCIATIC NERVE BLOCK IN BELOW KNEE SURGERY-A ONE YEAR HOSPITAL BASED DOUBLE BLIND RANDOMIZED CLINICAL 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:

will not receive any payment for participating in this study.

ANNEXURE - II - PROFORMA

Date:

PROFORMA

Title: "EVALUATION OF THE EFFECT OF DEXMEDETOMIDINE AS ADJUVANT TO ROPIVACAINE IN ULTRASOUND GUIDED POPLITEAL SCIATIC NERVE BLOCK IN BELOW KNEE SURGERY– A ONE YEAR HOSPITAL BASED DOUBLE BLINDED RANDOMIZED CLINICAL TRIAL".

Patient no:

Patient's Name : I.P No. :

Age : Date of Examination :

Gender : Anaesthesiologist :

Address :

Pre-anesthetic evaluation:

Chief complaints:

HOPI:

Past History:

H/o co-morbidities and drug intake :

H/o previous surgery/(s) where difficult airway was encountered :

Previous anaesthetic experience :

Family History:

General physical examination:

Height (cm) : Weight (Kg): BMI :

Pallor :: Icterus :

Cyanosis : Clubbing :

PR : BP: RR : SpO2 :

Systemic examination:

RS: Breath sounds: **CVS:** Heart sounds:

CNS: **GIT:**

Airway Assessment:

Teeth:

Jaw movements:

Local examination:

Investigations:

Hb(gm/dl): TLC: Platelet count:

Serum Creatinine**:** FBS: PT/INR:

Chest x-ray: ECG:

Preoperative physical status: ASA Grade I II III

Diagnosis:

Proposed surgery:

PATIENT NO:

NAME OF THE PATIENT:

Post Popliteal sciatic nerve block HR (bpm),SBP, DBP and MAP (mm of Hg) variation from time of drug administration till 90 mins.

TIME	**HR**	**SBP**	**DBP**	**MAP**
BASELINE				
10 MIN				
20 MINS				
30MINS				
40 MINS				
50 MINS				
60 MINS				
70 MINS				
80 MINS				
90 MINS				
↓				
120 MINS				
150 MINS				
180 MINS				
At the end of surgery				

Sensory Block:

a)	**Onset (min)**	
b)	**Duration (min)**	

Motor Block:

a)	**Onset (min)** **Grade 3 motor blockade**	
b)	**Total duration of Motor blockade(min)**	

Time to 1st dose of analgesic in postoperative period:	
Total analgesic requirement in 1st 24 hours post surgery:	

Investigator's name:---------- Signature:----------

Date:

Place:

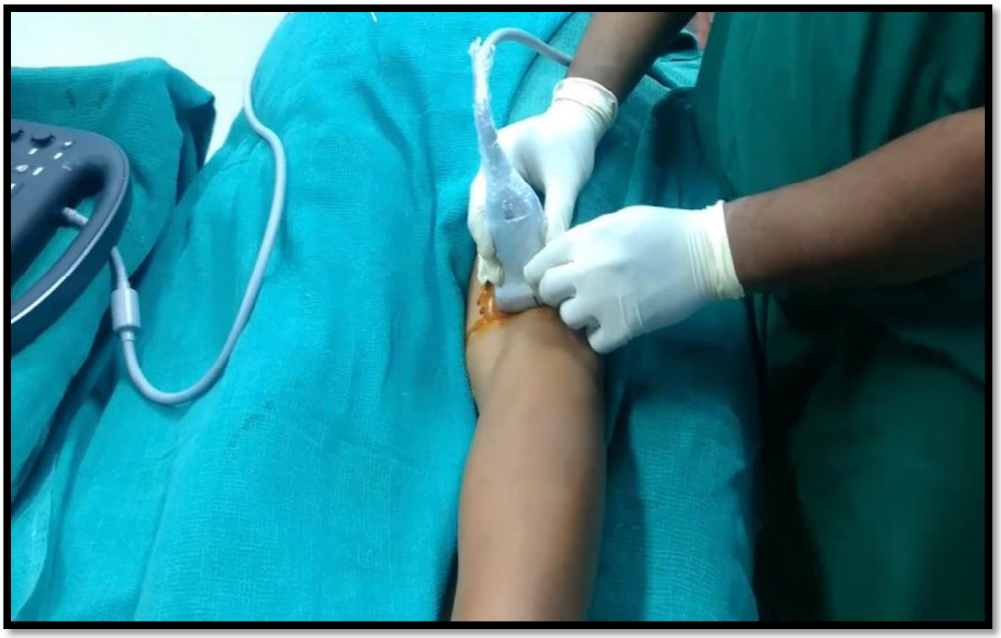
ANNEXURE - III – PHOTOGRAPHS

PHOTOGRAPH 1:Dexmedetomidine vial

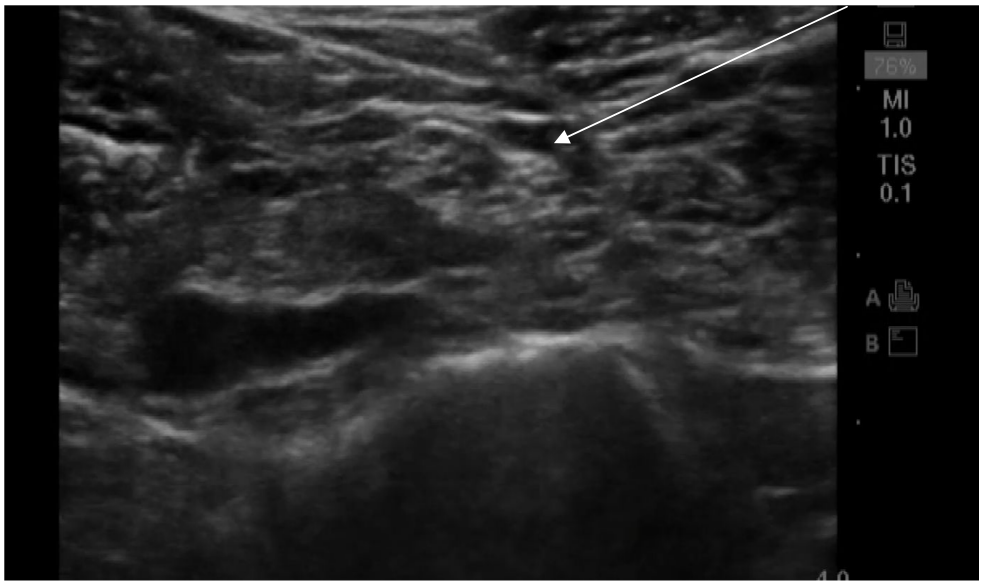
PHOTOGRAPH 2: Ropivacaine vial

PHOTOGRAPH 3: USG machine with probe

PHOTOGRAPH 4: Linear ultrasound probe



PHOTOGRAPH 5: Procedure



PHOTOGRAPH 6: Popliteal sciatic nerve

ANNEXURE IV: MASTER CHART

Group A (Dexmed)

Sl.No	Patient name	Age (years)	Sex	ASA	Heart rate (bpm)							BP (mmHg)							Sensory block onset (min)	Sensory block duration (hours)	Motor block onset (min)	Motor block duration (hours)	Time to 1st rescue Analgesic (hours)	Total Analgesic (Paracetamol) in 1st 24 hours (gram)
					0min	10min	20min	30min	40min	50min	60min	end of sx / 90min	0min	10min	20min	30min	40min	50min	60min	end of sx / 90min					
1	Parashuram	60	M	2	88	75	70	74	70	68	70	70	135/98	133/89	132/95	129/87	126/88	128/86	126/86	126/88	9	14	12	13	15	1g
2	Krishna naik	71	M	3	97	94	90	92	88	80	78	76	122/87	122/85	122/84	119/83	124/78	122/78	120/80	120/80	10	18	12	17	20	1g
3	Vijaya unni	55	F	2	102	100	98	95	90	84	86	84	123/72	129/77	128/75	122/82	126/84	124/84	126/88	122/82	8	10	10	9	11	2g
4	Rajaram	75	M	3	78	76	74	76	72	68	70	72	102/74	101/66	98/65	102/64	102/74	102/76	103/68	102/74	8	14	12	13.5	15	1g
5	Chandrakant	41	M	2	92	89	84	82	84	82	80	84	124/72	120/72	126/83	121/81	120/81	123/80	124/82	120/81	10	14	11	13	14	1g
6	Nazirsab rajasab	72	M	3	84	80	76	68	72	70	74	72	170/100	168/85	158/82	151/94	147/95	146/92	148/96	147/95	9	16	11	15.5	16	1g
7	Sadeppa ravalappa	65	M	3	88	76	68	64	65	68	66	64	122/87	115/83	113/82	119/85	120/84	119/84	118/86	119/85	10	15	12	14.5	16	1g
8	Limtavva	60	F	1	82	76	74	70	74	72	68	70	151/94	147/95	146/102	147/95	146/92	148/96	146/96	147/95	10	13	12	12	12	1g
9	Noorjaan	45	F	3	90	92	88	86	84	82	84	84	139/98	136/86	133/86	129/87	133/89	126/88	125/84	126/88	11	19	14	18	20	nil
10	Ishwar	40	M	3	90	88	75	70	72	68	70	72	135/98	138/95	138/91	136/86	133/86	134/82	135/85	133/86	10	14.5	13	14	15	1g
11	Yallappa	63	M	2	88	75	70	74	70	74	72	70	135/98	133/89	132/95	129/87	126/88	130/90	128/88	126/88	11	14	12	13	15	1g
12	Nilavva	52	F	2	76	74	70	69	72	74	76	78	140/90	142/87	140/88	138/86	136/88	138/88	136/82	134/84	9	15	11	14	15	2g
13	Parappa	72	M	3	60	58	58	58	62	60	62	64	142/80	140/76	142/78	136/76	138/74	136/74	134/74	130/76	8	16	12	15.5	15	2g
14	narayan Devagekar	60	M	1	76	74	70	69	72	74	76	78	144/93	142/78	140/80	136/78	136/74	134/74	130/80	132/70	10	13	14	12	14	2g
15	Raosaheb	55	M	3	100	94	88	80	76	78	80	78	160/100	164/102	156/100	148/96	150/100	148/98	146/88	144/78	8	15	10	14	16	1g
16	Ramanna	65	M	2	80	79	76	77	74	76	82	80	140/80	136/76	134/76	130/80	132/78	130/80	128/82	126/78	9	17	12	16	18	1g
17	ishwarappa pattan	68	M	2	90	92	88	86	84	83	84	84	139/98	136/86	133/86	129/87	133/89	126/88	125/86	126/88	7	16	10	15	16	1g
18	Bhimagouda	55	M	1	88	76	74	82	76	75	74	72	132/70	135/98	132/70	126/88	128/86	127/88	128/88	126/88	12	12	15	11	12	1g
19	Shankarappa	66	M	3	90	88	75	76	81	78	73	72	146/102	140/98	138/95	137/94	133/86	136/84	134/86	133/86	10	17	14	16	18	1g
20	Nagavva	62	M	2	88	75	70	74	70	74	72	70	135/98	133/89	132/95	129/87	126/88	130/90	128/88	126/88	8	17	10	16.5	18	1g
21	badshah nanusab	74	M	3	77	76	68	66	66	68	62	68	130/70	140/80	130/70	140/80	130/70	150/70	140/80	150/70	10	16.5	12	15	18	1g
22	Gangaram	64	M	2	84	80	76	68	64	68	63	64	170/100	168/85	158/82	151/94	147/95	146/94	148/94	147/95	9	15	11	14	16	1g
23	Umapati	72	F	3	100	94	88	80	76	78	80	78	160/100	164/102	156/100	148/96	150/100	148/98	146/88	144/78	8	12	10	11	13	2g
24	Nagulirama	55	F	3	90	88	75	70	72	71	70	72	135/98	138/95	138/91	136/86	133/86	132/84	134/86	133/86	10	19.5	13	19	20	nil
25	Mohan narayan	60	M	1	118	106	102	100	102	98	96	98	164/102	150/81	149/81	147/95	149/81	148/80	150/84	149/81	9	18	10	17	18	1g
26	Shivanand	54	M	2	90	88	75	76	81	78	73	72	146/102	140/98	138/95	137/94	133/86	136/84	134/86	133/86	9	13	12	12.5	15	2g
27	Sangappa	48	M	1	88	75	70	74	70	74	72	70	135/98	133/89	132/95	129/87	126/88	130/90	128/88	126/88	8	15	10	14	16	1g
28	Kallappa	83	M	3	92	88	84	86	82	78	80	82	139/98	136/86	133/86	137/94	132/84	133/82	130/88	132/84	10	17	12	16	18	1g
29	Mallayya	69	M	2	90	88	75	76	81	78	73	72	146/102	140/98	138/95	137/94	133/86	136/84	134/86	133/86	8	14	10	13	15	2g
30	Kamalavva	70	F	3	68	63	59	54	50	54	56	56	132/70	135/98	138/88	135/98	126/88	130/90	125/90	126/88	9	16	11	15	18	1g

Group B (Control)																								
Sl.No	Patient name	Age (years)	Sex	ASA	Heart rate (bpm)								BP (mmHg)								Sensory block onset (min)	Sensory block duration (hours)	Motor block onset (min)	Motor block duration (hours)	Time to 1st rescue Analgesic (hours)	Total Analgesic (Paracetamol) in 1st 24 hours (gram)
					0min	10min	20min	30min	40min	50min	60min	end of sx / 90min	0min	10min	20min	30min	40min	50min	60min	end of sx / 90min						
1	Shainaz azadmulla	55	F	3	100	96	98	100	96	98	100	100	170/110	160/110	160/96	157/88	155/86	158/90	154/82	156/88	12	12	14	10	14	2g
2	Ashwin kumar	39	M	2	92	88	96	92	90	88	90	92	143/85	136/81	134/76	136/76	138/78	135/74	134/72	136/76	11	12	12	8	13	2g
3	Roopa shivaji	68	F	3	103	105	94	89	90	92	90	89	160/86	155/85	148/78	143/81	146/81	145/79	144/80	143/81	10	11	12	10	14	2g
4	Vilas Naik	46	M	2	97	98	93	90	93	98	94	98	128/82	125/79	128/76	127/72	120/72	119/72	120/76	117/69	10	14	11	13	17	1g
5	Nazirsab rajasab	72	M	3	68	63	69	64	68	64	62	64	132/70	135/98	138/88	135/98	126/88	126/84	128/84	126/88	10	13	15	12.5	16	1g
6	Suvarna Anil	50	M	3	90	94	92	95	94	90	92	92	133/90	128/89	129/83	127/86	134/88	117/82	123/82	123/82	7	12.5	10	11	14	2g
7	Shainaz	55	F	3	120	118	116	113	115	119	110	100	175/81	157/76	167/84	172/80	177/82	173/81	174/88	173/81	12	14	15	12.5	18	1g
8	Laxmi laxman	67	F	2	67	70	69	62	60	64	62	64	154/82	140/78	146/98	146/90	142/86	140/76	138/68	138/68	15	15	16	14	16	2g
9	Vilas powar	32	M	2	97	98	93	90	93	98	96	98	128/82	125/79	118/76	107/72	110/72	107/69	108/70	107/69	10	14	11	13	18	1g
10	Iman hussain	75	M	3	94	95	90	86	83	81	82	81	146/66	152/81	143/65	130/61	124/56	126/61	124/68	122/61	13	13	15	12.5	16	1g
11	Anita gauwda	52	M	2	88	90	86	80	84	88	83	84	152/81	146/66	136/102	134/88	130/76	134/81	132/76	130/76	10	11	11	10	12	2g
12	Kallappa	83	M	3	92	88	84	86	82	84	80	82	139/98	136/86	133/86	137/94	132/84	133/82	134/86	132/84	10	13	12	12	16	1g
13	Shivanappa	72	M	3	67	65	66	63	61	64	62	61	175/92	168/85	162/83	158/82	158/80	157/84	156/82	158/80	8	10	12	8	13	2g
14	Shankarappa	66	M	3	90	88	75	76	81	84	82	81	146/102	140/98	138/95	137/94	133/86	135/84	134/82	133/86	10	8	14	7.5	10	2g
15	Mohan narayan	60	M	1	118	106	102	100	102	100	98	102	164/102	150/81	149/81	147/95	149/81	146/82	148/88	149/81	9	14	10	13	15	2g
16	Chandrakant	41	M	2	92	89	84	82	84	82	80	84	124/72	120/72	126/83	121/81	120/81	124/82	122/78	120/81	9	12	11	11	13	2g
17	Jyotiba	69	F	2	92	90	88	90	84	80	83	84	151/94	147/95	146/90	145/88	146/90	145/84	146/88	146/90	10	11	13	10.5	13	2g
18	Kasturi	59	F	2	90	88	75	76	81	78	80	81	146/102	140/98	138/95	137/94	133/86	134/82	133/82	133/86	10	8	14	7.5	10	2g
19	Mallappa	73	M	3	92	88	84	86	82	80	84	82	139/98	136/86	133/86	137/94	132/84	135/86	134/88	132/84	10	11	12	9	12	2g
20	Shantavva	60	M	2	87	85	86	85	82	84	80	82	123/72	116/71	111/70	110/70	111/71	112/72	113/72	111/71	11	12	14	10	12	2g
21	Laxmavva	67	F	2	67	70	69	62	60	64	65	64	154/82	140/78	140/76	136/78	132/68	130/76	138/68	138/68	15	10	17	9	10	2g
22	Babu powar	60	M	2	98	94	88	92	86	88	84	86	149/81	148/80	144/78	142/76	134/75	135/74	136/78	134/75	10	11	15	10.5	12	2g
23	Satteppa	65	M	3	68	65	63	61	66	68	64	66	175/94	174/90	172/88	162/83	158/82	160/88	161/92	158/82	12	16	15	15	16	2g
24	Shakuntala	48	F	1	76	75	73	71	68	65	66	65	123/72	120/68	122/82	121/81	118/80	120/68	122/76	120/68	8	10	10	9	11	2g
25	Gangaram	60	M	1	76	78	70	72	74	72	72	72	147/95	149/81	134/75	132/74	135/83	140/78	136/79	136/79	11	9	13	8	10	2g
26	Kavita ashok	48	F	1	84	82	84	86	84	80	84	86	139/98	133/86	136/86	132/84	132/82	134/82	131/80	132/84	13	10	15	9.5	12	2g
27	Suvarna	62	F	2	94	90	84	82	80	76	80	82	122/87	119/85	115/83	113/82	115/82	116/78	114/78	113/82	8	10	10	9.5	11	2g
28	Parappa	70	M	2	64	63	59	64	66	62	64	64	158/82	151/94	147/95	151/94	154/92	150/88	149/96	151/94	12	11	13	10	12	2g
29	Bhimagouda	55	M	1	88	76	74	82	80	79	78	74	132/70	135/98	132/70	126/88	130/82	128/84	130/86	126/88	12	12	15	11	12	2g
30	Ishwar	40	M	3	90	88	86	83	86	82	78	80	135/98	138/95	138/91	136/86	133/86	134/82	136/86	133/86	10	10	15	9	12	2g