*Nanotechnology Perceptions*
*ISSN 1660-6795*
*www.nano-ntp.com*

# A Comparative Study between Ultrasound Guided Four in One Block versus Femoral Nerve Block with Dexmedetomidine Additive in Enhanced Recovery After Knee and Below Knee Surgeries

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Orthopaedic operations are increasingly incorporating improved recovery times following knee surgeries. The optimal postoperative objective is motor preservation with sufficient analgesia, which facilitates earlier physical treatment, an early hospital discharge and a faster recovery. Compared to epidural or IV case-controlled analgesia, femoral nerve block (FNB) is recognized for its better control of pain, reduced hospitalization, and accelerated functional recovery, all without any correlated negative consequences. Nevertheless, it decreases strength of quadriceps muscle and elevate probability of falling. It was reported that patients who undergo total knee arthroplasty (TKA) and receive FNB frequently encounter postoperative posterior knee pain requiring supplemental opioid drugs. FNB or adductor canal block alone aren't able of providing complete analgesia throughout whole knee following knee operations, as knee is innervated by two sacral plexus (sciatic nerve) and lumbar plexus (femoral and obturator nerves). Consequently, cases who had FNB or ACB commonly experienced postoperative posterior knee pain that necessitated use of supplemental analgesic drugs. Dexmedetomidine was demonsterated to reduce anesthesia onset, extend duration of motor and sensory nerve blocks, and offer an adequate sedative impact in peripheral nerve blocks, according to numerous investigations.

## 1. Introduction

In comparison to general anesthesia, spinal anesthesia has more favourable outcomes for knee arthroplasty. This is attributable to beneficial physiological effects of sympathetic blockade, which include improved initial pain relief, elevated leg blood flow, and reduced blood loss. All of these factors contribute to a decrease in cardiopulmonary and thromboembolic

morbidity. However, potential cost is a diminished ability to mobilize early following surgery because of prevalence of pain following resolution of spinal anesthesia (1).

An ideal nerve block that targets sensory nerves and spares motor function can help early rehabilitation and ambulation, which is a major objective for cases having knee surgeries (2).

Roy et al., 2018 (3) defined a new and single-administration method for combined 4-in-1 block (obturator nerve, saphenous nerve, sciatic nerve and nerve to vastus medialis) that provides full postoperative analgesia through a single injection point. They suggested that combination of a ACB, sciatic nerve block and peripheral nerve block presented positional and technical challenges that may be resolved with ease and certainty by utilizing a single administration 4-in-1 block method. Nevertheless, the 4-in-one methods have not been revealed in any other investigations. Consequently, its desirable to conduct more extensive research to confirm results.

There are many studies that show efficacy of FNB techniques concerning postoperative pain control. However, no enough stablished data are present to explain the 4-in-1 techniques regarding postoperative pain management.

So, in this study, we aimed to test whether the simple (with less local anaesthetic dose) femoral nerve block while being augmented by addition of dexmedetomidine can replace the more complicated (with more local anaesthetic dose) four in one block in enhanced recovery after knee and below knee surgeries.

Anatomy of the femoral and sciatic nerves

Anatomy of femoral nerve

Origin

The femoral nerve, is the largest nerve within lumbar plexus, originates from the front branches of the $2^{nd}$, $3^{rd}$, & $4^{th}$ lumbar nerves (L2, L3, & L4) (figure no.1). This nerve plays a vital role in both movement & sensory functions in lower extremities. Sensory functions: Provides cutaneous branches to anteromedial thigh's (anterior cutaneous branches of femoral nerve) & the medial side of foot & leg (saphenous nerve). Motor functions: Innervates the anterior thigh muscles that flex hip joint (sartorius, iliacus, pectineus) & extend knee (quadriceps femoris: vastus medialis, vastus lateralis, vastus intermedius, & rectus femoris). (4).

Anatomical Course

Femoral nerve travels inferiorly through psoas major muscle of posterior abdominal wall following originating from lumbar plexus. It provides implications to pectineus and iliacus muscles prior to entering thigh. Femoral nerve then reaches femoral triangle by passing below inguinal ligament. Femoral nerve is situated lateral to femoral vessels in this triangle. In contrast to nerve, femoral vein and artery are enclosed in femoral sheath (5).

Anatomy of the femoral triangle

Femoral triangle is a clinically important region located in superomedial aspect of anterior thigh.

Borders:

Femoral triangle composed of 3 borders, a floor and a roof.

The boundaries of femoral triangle provide important anatomical landmarks. The roof is made of fascia lata superficial fascia, and skin. The adductor canal is the direction in which apex of the triangle is directed downward. floor is formed by iliopsoas, adductor longus and pectineus, muscles. Superior boundary is formed by inguinal ligament, a strong fibrous band extending from anterior superior iliac spine to the pubic tubercle. The sartorius muscle's medial border constitutes lateral boundary, whereas the adductor longus muscle's medial border establishes medial boundary. Inguinal ligament functions as a flexor retinaculum, providing support for contents of femoral triangle throughout hip flexion (6).

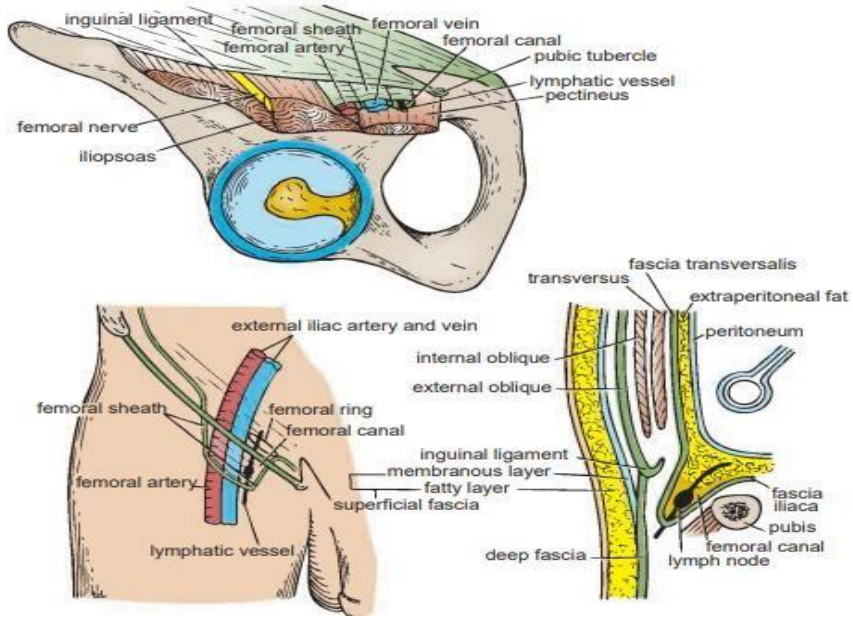


Figure (1): content of the femoral triangle (7).

Anatomy of the sciatic nerve

Origin

Sciatic nerve plays a critical role in the innervation of lower limb and is the largest nerve in human body. It is formed by the combination of nerve fibers from the ventral rami of the fourth and fifth lumbar (L4-L5) spinal nerves and the 1st three sacral (S1-S3) spinal nerves (5).

Course

The lumbosacral plexus is source of sciatic nerve. It goes away pelvic region & enters gluteal region by passing through greater sciatic foramen after its formation. It descends in an inferolateral direction & originates inferiorly to piriformis muscle. As nerve travers's gluteal region, it crosses over obturator internus, posterior surface of superior gemellus, quadratus femoris, & inferior gemellus muscles. It proceeds by going beneath long head of biceps

femoris muscle to enter posterior of thigh. Within posterior thigh, nerve generates branches that supply hamstring muscles & adductor magnus. Upon reaching apex of popliteal fossa, sciatic nerve terminates its course by dividing into tibial & common fibular nerves (4).

Innervation

The sciatic nerve is a combined nerve that supplies muscles & skin of lower limb with a variety of motor and sensory branches. Furthermore, it supplies articular branches to innervate lower limb joints (8).

Motor innervation

The gluteal region isn't innervated by sciatic nerve, despite fact that it travels through it. Nevertheless, muscles of posterior thigh are supplied with direct motor activity by sciatic nerve through minor muscular branches. These muscles consist of ischial portion of adductor magnus, semimembranosus, semitendinosus, and biceps femoris (4).

Sensory innervation

Skin of lower leg and foot is additionally sensory innervated by sciatic nerve, with exception of medial leg, that's innervated by saphenous nerve. Lateral & medial plantar nerves, that are responsible for sole sensation, are further divided by tibial nerve. Superficial peroneal nerve and deep peroneal nerve are additional branches of common peroneal nerve. Sensory innervation of dorsum of foot and lateral limb is provided by superficial peroneal nerve. Sensation among the 1$^{st}$ and 2$^{nd}$ toes is responsibility of deep peroneal nerve. Calf and a small lateral part of foot are supplied with sensation by medial & lateral sural nerves, which are composed of collateral branches from tibial & common peroneal nerves (9).

Pharmacology of local anaesthesia

Local anaesthetics are medications utilised for producing reversible sensation loss in a specific area of body, primarily by blocking nerve conduction. They are commonly used in various medical procedures to provide analgesia and anaesthesia. Local anaesthetic substances hinder action potentials in responsive tissues by obstructing voltage-gated sodium channels. This action leads to the inhibition of action potentials in pain-sensing fibres, effectively impeding conveyance of pain signals (10).

Mechanism of action

Suppression of voltage-gated sodium channels is mechanism by which local anaesthetic agents block action potentials in neurons. Voltage-gated sodium channels are present in three potential conformations: inactive, closed, and open (figure no.8). If nerve cell membrane is at rest, channel is in a closed state. However, if depolarization happens, channel opens & subsequently enters an inactive state, thereby facilitating repolarization. Local anaesthetic is bound to ion channel, which Favors inactive states of sodium channel, thereby suppressing movement of action potentials along impacted nerve fibres. The most favorable conditions for local anaesthetic binding are provided by inactive and open phases of sodium channel if a local anaesthetic has been detected in cell. This suggests that the onset of local anesthetic impact is facilitated by stimulation of nerve fibers. Frequency-dependent blockade is name given to this condition (10).

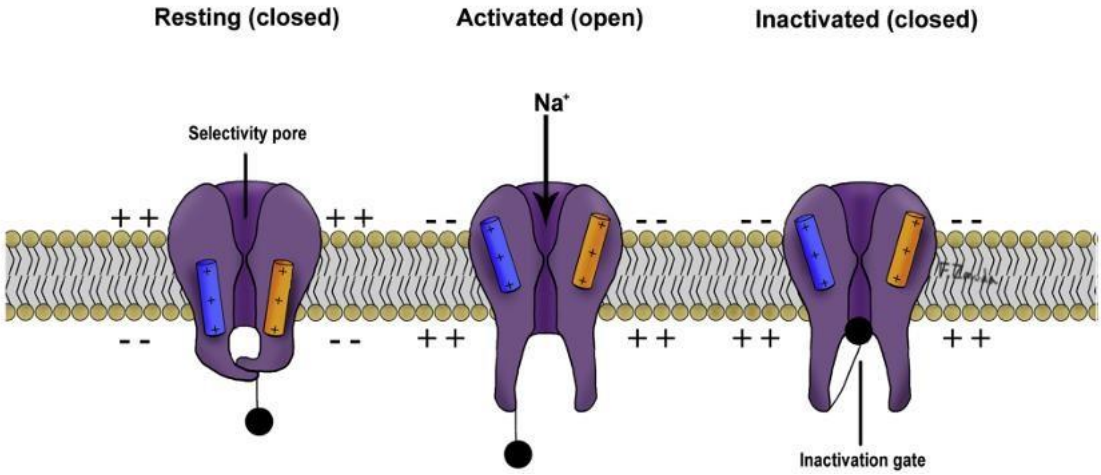


Figure (2): Configurations of the voltage gated sodium channel (11).

Physical and pharmacological properties of local anaesthetics

Local anesthetics are weak bases with a pKa (dissociation constant) of 7.5 to 8.5 and are classified as aminoesters or aminoamides according to their structure. An aromatic group has an association to a tertiary amine through an ester linkage (aminoester) or an amide linkage (aminoamide) in general structure of all agents (figure no. 9). There are 4 physicochemical characteristic that detect local anesthetic agents activity: pKa, molecular weight (MW), degree of protein binding and lipid solubility (10).

pKa

The pKa and pH of tissue into that amphipathic local anesthetics are administered identify their dissociation. pKa is pH where substance is in equilibrium with fifty percent in ionized form and fifty percent in non-ionized form, as defined. Ionized fraction in solution increases as pKa increases for bases, including local anaesthetics. Ratio of both states is defined by Hendersone Hasselbalch equation:

log [Ae]/[AH] ¼ pKa e pH

wherever [Ae] is the ionized form and [AH]is non-ionized form. The medication should diffuse into the target neuron cell body to produce its effects, as local anaesthetic binding site on the sodium channel is located in cell. Non-ionized form of medication is capable of readily passing the cell membrane. Upon entering cell, a new equilibrium becomes calculated, and ionized (active) form of medication may produce its effects. generally, agents with a low pKa will have a rapid onset of action because of a lesser degree of ionization at physiologic pH, while agents with an elevated pKa will have a delayed onset of action because of a advanced degree of ionization at physiologic pH (10).

Molecular weight

The smaller the molecular weight, the more quickly molecules diffuse through membranes. The MW is inversely correlated with local anesthetic's capacity to diffuse through tissue, with

a range of 220 to 288 Da. Alterations in MW are typically correlated with alterations in pKa and lipid solubility, and similarly they are triggered by varying substitutions on aromatic group and tertiary amine (12).

Lipid solubility

The agent's solubility is primarily determined by aromatic group structure. Potency and period of action of agent are significantly influenced by degree of solubility. The partition coefficient, that is described as ratio of levels if local anesthetics are dissolved in a mixture of aqueous and lipid solvents, is a measure of lipid solubility of local anesthetics. The speed of onset is influenced by increased solubility of lipids, which allows for more rapid diffusion through lipid membranes to get to their site of action. However, other factors are additionally significant. Furthermore, a higher lipid solubility results in a higher volume of distribution (10).

Binding of proteins

The free fraction of drug available that is accessible for binding to target receptors and produce an impact is detected by binding of protein. generally, local anesthetics that demonstrate an elevated level of protein binding to α1-acid glycoprotein have a prolonged period of action & lesser bioavailability. Protein binding is reduced and probability of toxicity is elevated in hypoxia, hypercarbia, and acidaemia. Kids who are less than six months old have a reduced capacity for binding proteins (12).

Vasoactivity

The potency and period of action of local anesthetics are influenced by their vasoactivity. In particular, lidocaine induces a more rapid absorption than bupivacaine. Ropivacaine and levobupivacaine show a bimodal vasoactive response. The two agents demonstrate vasodilation at clinical concentrations and vasoconstriction at subclinical doses. Vasoconstriction of tissues can be induced by adrenaline levels below 1:800,000. This is possible in presence of local anesthetics (13).

Injection routes

A variety of methods are utilized to administrate local anesthetics. They involve topical, such as for skin and airway, as well as perineural, subcutaneous, IV, intra-thecal and epidural (10).

Mixtures of local anesthetics

Eutectic refers to the process of combining 2 compounds to generate a substance which possesses a single set of physical properties. The eutectic mixture of local anesthetic (EMLA) is a crystalline base mixture consisting of 2.5 percent prilocaine and 2.5 percent lidocaine in an oil/water emulsion. The mixture's melting point is below that of individual local anesthetics, which enables utilization of a greater level of local anesthetic. For ensuring that onset and period of block are sufficient, anesthetists frequently combine local anesthetics. The physicochemical properties of these mixtures aren't sufficiently understood, in comparison with EMLA. Clinical investigation has shown that the expected impact of a decreased duration of block onset might not always be achieved by raising the percentage of lidocaine in a lidocaine-ropivacaine mixture. In contrast, the onset of blockade can't be significantly accelerated, and period of action could possibly be reduced. This is most likely the result of

the agents' level and pKa being altered as a result of their combination. Clinician preference remains 1$^{ry}$ factor in defining combination of local anesthetic agents, instead of consensus (14).

Toxicity

Local Toxicity

The occurrence of tissue ischemic necrosis can be followed by administration of local anesthetics. This reaction may arise due to toxic properties of solution, heightened pressure resulting from significant volumes, or constriction of blood vessels induced by vasopressors. Moreover, there exists a concern concerning potential neurotoxic effects of formulations containing elevated concentrations.

Increased concentration levels of local anesthetic solutions elevate the risk of direct neurotoxic impact on nerve trunks; essentially, all local anesthetics have potential to cause direct nerve impairment if their intraneural concentrations reach sufficiently high levels. Physicians must be cognizant that concentrations of local anesthetic solutions themselves possess inherent neurotoxic properties, underscoring the importance of their dilution, either within tissue or at site of administration, to ensure safe usage (15).

Systemic Toxicity

Unintentional introduction of local anesthetics into blood vessels during administration has long been acknowledged as the leading cause of systemic toxicity associated with local anesthetics (LAST). However, specific underlying health conditions can also heighten probability of an overdose of local anesthetics, consequently increasing the possibility of systemic toxicity. These conditions encompass impaired liver function, heart-related issues, pregnancy, and metabolic disorders. Furthermore, individuals at extremes of age face an elevated susceptibility to toxicity due to decreased elimination of anesthetics. Notably, infants below age of 4 months' exhibit diminished levels of α-acid glycoprotein in their plasma, leading to reduced intrinsic clearance of bupivacaine (16).

Management

The initial management approach for local anesthetic systemic toxicity centers around addressing airway maintenance, circulatory support, and minimizing systemic adverse consequences. It is essential to act immediately on oxygenation and ventilation in to prevent hypoxia and acidosis, which will enable diminish and resuscitation probability of cardiovascular collapse otherwise seizure advancement. In the event of seizures, it is recommended that benzodiazepines be administered immediately for avoiding acidosis and injury. In the absence of benzodiazepines, alternatives such as propofol or thiopental may be used, though caution is warranted due to their potential to exacerbate hypotension or cardiac depression. For uncontrollable tonic-clonic seizure activity, intermittent administration of small doses of succinylcholine is recommended to halt muscular movements and mitigate further acidosis (14).

Specific properties of Bupivacaine

Bupivacaine hydrochloride is 2-Piperidinecarboxamide, 1butyl-N- (2, 6-dimethylphenyl)-, monohydrate, a white crystalline powder, monohydrochloride, & is freely soluble in ninety-five percent ethanol, acetone and soluble in water. Bupivacaine is a potent local anesthetic that

is distinguished from the amide group of local anesthetics by its unique properties. Local anesthetics are utilized in epidural anesthesia, regional anesthesia, local infiltration & spinal anesthesia. generally, local anesthetics prevent generation of action potential in nerve cells by raising threshold for electrical excitation. It has following structural formula (17).

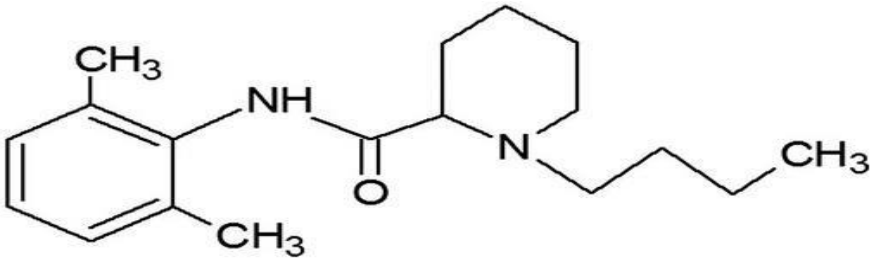


Figure (3): Bupivacaine structure (18).

Injection

Bupivacaine is obtainable in three variant levels: 0.25 percent, 0.5 percent, and 0.75 percent.

Local infiltration is used for peripheral nerve blocks, analgesia following surgery are used for dental or other minor operating producers, orthopedic procedures, spinal anesthesia is administered into cerebrospinal fluid for producing anesthesia for orthopedic operations, cesarean section, abdominal operation, or epidural anesthesia/analgesia is used for a caudal block and labor pain, is used to provide analgesia and anesthesia below umbilicus, typically for pediatric operation (19).

Negative Consequences

The dosage of bupivacaine is contingent upon operation, tissue vascularity, the area, segments blocked number, period or depth of anesthesia required, & case's physical disorder. Bupivacaine can react with ergot drugs that are utilized to treat blood thinners, migraine symptoms, monoamine oxidase inhibitors or antidepressants. Local anesthetics are uncommonly associated with immunologic reactions. It is extremely rare for preservative-free amide-type local anesthetics to cause allergic responses, and these events are typically not reported. On the other hand, responses to epinephrine-containing local anesthetics may be interpreted as reactions to allergens. Ester local anesthetics or preservatives are more probable for causing a true allergic reaction. In addition, cases can experience adverse reactions to preservatives, including methylparaben, that are commonly involved in local anesthetics.

Contraindications

The following are contraindications: infection at injection site, hypersensitivity to the medicine or any of its components, intra-articular continuous infusion, hypersensitivity to amide anesthetics, obstetric paracervical block, IV regional anesthesia, & obstetric anesthesia applying 0.75 percent level. Clinicians ought to utilize caution in cases who have hypersensitivity to sulfites, kidney impairment, hepatic impairment (liver clears amides), heart block, hypovolemia, hypotension, impaired heart function, and acutely ill, elderly, or debilitated cases (20).

Toxicity

The symptoms and signs of the majority of local anesthetics are comparable; nevertheless, the ratio of neurotoxicity to cardiotoxicity can alter, with bupivacaine being the most cardiotoxic. Frequency of toxicity is exceedingly infrequent, with a range of 1 to 1000 to 1 to 10,000. Take care in presence of abnormal cardiovascular or neurological symptoms that may indicate local anesthetic toxicity. The possibility of toxicity is additionally affected by local anesthetic injection site. Most frequent cause of bupivacaine toxicity is the unintended direct IV administration or rapid vascular uptake of medication, which has an upper limit of 2.5 to 3.5 milligram per kilogram. The toxicity of drugs might happen when upper limit of suggested dosage is administered, based on the technique and vascularity of the site of injection. Symptoms and signs of toxicity can appear quickly or delayed (21).

Treatment

The therapy of bupivacaine toxicity was a long-standing challenge because of its severe neurologic and cardiac toxicity. In past, therapy was favourable, consisting of management of airway, seizure regulator with quick-acting gamma-aminobutyric acid agonists like midazolam and standard cardiopulmonary resuscitation. The extended duration of bupivacaine's action makes it particularly hazardous in terms of toxicity. In centers in which cardiopulmonary bypass has been readily accessible, it has been utilized to provide support for toxic case till substance has been metabolized and removed to a satisfactory extent, that may require several hours. In the beginning of the 2000s, Guy Weinberg conducted important studies that demonstrated efficacy of lipid emulsions, including those used as carriers for total parenteral nutrition formulations, during recovering laboratory animals from bupivacaine toxicity. Profound outcome in animals (dogs and mice) resulted in numerous reports of cases in which lipid emulsion has been utilized as a last resort in human cases with profound cardiovascular collapse who had undergone nerve blocks with long-acting local anesthetics, including bupivacaine & ropivacaine. Therapy with lipid emulsion became recognized for its effectiveness over following fifteen years and has been adopted by the American Society of Regional Anesthesia as the standard for managing local anesthetics systemic toxicity. It has since been incorporated into their therapy algorithm. It has been previously utilized as a last resort therapy; however, it is now frequently utilized as the primary therapy for these cases. Lipid emulsions must be easily available in emergency situations at facilities that administer local anesthetics. It is remarkable that effectiveness of lipid emulsion for management of local anesthetics systemic toxicity has been found to be reduced in correlation with large doses of epinephrine. The significance of prompt therapy with lipid emulsion if local anesthetics systemic toxicity is suspected is further demonstrated by this proof. Detailed treatment algorithms are accessible on the website of the American Society of Regional Anesthesia (22).

Dexmedetomidine

Dexmedetomidine (DEX) is currently utilized in Intensive Care Unit for surgical sedation and anxiolysis. It was recently utilized in perioperative setting for premedication to reduce emergence delirium, to minimize stress response of anesthesia & operation, and to manage pain following surgery (23).

Physiological and Pharmacological Impacts of Dexmedetomidine

An imidazole component is dexmedetomidine. It is a right-handed isomer of medetomidine that possesses sedative and hypnotic characteristics. It is a highly selective membrane-bound G-protein coupling 2adrenergic receptor agonist. It has been accepted by United States Food and Drug Administration in 1999 for short-term sedation of mechanical ventilation cases in an adult critical care unit. It has been accepted for surgical sedation and has a variety of off-label indications for application in perioperative care. Additionally, European Medicines Agency awarded it approval for analgesia and sedation in 2017. The G-protein coupled receptor 2-adrenergic receptor has seven transmembranes. It is broadly dispersed throughout the autonomic ganglia, peripheral nervous system, and central nervous system. dexmedetomidine has the highest density in pontine nucleus, locus coeruleus, parahippocampal gyrus, pontine tegmental cingulate gyrus , and reticular nucleus (24).

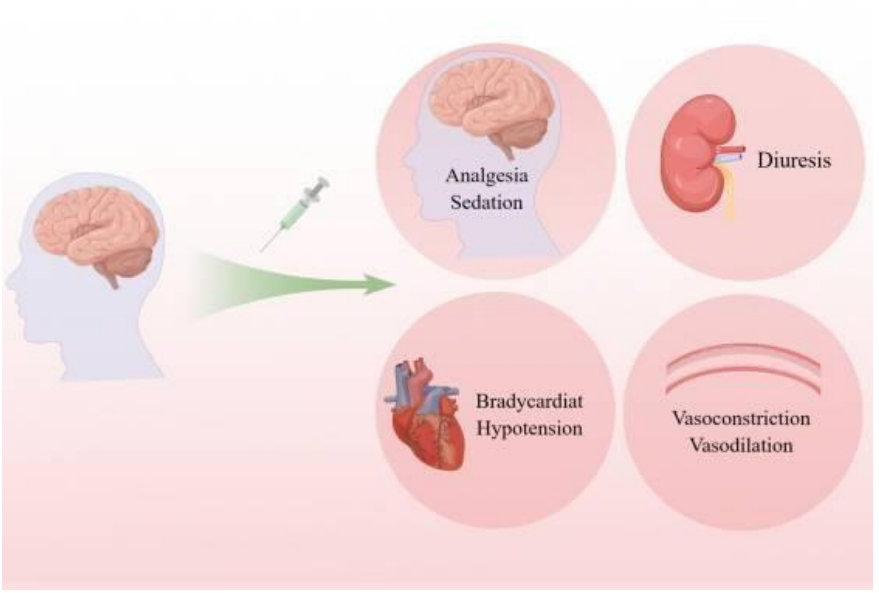


Figure (4): Dexmedetomidine impacts multiple locations (centrally and peripherally) (24).

Mechanism of DEX in Peripheral Nerve Block

The mechanism by which DEX acts as an adjuvant to local anesthetics, thereby improving their efficacy, has been the object of numerous theoretical explanations.

Peripheral Level

The inhibiting impact of dexmedetomidine as an adjuvant is widely recognized to be enhanced by peripheral action (24).

Brummett et al., 2011 (25) comparing DEX combined with ropivacaine alone; analgesic duration might be efficiently extended, whereas the analgesic impact of DEX wasn't reversed if an α2 receptor antagonist has been administered, demonstrating that dexmedetomidine prolonged analgesia via localized action. The hyperpolarization-activated currents defined as Ih (H current) control hypothalamic paraventricular nucleus (PVN) neurons, that are in direct

contact with noradrenergic synapses. The pacemaker current, additionally referred to as Ih, is supposed to be a critical factor in cell excitability. Dexmedetomidine may keep hyperpolarization of cells by suppressing activation of Ih current, prevent subsequent action potential by suppressing K channel, keep cells depolarization, and raise a suppression of Na channel, thus improving impact of LA (26).

Spinal Cord Level

Dex may inhibit release and reuptake of excitatory neurotransmitters through binds to α2 receptors in spinal dorsal horn following local diffusion or systemic absorption. Analgesia is generated if hyperpolarized interneurons suppress ascending spinal route that is associated with nociceptive sensation (27).

Supraspinal Level

Dexmedetomidine is most effective when administered peripherally; however, a few investigations have indicated that it could have systemic absorption, resulting in both peripheral and central impacts. It may produce analgesic properties at the central level by acting on α2A and α2C adrenergic receptors in the brainstem, inhibiting the descending noradrenergic pathway in the medulla, or reducing sympathetic nerve signals. Additionally, it may spread to the cerebrospinal fluid through systemic absorption following intradural delivery or perineural administration (28).

Negative consequences

The local toxicity of perineural DEX was examined in animal models, and the results indicate that delivery of this α2 agonist in a regulated and direct manner at high concentrations ( twenty microgramme per kilogramme) on days one and fourteen does not affect either myelin or axon (29). Paresthesias in the innervation area have been reported to persist for up to 72 hours in clinical settings. This occurred in two volunteers that had 150 microgramme of perineural DEX and three millilitres of 0.75 percent ropivacaine in the ulnar nerve at the elbow (ultrasonic application and non-dominant arm) (30).

Suggestions for Peripheral Nerve Blocks

The optimal dosage of DEX as an adjuvant in peripheral nerve block is dependent upon dosage, level and dosage of local anesthetic agent, location of block, operating technique, people, and other factors. Furthermore, investigator's objective is taken into account when selecting the dose (30).

We found that a dexmedetomidine dosage exceeding fifty microgramme has significant advantages for clinicians who wish to extend period of peripheral nerve anesthesia and enhance the onset of action, as evidenced by numerous of clinical investigations and meta-analyses (31).

Ultrasound measurements

Ultrasound is a form of sound that is unidentified to human ear and has a frequency exceeding 20,000 Hertz. Nevertheless, clinical ultrasound utilizes frequencies that are significantly greater, ranging from one to twenty megahertz and frequently reaching seventy-five megahertz in specialized fields like dermatology and ophthalmology (32).

Passage of ultrasound through the body

A variety of events may occur as ultrasound pulses travel the body. The waves may be scattered, attenuated, scattered in other directions, or reflected back to the instrument. Sound waves show comparable principles to light waves and behave similarly if they encounter an object or pass through air and tissues.

Reflection

A part of the ultrasonic waves is reflected back toward the instrument if they encounter a tissue or material with a varying acoustic impedance. At interfaces at which there is a significant acoustic impedance mismatch and variance, the maximum reflectance is observed. Specular reflectors are the term used to describe these regions of high reflection, which are characterized by the appearance of bright lines or spots (32).

Scattering

Scattering is a phenomenon that arises if an ultrasonography sound wave strikes with a structure that has a smaller wavelength compared to incident sound wave and a variant acoustic impedance compared to the adjacent tissue (Figure 2). 'Diffuse reflectors' are objects that cause scatter and are frequently RBC and small surface irregularities of visceral organs. The ultrasonography waves spread in all directions if they encounter a diffuse reflector. The outcome is the propagation of numerous echoes from numerous small structures. The wave amplitudes of the dispersed reflections are smaller, and these multidirectional waves subsequently intersect and interact with one another. The waves are subjected to constructive and destructive interference as a result of this interaction. The instrument receives multiple echoes with varying intensities, resulting in image known as a "speckle." The ultrasound instrument eventually receives the majority of its echoes from scattering, rather than true reflection. The grainy-grey appearance of organs and the grainy signal in doppler ultrasonography are both a result of these dispersed waves (33).

Blocks techniques

Ultrasonography Guided Femoral Nerve Block

A femoral nerve block induces anesthesia in front and inner parts of the thigh, extending down to knee beside a specific region of skin on inner leg and foot. Femoral nerve also supplies nerves to knee, hip, and ankle joints. The ultrasound-guided method for performing femoral nerve block enables real-time monitoring of distribution of local anesthetic and precise positioning of needle. This technology allows adjustments to be made to achieve the desired spread of the anesthetic. Furthermore, using ultrasound may lower the risk of accidentally puncturing femoral artery. While the success of procedure does not necessarily require nerve stimulation, the presence of a motor response during nerve stimulation can offer valuable safety information, particularly if the relationship between needle and nerve is not accurately identified by ultrasound alone (34).

Ultrasound anatomy (Sonoanatomy)

Being due to femoral nerve's relatively superficial position, femoral nerve block is optimally adapted for ultrasound guidance with a high-frequency linear probe (over ten megahertz). The femoral nerve is positioned on lateral to femoral artery, anterior aspect of iliopsoas muscle,

distal to inguinal ligament and deep to fascia iliaca. The artery is readily recognized by doppler because of its flow and/or pulsation. The femoral nerve is frequently located in a triangular hyperechoic region that is superficial to iliopsoas muscle and lateral to femoral artery. Femoral nerve may appear quite thin and flat in this region, as it branches out into multiple branches. nerve could additionally manifest as an oval or biconvex hyperechoic structure (35).

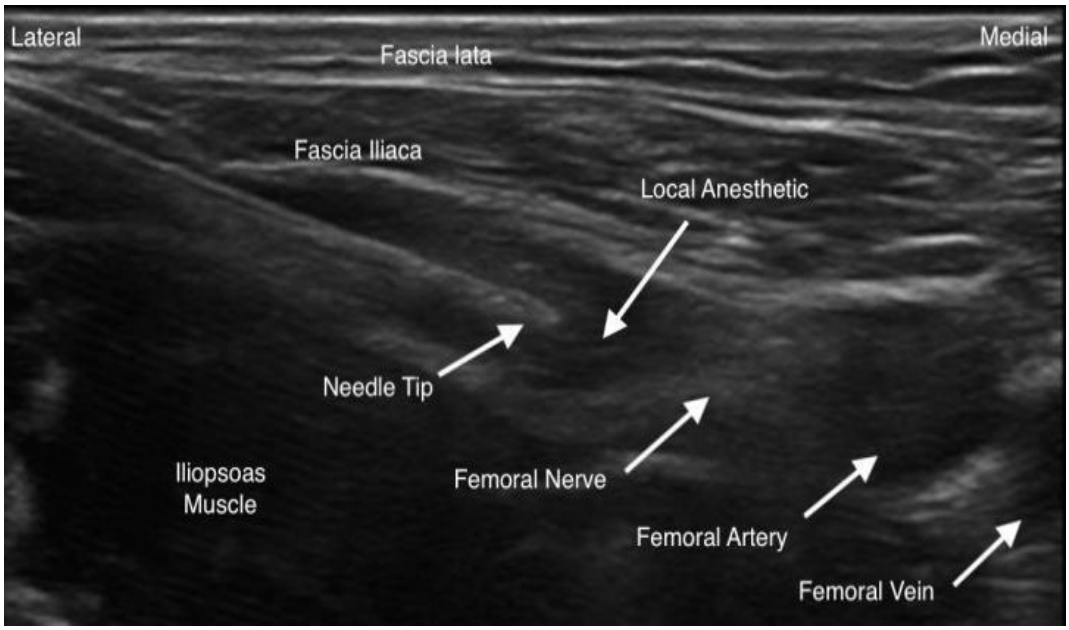


Figure (5): Femoral nerve block (36).

Ultrasound Guided four in one Block

The increased number of lower limb surgical procedures, such as total knee arthroplasty, arthroscopic knee operations, ankle and foot operations, and lower limb fracture fixations, requires improved results with minimal negative impacts on health and quicker mobilization and discharge. Regional anesthesia and central neuraxial and analgesia methods have shown significant reduction in perioperative complications and mortality rates. These techniques have also been shown to decrease metabolic and endocrine responses to operation. However, central neuraxial blocks have been linked to greater complications, morbidity, and delays in ambulation. Regional anesthesia techniques, on the other hand, have been associated with better and earlier results than central neuraxial methods (37).

Utilization of regional anesthesia methods for below knee and knee operations has been extensively researched and provides effective perioperative care for patients of all age groups. Studies have demonstrated that adductor canal blocks offer better recovery outcomes than femoral nerve blocks, especially for knee surgeries, due to their motor-sparing effects. Combining femoral nerve blocks with sciatic nerve blocks has also proven effective in providing adequate pain relief while reducing need for opioids during and after surgery for knee and below knee procedures (3).

Relevant Anatomy

The upper boundary of the Adductor Canal, a musculo-aponeurotic conduit, is Vasto-adductor membrane (VAM). This tunnel is located among adductor longus and adductor magnus muscles on posteromedial side and vastus medialis muscle on anterolateral side, with sartorius muscle situated anteriorly. Femoral vessels, particularly femoral nerve (which includes femoral cutaneous, saphenous, & nerve to vastus medialis branches), and anterior division of obturator nerve in its proximal part were discovered in this canal. The Popliteal vessels are formed by Femoral vessels passing through Adductor Hiatus. In addition to femoral vessels, posterior division of obturator nerve additionally crosses the adductor hiatus. Knee joint is innervated by genicular nerves that originate from a variety of sources, such as distal muscular branch of nerve to vastus medialis (NVM), that keeps going as medial retinacular nerve, in addition to tibial nerve, posterior division of obturator nerve, and saphenous nerve (38).

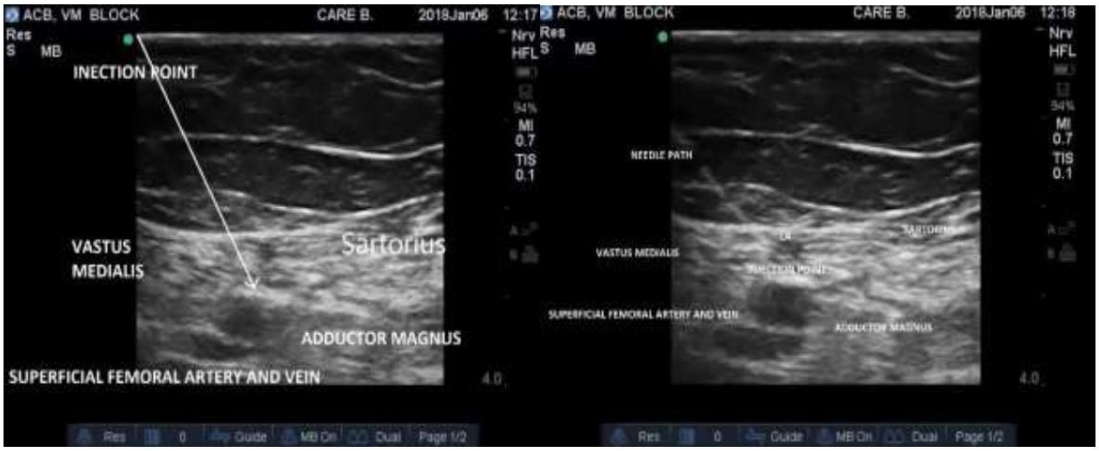


Fig. (6): The LA distributed peri-vascular region in adductor canal, as defined by USG (3).

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