"ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK USING CLONIDINE VERSUS DEXMEDETOMIDINE AS ADJUVANTS TO ROPIVACAINE FOR POST-OPERATIVE ANALGESIA IN UPPER LIMB SURGERIES"

By

Dr. TARUN KUMAR.R.

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

ANAESTHESIOLOGY

Under the Guidance of

Dr. RAVI MADHUSUDHANA

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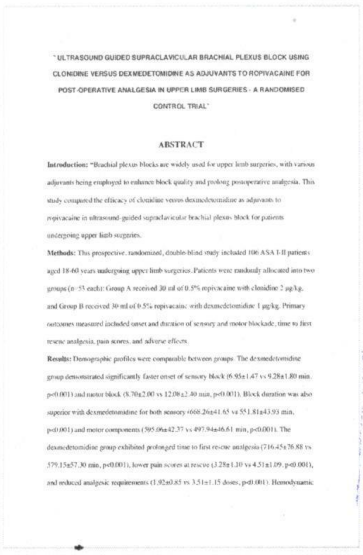

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ABSTRACT

Introduction: "Brachial plexus blocks are widely used for upper limb surgeries, with various adjuvants being employed to enhance block quality and prolong postoperative analgesia. This study compared the efficacy of clonidine versus dexmedetomidine as adjuvants to ropivacaine in ultrasound-guided supraclavicular brachial plexus block for patients undergoing upper limb surgeries.

Methods: This prospective, randomized, double-blind study included 106 ASA I-II patients aged 18-60 years undergoing upper limb surgeries. Patients were randomly allocated into two groups (n=53 each): Group A received 30 ml of 0.5% ropivacaine with clonidine 2 µg/kg, and Group B received 30 ml of 0.5% ropivacaine with dexmedetomidine 1 µg/kg. Primary outcomes measured included onset and duration of sensory and motor blockade, time to first rescue analgesia, pain scores, and adverse effects.

Results: Demographic profiles were comparable between groups. The dexmedetomidine group demonstrated significantly faster onset of sensory block (6.93±1.47 vs 9.28±1.80 min, p<0.001) and motor block (8.70±2.00 vs 12.08±2.40 min, p<0.001). Block duration was also superior with dexmedetomidine for both sensory (608.26±41.65 vs 551.81±43.93 min, p<0.001) and motor components (595.09±42.37 vs 497.94±46.61 min, p<0.001). The dexmedetomidine group exhibited prolonged time to first rescue analgesia (716.45±76.88 vs 579.15±57.30 min, p<0.001), lower pain scores at rescue (3.28±1.20 vs 4.51±1.09, p<0.001), and reduced analgesic requirements (1.92±0.85 vs 3.51±1.15 doses, p<0.001). Hemodynamic

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" ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK USING CLONIDINE VERSUS DEXMEDETOMIDINE AS ADJUVANTS TO ROPIVACAINE FOR POST-OPERATIVE ANALGESIA IN UPPER LIMB SURGERIES - A RANDOMISED CONTROL TRIAL" ABSTRACT Introduction: "Brachial plexus blocks are widely used for upper limb surgeries, with various adjuvants being employed to enhance block quality and prolong postoperative analgesia. This study compared the efficacy of clonidine versus dexmedetomidine as adjuvants to ropivacaine in ultrasound-guided supraclavicular brachial plexus block for patients undergoing upper limb surgeries. Methods: This prospective, randomized, double-blind study included 106 ASA I-II patients aged 18-60 years undergoing upper limb surgeries. Patients were randomly allocated into two groups (n=53 each): Group A received 30 ml of 0.5% ropivacaine with clonidine 2 µg/kg, and Group B received 30 ml of 0.5% ropivacaine with dexmedetomidine 1 µg/kg. Primary outcomes measured included onset and duration of sensory and motor blockade, time to first rescue analgesia, pain scores, and adverse effects. Results: Demographic profiles were comparable between groups. The dexmedetomidine group demonstrated significantly faster onset of sensory block (6.95±1.47 vs 9.28±1.80 min, p<0.001)

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Date: **Dr. TARUN KUMAR.R.**

Place: Kolar

ABBREVIATIONS

ASA	**American Society of Anesthesiologists**
BMI	**Body Mass Index**
BP	**Blood Pressure**
bpm	**Beats per minute**
DEX	**Dexmedetomidine**
DBP	**Diastolic Blood Pressure**
ECG	**Electrocardiogram**
HR	**Heart Rate**
IV	**Intravenous**
kg	**Kilogram**
LA	**Local Anesthetic**
MAP	**Mean Arterial Pressure**
mcg	**Microgram**
mg	**Milligram**
min	**Minute**
ml	**Milliliter**
mmHg	**Millimeters of mercury**
NRS	**Numeric Rating Scale**
NSAID	**Non-Steroidal Anti-Inflammatory Drug**
OT	**Operation Theatre**
PACU	**Post Anesthesia Care Unit**
PNB	**Peripheral Nerve Block**
PONV	**Postoperative Nausea and Vomiting**
RCT	**Randomized Controlled Trial**
RSS	**Ramsay Sedation Scale**
SBP	**Systolic Blood Pressure**
SCB	**Supraclavicular Block**
SD	**Standard Deviation**
SpO2	**Peripheral Oxygen Saturation**
USG	**Ultrasonography/Ultrasound guided**
VAS	**Visual Analog Scale**
α2	**Alpha 2 (adrenergic receptor)**

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ABSTRACT

Introduction: Brachial plexus blocks are widely used for upper limb surgeries, with various adjuvants being employed to enhance block quality and prolong postop analgesia. This study compared the efficacy of clonidine versus dexmedetomidine as adjuvants to ropivacaine in ultrasound-guided supraclavicular brachial plexus block for patients undergoing upper limb surgeries.

Methods: This prospective, randomized, double-blind study included 106 ASA I-II patients aged 18-60 years undergoing upper limb surgeries. Patients were randomly allocated into two groups (n=53 each): Group A received 30 ml of 0.5% ropivacaine with clonidine 2 μg/kg, and Group B received 30 ml of 0.5% ropivacaine with dexmedetomidine 1 μg/kg. Primary outcomes measured included onset and duration of sensory and motor blockade, time to first rescue analgesia, pain scores, and adverse effects.

Results: Demographic profiles were comparable between groups. The dexmedetomidine group demonstrated significantly faster onset of sensory block (6.95±1.47 vs 9.28±1.80 min, $p<0.001$) and motor block (8.70±2.00 vs 12.08±2.40 min, $p<0.001$). Block duration was also superior with dexmedetomidine for both sensory (668.26±41.65 vs 551.81±43.93 min, $p<0.001$) and motor components (595.06±42.37 vs 497.94±46.61 min, $p<0.001$). The dexmedetomidine group exhibited prolonged time to first rescue analgesia (716.45±76.88 vs 579.15±57.30 min, $p<0.001$), lower pain scores at rescue (3.28±1.10 vs 4.51±1.09, $p<0.001$), and reduced analgesic requirements (1.92±0.85 vs 3.51±1.15 doses, $p<0.001$). Hemodynamic parameters remained stable in both groups, with no significant differences in the incidence of adverse effects.

Conclusion: Dexmedetomidine is superior to clonidine as an adjuvant to ropivacaine in

ultrasound-guided supraclavicular brachial plexus block, providing faster onset, prolonged duration of blockade, extended postoperative analgesia, and a favourable safety profile for patients undergoing upper limb surgeries.

Keywords: Supraclavicular block, Ropivacaine, Clonidine, Dexmedetomidine, Postoperative analgesia, Ultrasound-guided regional anaesthesia.

INTRODUCTION

INTRODUCTION

Upper limb surgeries are commonly performed procedures that require effective perioperative pain management for optimal surgical outcomes and patient satisfaction. Peripheral nerve blocks, particularly brachial plexus blocks, have become an integral component of multimodal analgesia for these procedures, offering superior pain control while minimizing the systemic side effects associated with general anaesthesia and opioid analgesics. The supraclavicular approach to brachial plexus blockade has gained widespread acceptance due to its reliability, comprehensive sensory coverage, and relative technical ease when performed under ultrasound guidance. This approach targets the brachial plexus at the level of the trunks and divisions, where the neural elements are compactly arranged, facilitating complete upper extremity anaesthesia with a single injection technique.[1]

The advent of ultrasound guidance has revolutionized regional anaesthesia practice by allowing real-time visualization of neural structures, surrounding vasculature, pleura, and the advancing needle. This technological advancement has significantly enhanced the safety profile and success rate of supraclavicular blocks, greatly reducing the risk of pneumothorax and inadvertent vascular puncture which were previously concerning complications of landmark-based techniques. Kapral et al. demonstrated that ultrasound guidance for supraclavicular brachial plexus blocks increased success rates from 74% to 99% when compared to nerve stimulation techniques alone.[2] Additionally, ultrasound guidance has been shown to reduce the onset time of sensory and motor blockade, decrease the volume of local anaesthetic required, and extend the duration of analgesia, making it the current gold standard for performing this block.[3]

Local anaesthetics constitute the primary pharmacological agents utilized in peripheral nerve blocks, with ropivacaine emerging as a preferred choice for many practitioners. Ropivacaine, an amide local anaesthetic, offers a favourable profile with decreased cardiotoxicity and neurotoxicity compared to bupivacaine, while providing effective sensory blockade with less profound motor blockade. This differential blockade characteristic is particularly advantageous in ambulatory surgery settings, facilitating earlier mobilization and discharge. However, despite these advantages, the duration of analgesia provided by ropivacaine alone remains limited to approximately 6-8 hours, which is often insufficient for comprehensive post-operative pain management in many upper limb surgical procedures that may induce pain lasting 24-48 hours.[4]

The pursuit of extended post-operative analgesia without escalating local anaesthetic doses and associated toxicity risks has led to the exploration of various adjuvants to enhance block efficacy and duration. Among these, alpha-2 adrenergic receptor agonists have demonstrated considerable promise. These agents act through a variety of mechanisms including vasoconstriction (reducing systemic absorption of local anaesthetics), direct inhibition of peripheral nerve action potentials, suppression of the release of pro-inflammatory mediators, and modulation of spinal and supraspinal pain pathways.[5] Two such agents that have garnered significant clinical interest are clonidine and dexmedetomidine, with the latter being a more selective alpha-2 adrenergic receptor agonist with an alpha-2 selectivity ratio of 1620:1 compared to 220:1 for clonidine.[6]

Clonidine, originally developed as an antihypertensive agent, has been used as an adjuvant in regional anaesthesia for several decades. When added to local anaesthetics

in peripheral nerve blocks, clonidine has been shown to extend the duration of both sensory and motor blockade. A meta-analysis by Pöpping et al. demonstrated that clonidine prolongs the duration of postoperative analgesia by approximately 2 hours when added to intermediate or long-acting local anaesthetics in various peripheral nerve blocks.[7] The proposed mechanisms include local vasoconstriction, direct inhibition of impulse conduction in peripheral nerves, and enhancement of local anaesthetic activity through opening of potassium channels leading to hyperpolarization of nerve cells. Additionally, clonidine may exert anti-inflammatory effects at the surgical site, further contributing to its analgesic properties. However, the use of clonidine as an adjuvant is not without potential systemic effects, including sedation, hypotension, and bradycardia, which necessitate careful patient selection and monitoring.

Dexmedetomidine, a relatively newer alpha-2 adrenergic agonist, has emerged as a promising alternative to clonidine for peripheral nerve blocks. Its higher selectivity for alpha-2 receptors theoretically offers the potential for enhanced analgesic efficacy with a more favourable side effect profile. Experimental studies have demonstrated that dexmedetomidine enhances both sensory and motor blockade of local anaesthetics without inducing neurotoxicity. Brummett et al., in their animal studies, showed that perineural dexmedetomidine added to ropivacaine enhanced the duration of sensory blockade in a dose-dependent manner without evidence of histological damage to neural tissues.[8] This preclinical evidence has translated into clinical benefits, with numerous studies reporting significant prolongation of analgesia when dexmedetomidine is added to local anaesthetics in various peripheral nerve blocks.

The comparative efficacy of clonidine versus dexmedetomidine as adjuvants to local anaesthetics in brachial plexus blocks represents an area of active clinical research.

While both agents have demonstrated analgesic benefits, the literature suggests potential advantages of dexmedetomidine over clonidine. Das et al. conducted a randomized trial comparing these two adjuvants in supraclavicular brachial plexus blocks and found that dexmedetomidine (1 μg/kg) added to ropivacaine (0.5%) provided longer duration of sensory and motor blockade, extended postoperative analgesia, and better preservation of hemodynamic parameters compared to an equivalent dose of clonidine.[9] These findings may be attributed to the higher alpha-2 selectivity of dexmedetomidine, resulting in more potent activation of alpha-2 adrenoreceptors in the peripheral nerves.

Despite these promising results, several aspects of adjuvant usage in peripheral nerve blocks remain inadequately defined. The optimal dosing regimens for both clonidine and dexmedetomidine when used as adjuvants to ropivacaine in supraclavicular blocks have not been conclusively established. Additionally, while most studies report prolongation of analgesia with these adjuvants, the magnitude of this effect varies considerably across different investigations, potentially due to methodological differences, variability in patient populations, and diverse surgical procedures. Furthermore, concerns regarding the safety profile of these adjuvants, particularly with respect to potential neurotoxicity and systemic side effects, warrant continued investigation."

The impact of adjuvant-enhanced peripheral nerve blocks on broader perioperative outcomes represents another important area of inquiry. Enhanced and prolonged analgesia may translate into improved rehabilitation outcomes, reduced opioid consumption and associated adverse effects, shortened hospital stays, and increased patient satisfaction. However, these potential benefits must be weighed against the risks of adjuvant-related adverse events. While the safety profile of perineural

dexmedetomidine and clonidine appears favourable in the short term, long-term safety data remain limited. Theoretical concerns include potential neurotoxicity, prolonged motor blockade delaying mobilization, and systemic absorption leading to cardiovascular effects. Abdallah et al. conducted a meta-analysis of randomized controlled trials investigating perineural dexmedetomidine as an adjuvant to local anaesthetics and found no serious adverse events attributable to the adjuvant, though transient bradycardia and hypotension were reported in some studies.[10]

The current research landscape surrounding alpha-2 adrenergic agonists as adjuvants in supraclavicular brachial plexus blocks suggests that both clonidine and dexmedetomidine enhance the duration and quality of ropivacaine-induced analgesia. However, the comparative efficacy of these agents, their optimal dosing, safety profiles, and impact on perioperative outcomes require further elucidation through well-designed, adequately powered randomized controlled trials. The present study aims to address these knowledge gaps by directly comparing the efficacy and safety of clonidine versus dexmedetomidine as adjuvants to ropivacaine in ultrasound-guided supraclavicular brachial plexus blocks for patients undergoing upper limb surgeries. The findings will contribute to the evolving evidence base guiding adjuvant selection in peripheral nerve blocks and potentially inform clinical practice guidelines for optimizing perioperative pain management in upper limb surgical procedures.

OBJECTIVES

AIMS & OBJECTIVES

1. **"Primary objective :** to compare the effects of clonidine or Dexmedetomidine as an adjuvant to ropivacaine in SCBP block for upper limb surgeries in terms of post-operative analgesia.
2. **Secondary objective:** to determine the onset times of sensory and motor blocks, duration of sensory and motor blocks, first rescue call for pain post operatively and adverse effects if any."

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Historical Perspectives of Regional Anaesthesia of the Upper Limb[11]

"Upper limb regional anaesthesia is one of the most significant advancements in surgical pain treatment. Its origins can be traced back to the late 1800s, when cocaine was initially used as a local anaesthetic. In 1884, Austrian ophthalmologist Carl Koller demonstrated cocaine's anaesthetic qualities for eye surgery, ushering in the era of local anaesthesia in clinical practice. Upper limb blocks were developed shortly thereafter, with William Stewart Halsted performing the first brachial plexus block using cocaine in 1885. His technique included surgically exposing the nerves prior to injection, which was significantly more intrusive than modern procedures. In 1897, August Bier devised intravenous regional anaesthesia (today known as the Bier block), making upper limb procedures easier. In the early twentieth century, brachial plexus blocks were significantly refined. Georg Hirschel originally reported the percutaneous axillary route to the brachial plexus in 1911. That same year, Diedrich Kulenkampff created the supraclavicular technique, which quickly gained favour because to its dependability in the face of pneumothorax hazards. Achille Dogliotti invented the interscalene technique in 1928, which expanded the choices for shoulder and upper arm surgeries. The post-World War II era saw significant technological breakthroughs. The advent of longer-lasting local anaesthetics such as lidocaine (1948) and bupivacaine (1963) allowed for longer surgical procedures. The advancement of nerve stimulator technology in the 1960s increased block precision while decreasing complications and failure rates. Perhaps the most significant improvement occurred in the 1990s, with the introduction of ultrasonography guiding for regional blocks. This technology enabled practitioners to visualise brain structures

in real time, significantly increasing accuracy and safety. Today, ultrasound-guided procedures are the standard of care, allowing for targeted nerve blocks with less anaesthetic volumes and lower complication rates. Modern treatment is evolving with continuous catheter procedures and the development of increasingly safer, longer-acting local anaesthetics, ensuring that regional anaesthesia remains a cornerstone of upper limb surgery care.

BRACHIAL PLEXUS BLOCK

The brachial plexus can be obstructed at numerous locations, resulting in varying results. As a result, being familiar with numerous techniques is advantageous given the variety of patient anatomy and indications. The various brachial plexus blocks include:

- "Interscalene
- Superior trunk, a potentially phrenic nerve-sparing alternative to the interscalene approach[12]
- Supraclavicular
- Infraclavicular
- Traditional
- Retrograde approach (RAPTIR)[13]
- Axillary"

The interscalene block, which provides anaesthesia for the shoulder, arm, and forearm, is the most common brachial plexus block. It is carried out by injecting a local anaesthetic around the brachial plexus at the nerve roots between the anterior and middle scalene muscles. The needle is placed cephalad towards the neck until it reaches the bone. [14]

The supraclavicular block gives anaesthesia and pain relief to the upper extremity below the shoulder. It's a good option for elbow and hand surgery. This method includes putting a needle into the supraclavicular fossa, which is immediately lateral to the clavicle. A local anaesthetic can be injected around the brachial plexus, at its confluence with the subclavian artery. [15]

The infraclavicular block is used for surgeries on the lower arm and hand. This method entails placing a needle into a depression just medial to the scapular coracoid process to administer local anaesthetic around the brachial plexus at its connection with the axillary artery. [16] The retrograde method for an infraclavicular block (RAPTIR) uses ultrasound guidance to locate particular nerve branches within the proximal axilla, allowing for selective blocking of certain nerves or combinations of them. [17]

The classic axillary block is used for procedures that include the lower arm and hand nerves. This method entails putting a needle into the axilla near the humerus and injecting local anaesthetic around the brachial plexus at its confluence with the axillary artery.[18]

RELAVANT ANATOMY

Neural Elements

Upper extremity regional anaesthesia requires a good understanding of brachial plexus anatomy to assist block placement and optimise patient-specific block selection. According to Gray's Anatomy, the brachial plexus is a network of nerves that originate as spinal nerve roots and extend to the terminal branches that serve the upper extremities. The brachial plexus begins with the fusion of the ventral major rami of cervical nerves 5 through 8 (C5-C8), which includes a significant portion of

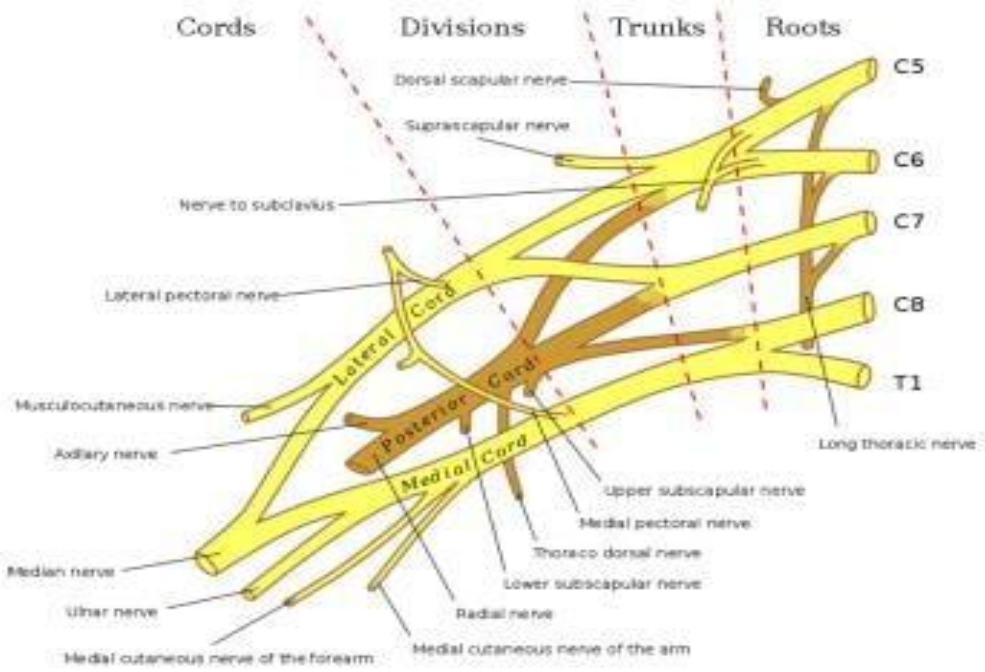
the first thoracic nerve (T1). Variable contributions may also arise from the fourth cervical (C4) and second thoracic (T2) nerves. 19 The brachial plexus roots are located in the ventral rami.

The C5 and C6 rami normally join along the medial border of the middle scalene muscle to produce the superior trunk of the plexus; the C7 ramus forms the middle trunk; and the C8 and T1 rami form the inferior trunk. The absence of an anterior tubercle on the C7 transverse process makes it easier to identify the C7 nerve root using ultrasonography. [20] The roots and trunks flow via the interscalene groove, which is a palpable surface anatomical marker connecting the anterior and middle scalene muscles. The three trunks are anatomically separated into anterior (flexor) and posterior (extensor) divisions at the lateral boundary of the first rib. Divisions are further reorganised into cords, which are distinguished by their spatial relationship to the second section of the axillary artery. The lateral cord of the plexus is formed by the anterior divisions of the superior and middle trunks, the posterior cord by the posterior divisions of all three trunks, and the medial cord by the inferior trunk's anterior division. The plexus' terminal branches are formed by the three cords, each of which has two major terminal branches and a variable number of lesser intermediary branches. The lateral cord provides the musculocutaneous nerve and the lateral portion of the median nerve. The dorsal portion of the upper extremity is typically supplied by the posterior cord via the radial and axillary nerve. The medial cord contains the ulnar nerve and the medial component of the median nerve. The medial antebrachial cutaneous nerve and the medial cutaneous nerve, which connect with the lesser intercostobrachial nerve (T2) to innervate the skin on the medial portion of the arm, are important intermediate branches of the medial cord. [19] Despite the aforementioned "classic" schema, 7 primary topologies of the brachial plexus have

been reported, with none having more than 57% representation; in fact, 61% of individuals have right/left asymmetry. [21] These normal anatomic variations are especially important during ultrasonic examination of the upper extremity neural components because they allow for direct visualisation of normal variants such as a solitary trunk, a postfixed plexus where T2 contribution leads to a lesser or absent C5 nerve root,22 or C5 and C6 nerve roots that penetrate the anterior scalene muscle rather than reside within the interscalene groove. It is unclear if these anatomical changes have a major impact on the successful delivery of upper extremity regional anaesthesia. [23] The architecture of the brachial plexus and the anatomy of peripheral nerves help to explain the pathophysiology of perioperative nerve damage. Peripheral nerves are made up of a variety of fascicles, each of which contains distinct nerve fibres (axons) within the endoneurium. The perineurium contains fascicles, whereas the epineurium contains fascicle groups. As the nerve moves out from the spinal cord, the density of the epineurium (stroma and connective tissue) decreases as the total volume grows. The amount of neural tissue remained steady. Thus, the ratio of nonneural to neural tissue in the epineurium grows from 1:1 in the proximal plexus to 2:1 in the distal plexus, where a peripheral nerve's cross-sectional area can be 70% loose connective tissue. [24] This observation may have clinical implications since when a needle inadvertently penetrates a peripheral nerve, it does not always end up in a fascicle but can also end up in connective tissue. Peripheral nerve architecture also influences patterns of local anaesthetic blockage and clearance. Local anaesthetic is first absorbed by the mantle fibres on the nerve's periphery, causing blockage from proximal to distal. Block resolution, on the other hand, occurs in a distal-to-proximal pattern, implying that the core fibres' vascularity preferentially clears local anaesthetics. [25] The higher ratio of nonneural to neural tissue as one advances out

from the spinal cord may also explain the somewhat longer block onset periods with distal versus more proximal techniques."[24]

Figure 1: Anatomy of Brachial Plexus



Other Pertinent Neuroanatomy

"Several nerves that are either substantial branches of or not part of the brachial plexus are clinically significant in upper extremity surgery because they may necessitate separate blocking or suggest needle malposition. The supraclavicular nerves, which are branches of the superficial cervical plexus (C3-C4), innervate the "cape" of the shoulder, which extends from the anterior second rib to the top of the scapula. The phrenic nerve (C3-C4, occasionally C5) runs over the anterior scalene muscle and can be accidentally stimulated if the block needle is positioned too far anterior during interscalene block (ISB). The C5 anterior rami and the phrenic nerve are separated by only 2 mm, and the gap between these two structures grows as one approaches caudad. Stimulation of the dorsal scapular nerve (C5) results in rhomboid

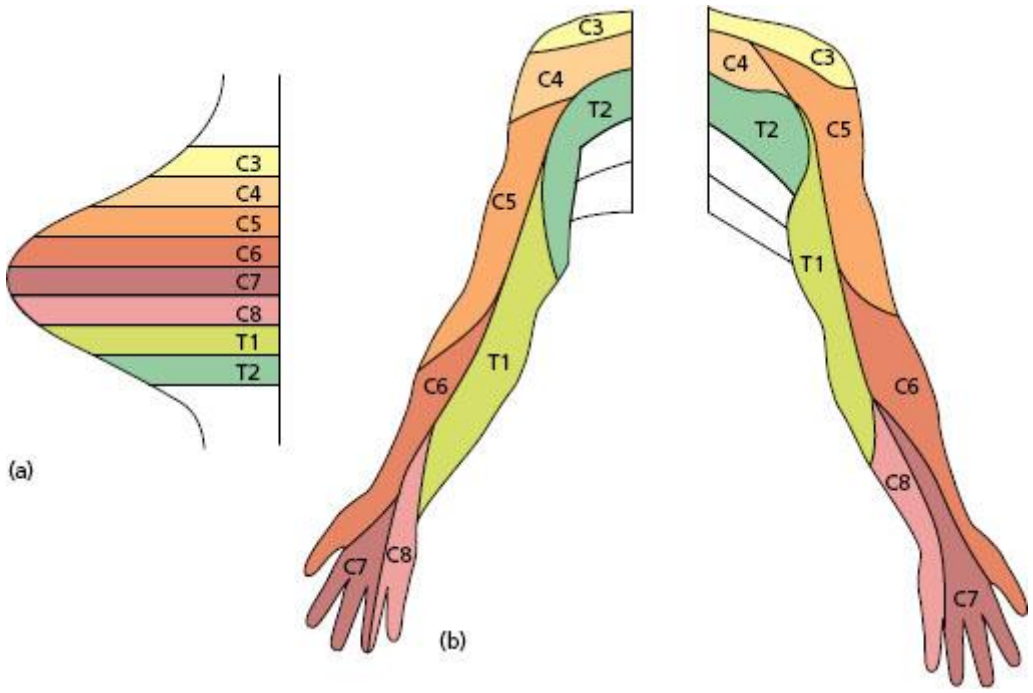
and levator scapulae motor responses, indicating that the block needle is positioned too far posteriorly. The supra-scapular nerve (C5-C6) originates in the upper trunk and distributes sensory fibres to the shoulder capsule and acromioclavicular joint. The intercostobrachial nerve, together with the medial cutaneous nerve, innervates the upper part of the arm's posterior and medial skin. It starts from the second intercostal nerve (T2).[26]

Innervation of the Arm- Sensory and Motor

The sensory and motor innervation of the upper extremity is clinically significant because it determines which cutaneous nerve distributions within a surgical field require conduction blockade, which terminal nerve branches require supplementation for an incomplete block, and the presence and distribution of preoperative and postoperative neurological deficit. The upper extremity's cutaneous nerves are a collection of neural fibres that arise from several spinal cord segments. Assigning cutaneous region to a particular peripheral nerve is inconclusive, if not impossible. Indeed, this scenario greatly undermines the scientific evaluation of sensory blockage in most research studies. Motor innervation is therapeutically relevant because it allows us to link a peripheral nerve stimulator (PNS)-induced motor response to the main nerve(s) that were stimulated. For example, superior trunk stimulation causes a deltoid motor response. Musculocutaneous nerve stimulation causes the arm to bend at the elbow. Median nerve stimulation causes forearm pronation, wrist flexion, and thumb opposition. Ulnar nerve motor responses include ulnar deviation of the wrist, little finger flexion, thumb adduction, and finger flaring. Radial nerve stimulation is associated with wrist and finger extensions.[26]

Figure 2: Cutaneous sensory distribution of the upper extremity. Terminal nerves of the brachial plexus provide sensory innervation to the arm.

The sensory distribution of these nerves is variable and overlapping—as depicted by blended colours as the zones converge.



Vascular Elements

In addition to neurological elements, various circulatory structures are clinically significant as anatomic markers or structures to avoid. The vertebral artery travels cephalad from its origin in the subclavian artery to the C6 level, where it enters the vertebral foramen in each cervical vertebral transverse process. As the cervical roots of the brachial plexus exit the intervertebral foramina, they run immediately posterior to the vertebral artery, creating an interposed portal for potential intravascular injection, especially if the anaesthetising needle runs anterior and medial

to the anterior scalene muscle. The external jugular vein frequently overlies the interscalene groove at the level of C6, although it is not a reliable anatomic marker.

The subclavian artery runs alongside the brachial plexus as it passes over the first rib. The trunks/divisions of the brachial plexus lay posterior, cephalad, and finally lateral to the subclavian artery,[27] providing a useful anatomical link for supraclavicular block implantation. The cords are distinguished by their lateral, posterior, or medial proximity to the second section of the axillary artery, however their precise location varies somewhat across people. The axillary artery is located in the base of the axilla, anterior to the radial nerve, posteromedial to the median nerve, and posterolateral to the ulnar nerve. However, there is great individual variance. Changes in arm posture and/or external pressure during block performance have an impact on nerve-vascular interactions, which is practical.[28]

Ultrasound Guided Regional Anaesthesia

In the mid-1990s, anaesthesiologists at the University of Vienna investigated the use of ultrasonography (US) to guide peripheral nerve block (PNB). Although radiologists had previously used ultrasound technology to guide needles for biopsy, using this imaging modality for PNB was new at the time. The ability of ultrasonography to help a variety of regional anaesthesia methods, including brachial plexus and femoral blocks, was established. A decade later, colleagues at the University of Toronto in Canada began to adopt this technology, demonstrating its value and detailing the sonoanatomy of the brachial plexus. Ultrasound guiding has gained popularity as a nerve localisation method in humans, and its application during regional anaesthesia has lately been dubbed the "new gold standard."

Ultrasound is produced when several piezoelectric crystals within a transducer (the

probe) vibrate rapidly in response to an alternating electric current.

This produces a mechanical kind of energy called a "high" frequency sound wave.

Ultrasound then travels through the body, where it might be reflected, refracted, or scattered when it comes into touch with different tissues. Structures that reflect ultrasound more strongly seem whiter, or hyperechoic. Structures that reflect ultrasound to a lower extent are termed as hypoechoic, appearing darker. As a result, all structures appear in various shades of grey, allowing for precise tissue diagnosis (e.g., needle from nerve).

Advantages of Ultrasound

Previous surface anatomy-based procedures, such as nerve stimulation, landmark palpation, fascial "clicks," paresthesias, and transarterial approaches, did not allow for monitoring of the local anaesthetic injectate's disposition. However, ultrasound guidance has some significant practical advantages for nerve block. Ultrasound enables visualisation of the anatomy of the region of interest. This enables for better informed guidance of the needle passage to the target while avoiding structures that the needle may damage. Ultrasound also enables for visualisation of the needle tip as it passes through the tissues, ensuring alignment with the desired path and lowering the risk of unintentional needle harm to other structures. Perhaps most importantly, real-time ultrasound imaging allows for continuous viewing of local anaesthetic solution supply to ensure adequate distribution, with the option of adjusting the needle tip location as needed to optimise local anaesthetic distribution.
The use of ultrasound guidance in regional anaesthesia has resulted in the refining of several nerve block procedures, increased usage of PNB, and higher acceptance by surgical colleagues and patients.

Ultrasound guiding has the advantage of eliminating the detrimental impact of individual patient anatomy on block success rates. Compared to nerve stimulation alone, ultrasonic guiding has been demonstrated to result in a higher rate of successful peripheral nerve blockade, shorter block set-up times, and longer block durations.[29]

Nerve Imaging with Ultrasound

Because peripheral nerves can be difficult to recognise from surrounding background structures, it is critical to understand all of their differentiating characteristics. The smallest peripheral nerves observed with ultrasonography are the digital nerves. 30 These nerves, which measure 2 mm in diameter, were tested to assess nerve repair. While resolution continues to improve, most nerves for regional blockage may now be visualised using ultrasound technology. Nerves can be circular, oval, or triangular in shape. Interestingly, as a neurone travels between adjacent structures, it can take on all three configurations.[31]

Scan along the nerve's known route to determine its identity. Ultrasound can easily track the oblique course of nerves, which is difficult to do with other imaging modalities like magnetic resonance imaging. To follow the path of a nerve, short axis (transverse) scanning is preferred.

Advances in ultrasound technology will enable the imaging of smaller and deeper nerves. High-frequency broadband linear probes are now the most effective nerve imaging tools. Greyscale postprocessing maps are often used to display nerve sonograms, however recent data suggests that colour encoding received echoes improves musculoskeletal imaging.[32]

Needle Visibility

The needle's ultrasonic visibility is mostly determined by its insertion angle and gauge. At steep angles, the ultrasonic transducer receives backscatter from the needle rather than specular reflections. This results in a significant reduction in needle tip visibility. Many authors have emphasised the vital need of establishing needle tip visibility before extending the needle when using the in-plane technique. However, needle tip vision is inherently limited at steep angles, which might cause complications. Entering the skin with the needle near to the transducer disrupts the surface contact and forces steep angles towards the target.[33]

Larger-bore needles are easier to see for two reasons. First, the increased cross-sectional area makes the needle simpler to find. Second, larger needles are less flexible, making them less likely to bend away from the imaging plane. One method has been to utilise large-bore (17-gauge) needles to improve needle tip visibility in deeper blocks. [34]

The role of the acoustic background is important. The needle tip is better seen in dark (anechoic) veins or under local anaesthetic. A dark background, which can be generated by lowering the receiver gain, can improve needle tip visibility. Commercial changes (coating or dimpling) to improve echogenicity of regional block needles are technically viable, but have not yet been sold.

Vascular punctures have been documented despite the use of the in plane approach, highlighting the necessity of needle tip visibility in clinical practice. These unintentional vascular punctures occurred despite the fact that arteries are the easiest anatomical structures to identify with ultrasonography. However, the arterial puncture rates with ultrasound guidance are likely lower than with other approaches to regional block.[35]

Local Anaesthetic Solutions and Injection

Injecting a quiescent (unagitated) solution can act as a reverse contrast agent, highlighting the boundaries of the anaesthetised nerve. Nerves are generally simpler to recognise following the administration of an undisturbed local anaesthetic, and they can occasionally be seen floating freely inside the injected fluid. To determine the location of the tip, inject small volumes of air (0.3-0.5 ml) into the tissue using a needle. 36 Although bubbles are easily identified sonographically and can serve as a useful marker of the needle tip, they can also scatter in tissue and generate acoustic shadowing, which can be problematic. As a result, the local anaesthetic solution is cleared of any air bubbles before to injection. Most practitioners avoid using bicarbonate-based local anaesthetic solutions because they produce carbon dioxide, which obscures imaging.

One of the most significant advantages of ultrasonic imaging is the ability to reposition the needle following an initial administration of local anaesthetic. Test injections to visualise local anaesthetic distribution should be small (1-2 mL). If the local anaesthetic distribution is not visible on the monitoring screen, immediately stop, aspirate, and adjust the transducer or needle (do not continue to inject because accidental intravascular injection is a possibility). If the local anaesthetic distribution does not sufficiently encompass the nerves, the block needle can be adjusted, and the test injections can be repeated. If the relevant fascial planes are recognised, there is no need to contact nerves with the block needle in order to surround them with local anaesthetic. After injection, the local anaesthetic distribution can be examined by sliding the transducer down the nerve path while seeing the nerve in the short axis.

Imaging planes and approaches to the regional block.

Imaging Planes for Nerves

Nerves can be seen in both short and long axes. Many anaesthesiologists are familiar with this nomenclature because it is used in transoesophageal echocardiography. Similarly, the phrases transverse and longitudinal have been employed in radiology research. For a variety of reasons, ultrasound-guided nerve blocks are commonly administered using short axis imaging. First, peripheral nerves are rather easy to identify. Second, there is strong resolution of the fascial barriers that surround nerves. Third, dynamic measurement and verification of circumferential distribution of local anaesthetic with injection are feasible. Finally, if the transducer moves slightly, the image remains usable (an oblique view of the nerve). For these reasons, short axis views of peripheral nerves for regional blocks have become standard practice in many institutions.

The Out-of-plane Needle Approach

The out-of-plane (OOP) technique includes inserting the needle so that it crosses the imaging plane close to the target. With this method, the target is normally centred inside the field of view and the depth is recorded. If the needle tip is not visible, the injection endpoint becomes less evident, and small-volume test injections may be required to visualise proper local anaesthetic dispersion. The OOP technique can be made similar to the in-plane (IP) technique (see section entitled The In-plane Needle Approach) by sliding and tilting the transducers such that they follow the needle tip.[37]

The In-plane Needle Approach

To visualise the entire shaft and tip, place the needle within the imaging plane. For the IP technique, the imaged needle path should be maximised by positioning the target on the opposite side of the imaging field of view from the approaching needle. The transducer can be moved to bring the needle into the imaging plane. If the needle tip cannot be readily detected inside the imaging plane, do not advance it. When the needle is in plane (longitudinal scan), the in vivo sonographic image will be hyperechoic, with parallel hyperechoic trails visible away from the transducer. These hyperechoic traces come from reverberations within the needle itself.[38]

SUPRACLAVICULAR BLOCK

"The supraclavicular technique to the brachial plexus block was first reported in the early twentieth century, and it is often regarded as the most effective brachial plexus (BP) approach. With this method, the BP trunks/divisions are compact and superficial to the skin, making them simple to see on ultrasonography. Due to the restricted surface area, the entire BP is anaesthetized. The supraclavicular block also has the most scope of sensory blocking of any BP method, making it excellent for providing dense, rapid onset, and effective anaesthesia and analgesia for procedures ranging from the shoulder joint and mid-humerus proximally to the hand distally. [39] According to the literature, the supraclavicular approach is one of the safest and most effective methods. [40]

Indications.

The supraclavicular block is used to provide an anaesthetic block during surgery or for intra- and postoperative pain relief for the entire upper limb, including the shoulder, arm, elbow, forearm, and hand. Common indications for this block include

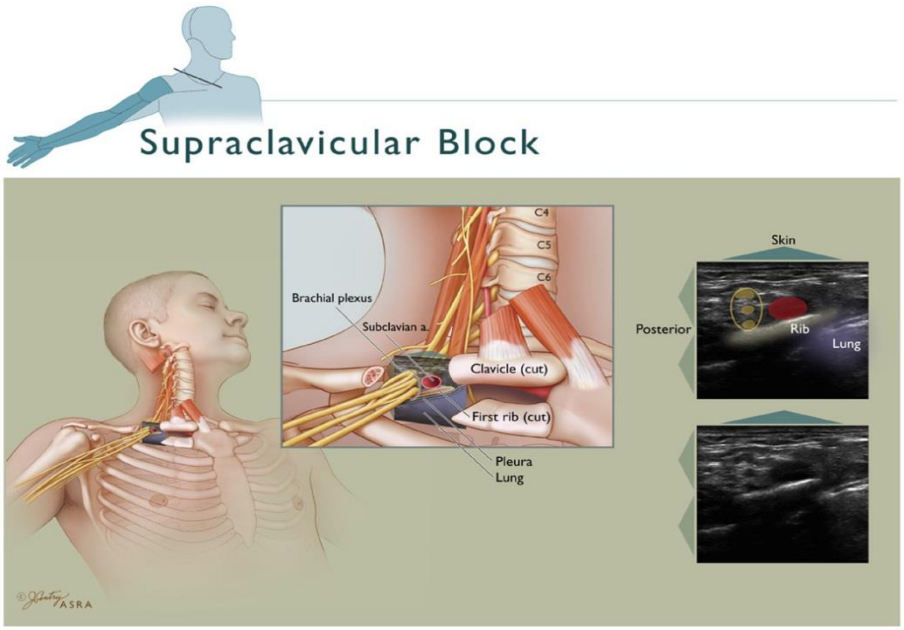
fractures, dislocations, and abscesses, as well as general upper extremity surgery for adults and children. It also treats upper extremity pain from surgery and trauma, complex regional pain syndrome, amputation, vascular disease, and tumours. Prior to use, relative and absolute contraindications should be examined, which may include pre-existing neuropathies or nerve injury, as well as severe coagulopathies or anticoagulants. Other contraindications include infection at the injection site, difficulties to implant the needle due to a splint or cast, and allergy to local anaesthetics.[41]

Procedure[42]

1. “Place the ultrasound probe in the supraclavicular fossa in a transverse orientation, aimed caudad relative to the thoracic cavity, to visualize the brachial plexus near the subclavian artery.
2. Visualize the visceral and parietal pleurae interface, seen as "lung sliding" on ultrasound. Additionally, visualize the first rib as a hyperechoic line with a dropout artifact deep relative to itself. The plexus and subclavian artery should be visualized overlying the first rib. This allows the first rib to be used as a bony backstop, decreasing the risk of pneumothorax. Rotating the lateral edge of the probe more posteriorly may aid in obtaining this window.
3. Insert a block needle in-plane from posterior to anterior and lateral to medial, aiming for the deep portion of the brachial plexus where it overlies the first rib.
4. Confirm negative aspiration, then inject a small amount of local anaesthetic to raise the plexus of the first rib.
5. Advance the needle along the first rib to the anterior aspect of the plexus, adjacent to the subclavian artery. This is called the "corner pocket." It is essential to completely cover this area for a successful block.

6. Confirm negative aspiration, then inject another 1 mL to 2 mL of the local anaesthetic. Repeat until about 10 mL has been injected deep relative to the plexus.
7. Retract the needle to the skin, then readvance at a shallow angle, guiding the needle to the superficial aspect of the brachial plexus.
8. Confirm negative aspiration, then inject 1 mL to 2 mL of local anaesthetic superficial to the plexus. Repeat until about 10mL has been injected superficially relative to the plexus.
9. Three primary variations of this block have been described—the subclavian perivascular approach, the "plumb-bob" approach, and an ultrasound-guided approach. This block is performed where the brachial plexus is presented most compactly—at the distal trunk/proximal division level. This compactness may explain the block's historical reputation for providing short latency and complete, reliable anaesthesia for upper extremity surgery, although confirmatory data do not exist. Several technical caveats apply to supraclavicular plexus block. First, the risk of pneumothorax may be substantially reduced by technical modifications of the block, which are discussed in the section on pneumothorax. Second, stimulation of the middle trunk (hand contraction or paresthesia) has been associated with higher success rates for hand surgery. Third, if ultrasound-guided regional anaesthesia (UGRA) is used, concomitant PNS is redundant for improving block success. Finally, in contrast to the contention that UGRA facilitates blockade with smaller volumes of local anaesthetic, the minimum volume required for UGRA supraclavicular blockade in 50% of patients is 23 mL, which is similar to recommended volumes for traditional nerve localization techniques."[35]

Figure 3: Supraclavicular Block



Complications

The supraclavicular brachial plexus block poses clinically considerable risks of pneumothorax and subclavian artery puncture. Ipsilateral hemi-diaphragmatic paralysis is a known consequence; however the risk is far smaller than that of an interscalene block. Furthermore, if complete anaesthetic spread between the first rib and the plexus is not obtained, the ulnar nerve may be spared.[35]

Advantages of ultrasound-guided supraclavicular BP blocks:

Several studies in the literature have compared the relative blocks for upper limb procedures, with the majority concluding that the supraclavicular block has the highest success rates and greater anaesthetic distribution. This is mostly because the anaesthetic solution is administered at the level of the BP's trunks/divisions. This is a compact component of the BP that ensures early onset, and because the needle is

normally put above the clavicle to a maximum depth of 1.5 cm, pneumothorax is less common. Success rates for supraclavicular BP have been reported to be 85% or higher.[43]

Disadvantages of ultrasound-guided supraclavicular BP blocks:

Despite the advantages of ultrasonography guiding, problems can sometimes occur. Additional research has been conducted to determine the prevalence of problems that may arise both with and without the use of ultrasonography guidance. The reported rate of pneumothorax without ultrasound guidance ranges from 0.5% to 6%, whereas the incidence with ultrasound guidance is 1%. Other consequences that may occur include recurrent laryngeal nerve injury, which causes a hoarse voice (a reported incidence of 1%), phrenic nerve injury, which causes hemi-diaphragmatic paresis (a reported incidence of 33%), and vascular puncture (0.4%). Horner's syndrome has also been recorded as a result of ipsilateral sympathetic cervical chain paralysis caused by medications, surgery, or local compression (1% reported occurrence). The latter can be generated by injecting significant amounts of local anaesthetic or by unusual proximal migration of the solution above the clavicle into the interscalene region. Horner's syndrome is distinguished by a trio of symptoms: miosis (excessive pupil constriction), ptosis (drooping of the upper eyelid), and anhidrosis (lack of sweating or failure of the sweat glands). The anaesthetist's level of experience also influences the rate of complications. Thus, adequate skill, regular practice, and a solid understanding of human anatomy are required to conduct the supraclavicular nerve block, whether under ultrasound guidance or without.[44]

ADJUVANTS IN REGIONAL ANAESTHESIA: [45]

The complexity of the pain transmission mechanism in both the central and peripheral nerve systems need a multimodal analgesic approach. Adding adjuvant drugs to local anaesthetics for peripheral nerve blocks (PNBs) improves and prolongs analgesia while lowering the amount of local anaesthetic required, hence reducing potential toxicity. Adjuvants such as opioids, NSAIDs, and α2-agonists can enhance the effects of local anaesthetics without causing neurotoxicity at clinical levels. However, they may cause adverse effects such as hypotension, drowsiness, and bradycardia. This multimodal strategy is especially useful in outpatient and day surgery settings, when sustained analgesia is critical, and it helps avoid continuous catheter placement, which reduces infection risks. Despite their broad clinical use, adjuvants for PNBs remain off-label without FDA approval, forcing anaesthesiologists to carefully balance benefits and hazards. Evidence supports the use of buprenorphine, dexmedetomidine, and dexamethasone as adjuvants with favourable benefit-to-risk profiles, however the medical community continues to debate their ideal utilisation.

Classification of adjuvants

Adjuvants are classified according to their mode of action, application, and other parameters. The major categories of adjuvants are as follows:

“Opioids

- Buprenorphine
- Morphine
- Fentanyl
- Sufentanyl

- Tramadol

Vasoactive agents/alpha-2 agonist

- Clonidine
- Epinephrine
- Dexmedetomidine

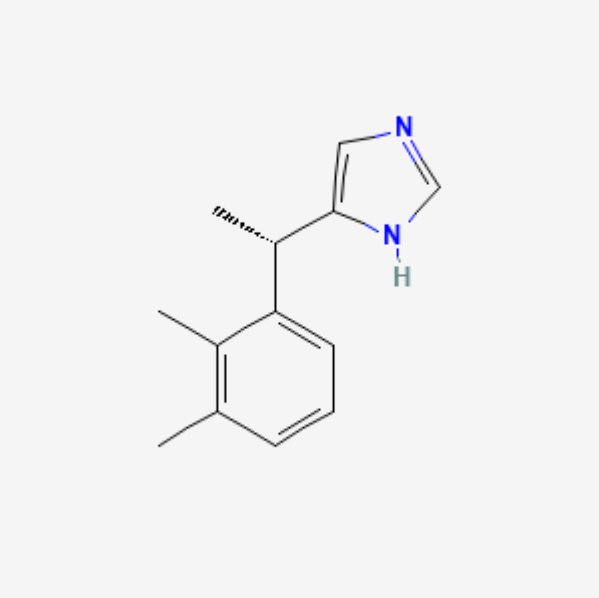
Anti-inflammatory agents/Steroids

- Dexamethasone
- Verapamil
- Ketorolac
- Methylprednisolone
- Adenosine

Other additives

- Ketamine
- Midazolam
- Neostigmine
- Magnesium
- Sodium bicarbonate”

PHARMACOLOGY OF DEXMEDETOMIDINE[46, 47]



α2-adrenergic receptor (α2-AR) agonists are commonly used in clinical settings for sedation, analgesia, anxiolysis, perioperative sympatholysis, cardiovascular stabilisation, reduced anaesthetic requirements, and preservation of respiratory function. The Food and Drug Administration (FDA) approved dexmedetomidine in 1999 for short-term sedation and analgesia in the critical care unit (ICU). Dexmedetomidine is an effective sedative medication with analgesic characteristics, haemodynamic stability, and the capacity to restore respiratory function in mechanically ventilated patients, allowing for early weaning. Aside from being a novel method of sedation and analgesia in ICU patient management, it has been examined in a variety of other perioperative situations.

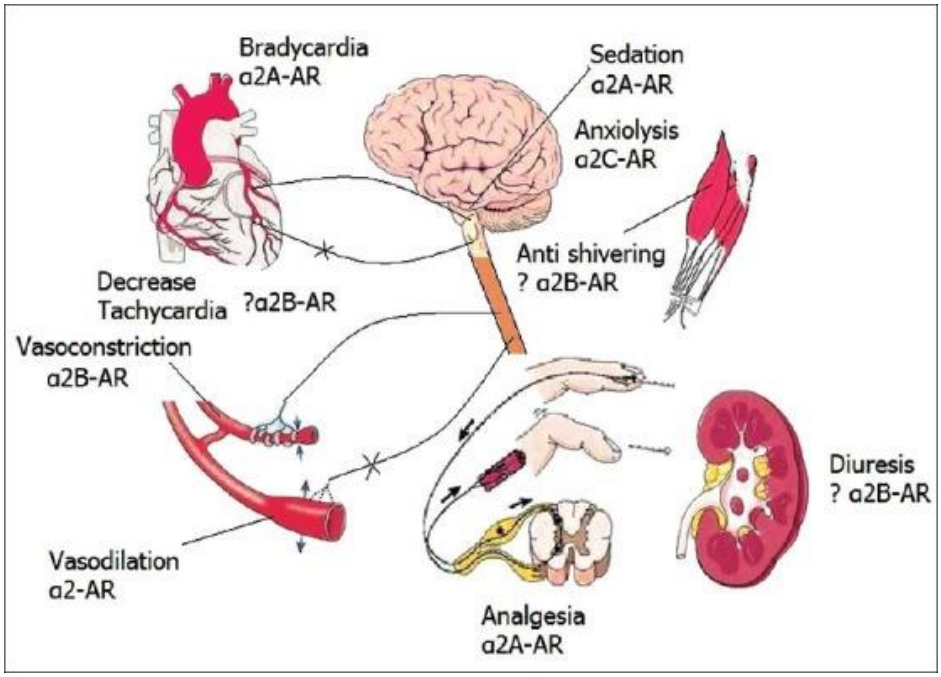
Chemical Structure:

Dexmedetomidine is the dextrorotatory S-enantiomer of medetomidine, which is utilised in veterinary medicine. Its chemical formula is (S)-4-[1-(2,3-dimethylphenyl)ethyl]-3H-imidazole.

Mechanism Of Action:

α2-AR agonists activate clinical effects by binding to G-Protein-coupled α2-ARs. There are three subtypes (α2A, α2B, and α2C), each with unique physiological functions and pharmacological properties. These receptor subtypes are present throughout the central, peripheral, and autonomic nervous systems, as well as essential organs and blood arteries. Dexmedetomidine is 8–10 times more selective for α2-AR than clonidine. Clonidine and dexmedetomidine are not completely selective for any of the α2-AR subtypes. However, dexmedetomidine appears to have stronger affinity for α2A-AR and α2C-AR than clonidine.
The sedative activity occurs in the locus coeruleus of the brain stem, whereas the analgesic action occurs in the spinal cord, both via α2A-AR pathways. α2-AR agonists reduce tachycardia and bradycardia in the heart by inhibiting the cardioaccelerator nerve and acting on the α2A-AR receptor, respectively. The peripheral vasculature experiences sympatholysis-mediated vasodilation and smooth muscle cell receptor-mediated vasoconstriction. The mechanism of anti-shivering and diuretic effect has yet to be fully identified."

Figure 4: Physiology of various α2-adrenergic receptors



Other responses to receptor activation include decreased salivation, decreased secretion, and decreased bowel motility in the gastrointestinal tract; contraction of vascular and other smooth muscle; inhibition of renin release, increased glomerular filtration, and increased sodium and water secretion in the kidney; decreased intraocular pressure; and decreased pancreatic insulin release. By combining these actions, dexmedetomidine avoids some of the negative effects of multiagent therapy.

Pharmacokinetics

Absorption and distribution:

Dexmedetomidine has linear pharmacokinetics in the recommended dose range of 0.2 to 0.7 μg/kg/hr, delivered as an intravenous infusion for up to 24 hours. The distribution phase is brief, with a half-life of around 6 minutes and an elimination half-life of 2 hours. The steady-state distribution volume is 118 L. The average protein binding is 94%, which is consistent across different plasma concentrations and

similar in males and females. Drugs such as fentanyl, ketorolac, theophylline, digoxin, and lidocaine have little effect on protein binding. Context-sensitive half life varies between 4 minutes after a 10-minute infusion to 250 minutes after an 8-hour infusion. Oral bioavailability is low due to substantial first-pass metabolism. However, sublingually given dexmedetomidine has a high bioavailability (84%), indicating a potential function in paediatric sedation and premedication.

Metabolism and excretion:

Dexmedetomidine is virtually completely biotransformed into inactive metabolites via direct N-glucuronidation and aliphatic hydroxylation by cytochrome P-450 (CYP 2A6). Metabolites are eliminated in urine (about 95%) and faeces (4%). Due to the slower rate of metabolism, patients with hepatic insufficiency require dose modifications.

Clinical Pharmacology

Cardiovascular system:

Dexmedetomidine causes a biphasic blood pressure response, consisting of a brief hypertension phase followed by hypotension. The two stages are mediated by two separate α2-AR subtypes: the α-2B AR is responsible for the first hypertensive phase, while the α2A-AR is responsible for hypotension. Bradycardia and sinus arrest have been reported in younger patients with high vagal tone, which were successfully treated with anticholinergic medications (atropine, glycopyrrolate).

Central nervous system:

Dexmedetomidine decreases cerebral blood flow and metabolic oxygen requirements, although its effect on intracranial pressure (ICP) is unclear. Dexmedetomidine

improves cognitive performance via modulating spatial working memory, while also acting as a sedative, analgesic, and anxiolytic via the α2-AR. Studies suggest that it may have neuroprotective effects by lowering circulation and brain catecholamine levels, so balancing the ratio of cerebral oxygen supply, reducing excitotoxicity, and enhancing perfusion in the ischaemic penumbra. It decreases the glutamate levels that cause cellular brain harm, particularly in subarachnoid haemorrhage. It has been proven to reduce the morphologic and functional effects of ischaemic (focal and global) and traumatic nervous system injuries.

Respiratory effects:

The effect of dexmedetomidine on breathing appears to be similar in magnitude to that found during heavy sleep. Dexmedetomidine does not impair respiratory function, even at high doses. It has no negative impact on respiratory rate or gas exchange in spontaneously breathing ICU patients after surgery. It aids in the maintenance of sedation without cardiovascular instability or respiratory drive depression, perhaps facilitating weaning and extubation in trauma/surgical ICU patients who have failed earlier weaning attempts due to agitation and hyperdynamic cardiopulmonary responses.

Endocrine and renal effects

Dexmedetomidine increases peripheral presynaptic α2-AR, reducing catecholamine release and sympathetic response during surgery.

Adverse Effects:

The following side effects have been reported: hypotension, hypertension, nausea, vomiting, dry mouth, bradycardia, atrial fibrillation, pyrexia, chills, pleural effusion,

atelectasis, pulmonary oedema, hyperglycemia, hypocalcaemia, acidosis, etc. Rapid dexmedetomidine infusion (loading dose of 1 μ/kg/hr if given in less than 10 minutes) can cause temporary hypertension due to peripheral α2B-AR vasoconstriction. However, continued therapy mediated by central α2A-AR may cause hypotension and bradycardia due to reduced noradrenaline release from the sympathetic nervous system. Long-term dexmedetomidine use causes receptor hypersensitization and upregulation; therefore, rapid termination can result in a withdrawal syndrome of uneasiness, agitation, headaches, and hypertensive crisis. Dexmedetomidine is not indicated for patients suffering from advanced heart block or ventricular dysfunction. The FDA has classed it as a category C pregnancy risk, therefore pregnant women should use extreme caution when using the medicine.

Clinical Applications Of Dexmedetomidine

Premedication

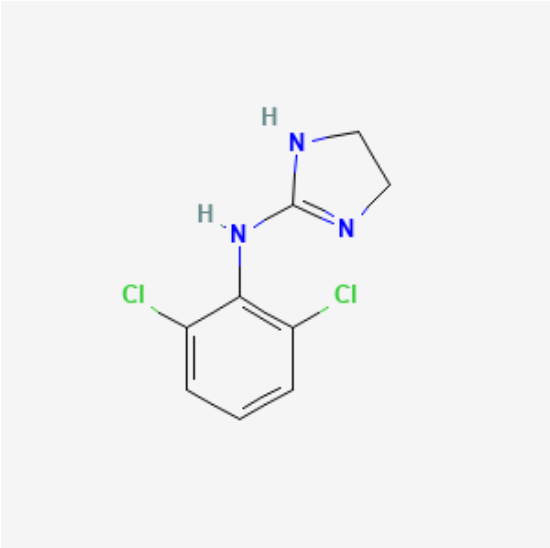
Dexmedetomidine is utilised as a premedication adjuvant, particularly in individuals who are prone to preoperative and perioperative stress, because to its sedative, anxiolytic, analgesic, sympatholytic, and stable haemodynamic profiles. Dexmedetomidine reduces oxygen use during and after surgery by up to 8% and 17%, respectively. The premedication dose ranges from 0.33 to 0.67 mg/kg IV, administered 15 minutes before operation.

- **Locoregional analgesia**

Highly lipophilic nature of dexmedetomidine allows rapid absorption into the cerebrospinal fluid and binding to α2-AR of spinal cord for its analgesic action. It prolongs the duration of both sensory and motor blockade induced by local anaesthetics irrespective of the route of administration (e.g., epidural, caudal, or spinal). Dexmedetomidine though enhances both central and peripheral neural blockade by local anaesthetics; however, the peripheral neural blockade is due to its binding to α2A-AR. Dexmedetomidine has been successfully used in intravenous regional anaesthesia (IVRA), brachial plexus block, and intraarticularly.

- Sedation in intensive care unit
- Procedural sedation
- Controlled hypotension
- Analgesia
- Cardiac surgery
- Neurosurgery"

PHARMACOLOGY OF CLONIDINE[48]



"Clonidine is an antihypertensive drug that targets alpha-adrenergic and imidazoline receptor agonists. Clonidine is an antihypertensive medication that lowers blood pressure and heart rate by relaxing the arteries and increasing blood supply to the heart. It also has other FDA-approved indications, including the treatment of attention deficit hyperactivity disorder (ADHD) in children (FDA approval 2010), the management of tics associated with Tourette syndrome, and adjunct therapy for severe cancer-related pain.

Indications:

"Clonidine is a 40-year-old antihypertensive medication that acts as an agonist on alpha-adrenergic and imidazoline receptors. Clonidine is an antihypertensive drug that lowers blood pressure and heart rate by relaxing the arteries and increasing the blood supply to the heart; it has the following FDA-approved indications:

- Hypertension, as mentioned above; pediatric use for hypertension is off-label
- Treatment of attention deficit hyperactivity disorder (ADHD) in children (FDA approval 2010 for the extended-release dose form)
- Management of tics commonly found with Tourette syndrome
- Adjunct therapy for severing cancer-related pain
- As an adjunct in neonatal opioid withdrawal syndrome"

Clonidine has a variety of off-label applications, including controlling opioid, benzodiazepine, and alcohol withdrawal symptoms, as well as treating anxiety, insomnia, and post-traumatic stress disorder (PTSD).

Clonidine has been employed in many different areas of medicine due to its influence on the sympathetic nervous system, notably the lowering of circulating epinephrine, such as the control of hot flashes in menopause, restless leg syndrome, and the

prevention of vascular migraine headaches. There is also a test for phaeochromocytoma called the clonidine suppression test; in the lab, they evaluate catecholamine levels before and after a dosage of oral clonidine, which, in healthy persons, should cause a fall in catecholamine levels in the blood.

Mechanism of Action:

Clonidine hydrochloride is an imidazoline derivative that acts as a central agonist of alpha-2 adrenergic receptors. Clonidine's chemical name is 2-((2,6-dichlorophenyl) amino)-2-imidazoline hydrochloride.

Clonidine acts as an alpha-adrenergic agonist in the nucleus tractus solitarii (NTS), inhibiting excitatory cardiovascular neurones. Clonidine has an alpha-antagonist action on the posterior hypothalamus and medulla. The ultimate response is reduced sympathetic outflow from the central nervous system (CNS), resulting in a clinically significant reduction in arterial blood pressure.

One theory about clonidine's mechanism of action in the treatment of pain in the CNS is that many pain signals originate in the dorsal horn of the spinal cord and are sent to higher centres of the CNS. The descending inhibitory bulbospinal neurones release norepinephrine, which binds to alpha-2-receptors in the dorsal horn, decreasing afferent pain transmission and producing analgesia. As a result, medications targeting alpha-2 receptors, such as clonidine, can alter pain transmission.

Epidural clonidine, when used in conjunction with local anaesthetics, has three distinct modes of action. First, stimulating alpha-2 receptors in the dorsal horn inhibits pain transmission. Second, clonidine can promote local vasoconstriction, which reduces the vascular clearance of local epidural anaesthetics. Finally, clonidine boosts neuraxial opioids and, when combined with fentanyl, interacts in an additive manner,

reducing the dose of each component by 60% for postoperative pain relief. The precise mechanism of action of clonidine in the treatment of attention-deficit hyperactivity disorder (ADHD) is unknown, but it is possible that prefrontal cortex brain activity is involved.

Clonidine has a half-life of 6–20 hours (17–40 hours in situations of renal impairment).

Injectable Solution:

- "Dosage: 100 mcg/ml, 500 mcg/ml
- Indications: epidural infusion form in cancer pain not controlled by opioid analgesics and as an adjunct in anaesthesia
- The initial dose of 30 mcg/hr and titration is necessary for pain management or potential side effects."

Adverse Effects:

"Like any other medication, clonidine has the potential for short-term and long-term side effects. Some of the common side effects based on FDA reports include:

Common Reactions (tend to resolve with continued therapy):

- Abdominal pain
- Headache
- Hypotension
- Fatigue
- Nausea
- Emotional instability

- Constipation
- Xerostomia
- Diarrhea
- Sexual dysfunction
- Dizziness
- Sedation

Serious Reactions:

- Angioedema
- Depression
- Hypersensitivity
- Atrioventricular (AV) block
- Bradycardia
- Syncope
- Severe hypotension

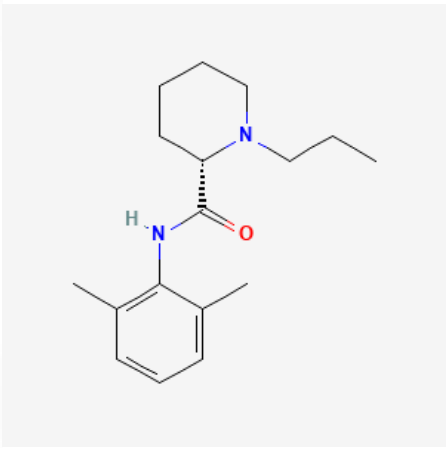
Note rebound hypertension and withdrawal symptoms if the medication is discontinued abruptly.

Other Symptoms:

- Fever, headache
- Fatigue
- Bradycardia

- Congestive heart failure
- Decreased sexual activity
- Thrombocytopenia
- Agitation
- Depression is one of the rarely reported side effects that can occur with the chronic use of clonidine; however, because of the variety of applications for this medication and also because of its slowly progressive onset, clinicians should monitor patients for signs of depression."

PHARMACOLOGY OF ROPIVACAINE[49]



Indications:

Ropivacaine is FDA-approved for use in surgical anaesthesia and acute pain treatment. It is used as an epidural block during surgery, including as caesarean sections. It is also utilised for large nerve blocks and local infiltration. Ropivacaine is used in epidurals (continuous infusion or intermittent bolus) for postoperative or labour pain relief. Researchers have recently examined the use of ropivacaine in the therapy of chronic pain. Clinical research suggest that with epidural administration

during caesarean delivery, ropivacaine (0.75% or 0.5%) has a clinically equivalent start of sensory and motor block to bupivacaine 0.5%. The median length of analgesia provided by ropivacaine to the T6-S3 dermatomes is comparable to that of bupivacaine, however the duration of motor block is significantly shorter (0.9 hrs vs. 2.5 hrs). For intrathecal administration, hyperbaric ropivacaine solutions are faster to onset and recover from than isobaric ropivacaine, however the spread and duration of the hyperbaric ropivacaine block are very varied. Co-administration with opioids decreases the amount of local anaesthetic required and extends analgesia without increasing the duration of the motor block.

An outpatient research comparing bupivacaine with ropivacaine in the treatment of persistent low back pain reported no significant difference in analgesia, motor blockage, or haemodynamic alterations between bupivacaine 0.125% and ropivacaine 0.2%. Ropivacaine was also found to be beneficial in treating severe refractory migraines by inactivating trigger points.

Mechanism of Action:

Ropivacaine is a long-acting, amide local anaesthetic. It works similarly to other local anaesthetics by reversibly inhibiting sodium ion inflow in nerve fibres. Amides preferentially bind to and inactivate sodium channels while they are open, preventing action potential propagation. The dose-dependent blockage of potassium channels enhances this effect. Ropivacaine has several distinguishing characteristics. Ropivacaine is less lipophilic than other local anaesthetics, including bupivacaine, and thus less likely to enter big myelinated motor fibres. It thus preferentially works on the nociceptive A, B, and C fibres over the AB (motor) fibres. Ropivacaine is also

available as a pure S(-) enantiomer, which has much lower cardiotoxicity and neurotoxicity.

Administration:

Ropivacaine is given in progressive doses. The area to be anaesthetised, tissue vascularity, the number of neuronal segments to be blocked, and the depth and duration of anaesthesia required all influence how much local anaesthetic is supplied. Before inducing the full block, administer a sufficient test dosage of 3 to 5 mL of a short-acting local anaesthetic with epinephrine. If the patient is moved in a way that causes the epidural catheter to be displaced, the test dosage should be repeated. When doing major nerve blocks, use 35 to 50 mL of 0.5% solution and 10 to 40 mL of 0.75 % solution. For field blocks (e.g., minor nerve blocks and infiltration), ropivacaine 0.5% solution is administered in doses ranging from 1 to 40 mL.

When dealing with post-operative pain, peripheral nerve blocks are continually injected at a rate of 5 to 10 mL/hr of 0.2% solution. For pain relief via lumbar or thoracic epidurals, the continuous infusion dose of ropivacaine is 6 to 14 mL/hr of 0.2% solution.

Long-term blocks should take into account the dangers of achieving hazardous plasma concentrations or triggering local brain damage. A 24-hour cumulative dose of up to 770 mg ropivacaine is generally well tolerated in adults for postoperative pain relief. Ropivacaine should be administered with caution to debilitated individuals for more than 70 hours.

Adverse Effects:

Ropivacaine is generally well-tolerated. A pooled analysis of controlled clinical studies (n=1,661) in which patients received ropivacaine concentrations ranging from

0.125 to 1% for nerve blocks revealed that the most common adverse events were hypotension (32%), nausea (17%), vomiting (7%), bradycardia (6%), and headache (7%). Patients who received bupivacaine concentrations ranging from 0.25 to 0.75% had a comparable side effect profile. When used correctly, ropivacaine causes few adverse consequences.

Patients aged 61 and over who received epidural ropivacaine 1% had a higher prevalence of bradycardia than patients aged 41 to 60 years (58% vs. 15%); they also had a higher incidence of hypotension than patients aged 18 to 40 years (74% vs. 20%). The general incidence of adverse events in children aged one to fifteen months is modest; the most common side effects of ropivacaine in this age range were nausea and/or vomiting.

The most prevalent foetal or neonatal adverse effects associated with ropivacaine use in women undergoing caesarean section or labour were foetal bradycardia (12%), newborn jaundice (8%), and unexplained neonatal problems (7%).

REVIEW OF RELATED ARTICLES

Abhishek M. S et al (2024)[50] We examined the onset, duration, and analgesia of 0.5% ropivacaine with clonidine 1mg.kg-1 and 0.5% ropivacaine with dexmedetomidine 1mg.kg-1. In Group A, 73.3% of the patients experienced an 8-minute sensory block, while 26.7% experienced it after 10 minutes. In Group B, 44.4% of patients experienced an 8-minute sensory block, whereas 26.7% experienced a 10-minute sensory block. Statistically, participants in Group A had a lower start of block and a longer mean duration of sensory and motor block than Group B. They concluded that adding dexmedetomidine to 0.5% ropivacaine in

supraclavicular brachial plexus block reduced the timing of onset of sensory and motor block while increasing the duration of analgesia.

Mandal S et al (2022)[51] Compare the efficacy of clonidine and dexmedetomidine as adjuvants to ropivacaine during SCBP block for upper limb procedures in terms of postoperative analgesic duration. The dexmedetomidine group experienced significantly longer post-operative analgesia compared to the clonidine and ropivacaine alone groups (664.13 vs. 551.77 vs. 465.47, respectively, $P < 0.001$). The duration of sensory and motor block was significantly longer in the dexmedetomidine group compared to the clonidine and control groups. Adverse effects were equivalent across all three groups. They determined that dexmedetomidine appeared to be a better adjuvant than clonidine in terms of sustained postoperative analgesia and similar side effects.

Cai H et al (2021)[52] The goal of this meta-analysis was to find the best dose of perineural DEX for sustained analgesia after brachial plexus block (BPB) in adult patients having upper limb surgery. There were 57 RCTs found, with 3332 patients. Subgroup and regression analysis indicate that a perineural DEX dose of 30-50 μg is suitable. The average duration of analgesia with short-/intermediate-acting LAs was 220.31 (153.13-287.48) minutes for doses less than 60 μg and 68.01 (36.37-99.66) minutes for doses greater than 60 μg, according to a 95% confidence interval. With long-acting LAs, the mean differences (95% CI) with less than and more than 60 μg dosages were 332.45 (288.43-376.48) minutes and 284.85 (2220.31-349.39) minutes. They concluded that 30-50 μg DEX as adjuvant can provide a longer analgesic period compared to LA alone, while not increasing the risk of bradycardia and hypotension.

Sane S et al (2021)[53] In upper extremity orthopaedic surgery, the effect of dexmedetomidine with bupivacaine combination versus bupivacaine alone on sensory and motor block duration time, pain score, and haemodynamic changes in the supraclavicular block was investigated. The onset time of sensory and motor block in patients receiving only bupivacaine was 31.03 ± 9.65 min and 24.66 ± 9.2 min, respectively, while in the dexmedetomidine group, it was around 21.36 ± 8.34 min and 15.93 ± 6.36 minutes. The increases in heart rate and mean arterial blood pressure were comparable in both groups. The intervention group had a longer period of sensory and motor block, as well as the time of the initial analgesic request. The intervention group experienced less postoperative discomfort for 24 hours (P = 0.001). They concluded that dexmedetomidine and bupivacaine reduced the onset time of sensory and motor blocks while increasing numbness and immobility length. Dexmedetomidine was also found to considerably minimise postoperative pain when combined with bupivacaine for supraclavicular blocks.

Kumari P et al (2021)[54] The efficacy of postoperative analgesia was tested between clonidine and dexmedetomidine as adjuvants with 0.5% ropivacaine using ultrasound-guided supraclavicular brachial plexus block for upper limb procedures. In a comparative study, dexmedetomidine 1 μg.kg−1 with 35 ml of 0.5% ropivacaine resulted in significantly longer postoperative analgesia and earlier sensory block than clonidine 1 μg.kg−1 with the same dose of ropivacaine in ultrasound-guided supraclavicular brachial plexus block. They found that the ropivacaine-dexmedetomidine group in our study achieved faster sensory block and had longer postoperative analgesia than the ropivacaine-clonidine group. Thus, ropivacaine-dexmedetomidine can be utilised effectively in all painful upper limb surgeries, particularly orthopaedic procedures.

Chinnappa J et al (2017)[55] Dexmedetomidine was added to patients undergoing upper limb procedures under supraclavicular brachial plexus block to see how it affected the duration of analgesia. The onset of sensory and motor block in Group A (13.0 ± 4.1 and 23.5 ± 5.6 min) was slower than in Group B (9.5 ± 5.8 and 15.6 ± 6.3 min; P = 0.009 for sensory and P < 0.001 for motor block). Group A had shorter sensory and motor block durations (400.8 ± 86.6 and 346.9 ± 76.9 min) compared to Group B (630.6 ± 208.2 and 545.9 ± 224.0 min, respectively; P < 0.001). Group A experienced a shorter duration of analgesia (411.0 ± 91.2 min) compared to Group B (805.7 ± 205.9 min; P < 0.001). Group B had a significantly higher incidence of bradycardia and hypotension than Group A (P < 0.001). They concluded that combining perineural dexmedetomidine with ropivacaine prolongs postoperative analgesia, accelerates the onset of sensory and motor block, and extends the duration of supraclavicular brachial plexus blocks.

Rustagi, P et al (2017)[56] Determine whether adding clonidine to the local anaesthetic solution for brachial plexus block via supraclavicular route prolongs sensory and motor blockade and improves postoperative analgesia. Also, keep an eye out for any negative effects. The addition of clonidine had no influence on the beginning of the block, but it was observed to prolong the sensory and motor block as well as the duration of analgesia. The group that received clonidine had a considerably lower visual analogue pain score. The study found that adding Clonidine 2 µg/kg to 10 ml of 0.5% Bupivacaine + 20 ml of 2% lignocaine with adrenaline (1:200000) improved the quality and duration of supraclavicular brachial plexus block.

Kirubahar, R et al (2016)[57] Dexmedetomidine and clonidine were evaluated as adjuvants to bupivacaine for supraclavicular brachial plexus block in upper limb

orthopaedic procedures. Group D had a shorter mean time of start for sensory and motor block than Group C. Except for the fifth minute, Group D's pulse rate and mean arterial pressure were lower than Group C. The mean duration of sensory and motor block was longer in Group D than in Group C. The duration of analgesia was longer in Group D than in Group C. They observed that adding dexmedetomidine to bupivacaine during supraclavicular brachial plexus block results in a shorter onset of sensory and motor block with a longer duration of analgesia than adding clonidine to bupivacaine.

Kulkarni, S. B et al (2016)[58] The impact of combining clonidine and ropivacaine on supraclavicular brachial plexus block was investigated. Sixty patients were randomly assigned to two groups: Group C and Group R. Group C received 0.5% ropivacaine with 1 ml normal saline, while Group R received the same amount of ropivacaine with 1 ml (100μg) of clonidine for supraclavicular brachial plexus block. Group C showed significantly longer durations of sensory and motor block, as well as analgesia, than Group R ($P<0.001$). There was no significant difference in the mean onset time of sensory and motor blockade, haemodynamic parameters (pulse rate, diastolic and systolic blood pressure) during and after surgery, or postoperative sedation score between the two groups ($P>0.05$). The study found that combining Clonidine 100μg with 0.5% ropivacaine for supraclavicular brachial plexus block resulted in prolonged sensory and motor blockade, improved analgesia, and reduced the need for systemic analgesics without affecting haemodynamics.

Patil KN et al (2015)[59] Clonidine was tested for its effect on the features of ropivacaine-induced supraclavicular brachial plexus block. Sensorimotor block onset occurred earlier in Group II (4.36 ± 0.81 min for sensory block and 9.83 ± 1.12 min for motor block) compared to Group I (4.84 ± 0.65 min for sensory block and 10.85 ±

0.79 min for motor block). Clonidine significantly lengthened both sensory and motor block durations ($P < 0.001$). Clonidine treatment led to a longer duration of analgesia (613.10 ± 51.797 vs. 878.33 ± 89.955 minutes). Although Group II had a higher frequency of hypotension and bradycardia than Group I, the difference was not clinically significant. They determined that Ropivacaine 0.75% is well tolerated and effective for both surgical anaesthesia and postoperative pain management. Clonidine as an adjuvant to ropivacaine improves the quality of supraclavicular brachial plexus block by speeding up the onset, prolonging the duration of sensory and motor block, and improving postoperative analgesia, all without causing any side effects at the recommended dose.

Swami SS et al (2012)[60] Clonidine and dexmedetomidine were compared as adjuvants to local anaesthetic agents in supraclavicular brachial plexus block in terms of sensory and motor block onset and duration, as well as analgesia duration. In group C, sensory and motor block durations were 227.00±48.36 and 292.67±59.13 min, respectively, but in group D, they were 413.97±87.13 and 472.24±90.06 min, respectively. There was no significant difference in the onset of sensory or motor block between the two groups. In group D, analgesia lasted 456±97 minutes, while in group C, it took 289±62 minutes. Statistically, this difference was significant ($P=0.001$). In group D, 80% of patients achieved grade IV quality (excellent) of block, compared to 40% in group C ($P<0.05$). They concluded that adding dexmedetomidine to local anaesthetic in supraclavicular brachial plexus block increased the duration of sensory and motor block, as well as analgesia. Patients who received dexmedetomidine had a longer time to achieve rescue analgesia. It also improved the quality of the block as compared to clonidine."

MATERIAL AND METHODS

MATERIAL AND METHODS

This study was a randomized control trial conducted at R.L. Jalappa Hospital and Research Centre, Tamaka, Kolar, from May 2023 to October 2024. After obtaining approval from the institutional ethics committee, 106 patients scheduled for elective or emergency upper limb surgeries were recruited for the study.

Study Design and Setting

A randomized controlled trial was conducted in the Department of Anaesthesiology at Sri Devaraj Urs Medical College, Kolar. The study was registered with the Clinical Trials Registry of India after obtaining ethical clearance from the institutional ethics committee.

Sample Size Calculation

The sample size was calculated based on a previous study by Sangita Mandal et al.[51] (2022), which reported a mean duration of postoperative analgesia of 664.13 minutes in the dexmedetomidine group and 551.7 minutes in the clonidine group, with standard deviations of 11.64 and 19.11, respectively. Assuming a minimum clinically important difference of 10 minutes in the duration of analgesia, with a 5% level of significance ($\alpha = 0.05$) and 90% power ($1-\beta = 0.90$), the minimum sample size required per group was calculated as 53 patients. Thus, a total of 106 patients (53 per group) were included in the study.

The formula used for sample size calculation was:

$$\mathbf{n = \{2(Z_{1-\alpha/2} + Z_{1-\beta})^2 \times \sigma^2\}/d^2}$$

Where:

- $Z_{1-\alpha/2} = 1.96$ at 5% level of significance

- $Z_{1-}\beta = 1.28$ at 90% power
- σ^2 = variance (standard deviation squared)
- d = minimum clinically important difference (10 minutes)

Inclusion Criteria

1. Patients aged 18 to 60 years of either gender
2. American Society of Anaesthesiologists (ASA) physical status grades I-II
3. Patients scheduled for upper limb surgeries
4. Patients not suffering from cardiovascular, neurological, renal, or coagulation disorders

Exclusion Criteria

1. Patients refusing to participate in the study
2. Patients with known allergies to the study drugs
3. Patients requiring surgeries in both upper limbs
4. Patients suffering from cardiovascular, neurological, renal, or coagulation disorders

Randomization and Allocation

Patients who met the inclusion criteria and provided written informed consent were randomly allocated into two groups using computer-generated random numbers. The allocation sequence was concealed using sequentially numbered, opaque, sealed envelopes. The envelopes were opened just before the administration of the block.

Group A (n=53): Patients received 0.5% ropivacaine 30mL along with clonidine 1mcg/kg diluted with normal saline, reconstituted to a total volume of 32mL.

Group B (n=53): Patients received 0.5% ropivacaine 30mL along with

dexmedetomidine 1mcg/kg diluted with normal saline, reconstituted to a total volume of 32mL.

Blinding

The study was conducted as a double-blind trial. The anaesthesiologist who prepared the study drug was not involved in the administration of the block or the assessment of outcomes. The patients and the anaesthesiologist who administered the block and assessed the outcomes were blinded to the group allocation.

Pre-operative Preparation

A detailed pre-anaesthetic evaluation was performed for all patients a day before surgery. Basic demographic data, including age, sex, weight, and height, were recorded. A thorough history was taken, and physical examination was conducted. Routine investigations including complete blood count, renal function tests, coagulation profile, and chest X-ray were performed. The Visual Analog Scale (VAS) for pain assessment was explained to all patients.

Patients were kept nil by mouth for 8 hours for solid food and 2 hours for clear fluids before the procedure. Written informed consent was obtained from all patients or their legal representatives after explaining the procedure in detail in a language they understood.

Procedure

On arrival in the operation theatre, standard monitoring including non-invasive blood pressure (NIBP), electrocardiogram (ECG), and pulse oximetry (SpO_2) was established. Baseline vital parameters including heart rate, blood pressure, and oxygen saturation were recorded. Intravenous access was secured with an 18G cannula, and

intravenous fluid (Ringer's lactate) was started at a rate of 10 ml/kg/hr.

The patients were positioned supine with the arm kept on the side of the body and extended along the side, with the head turned away from the side to be blocked. The ultrasound machine was set up, and the linear probe was covered with a sterile cover to prevent infection spread. Sterile gel was applied to provide better imaging.

The supraclavicular area was prepared with antiseptic solution and draped with sterile towels. The anaesthesiologist performing the block wore sterile gloves. A high-frequency linear ultrasound probe (8-13 MHz) was placed in the supraclavicular fossa in a coronal oblique plane to visualize the subclavian artery and the brachial plexus. The brachial plexus was identified as a cluster of hypoechoic structures lateral and superficial to the subclavian artery.

Under ultrasound guidance, using an in-plane approach, a 22G, 50mm needle was inserted from the lateral side and directed towards the brachial plexus. After confirming the needle position and negative aspiration for blood, the local anaesthetic mixture was injected slowly with intermittent aspiration. The spread of the local anaesthetic around the brachial plexus was visualized on the ultrasound image.

As per the randomization, patients in Group A received 0.5% ropivacaine 30mL along with clonidine 1mcg/kg, while patients in Group B received 0.5% ropivacaine 30mL along with dexmedetomidine 1mcg/kg. In both groups, the drugs were diluted with normal saline to make a total volume of 32mL.

Assessment Parameters

The following parameters were assessed:

1. **Sensory Block**:

- Onset time: The time from the end of local anaesthetic injection to the absence of sensation to pinprick in all the dermatomes (C5-T1).
- Duration: The time from the onset of sensory block to the return of sensation to pinprick in any of the dermatomes (C5-T1).

2. **Motor Block**:

- Onset time: The time from the end of local anaesthetic injection to the development of grade 2 motor block according to the modified Bromage scale.
- Duration: The time from the onset of motor block to the return of motor movement (modified Bromage scale score 1).

3. **Hemodynamic Parameters**:

- Heart rate, blood pressure, mean arterial pressure, oxygen saturation, and respiratory rate were recorded at baseline and at 5, 15, 30, 60, 90, 120, and 150 minutes after the block.

4. **Duration of Analgesia**:

- The time from the onset of sensory block to the first request for rescue analgesia.
- VAS score at the time of first rescue analgesia.
- Numerical Pain Rating Scale (NRS) score.
- The number of times patients asked for rescue analgesia in the first 24 hours of the postoperative period.

5. **Sedation**:

- Assessed using the Ramsay Sedation Scale at 30-minute intervals for the first 6 hours after the block.

6. **Adverse Effects**:

- The incidence of adverse effects such as hypotension, bradycardia, respiratory depression, nausea, vomiting, and any other complications were recorded.

Definitions

1. **Sensory Block**:

- Assessed by pinprick test using a 23G needle in the distribution of the median, ulnar, radial, and musculocutaneous nerves. Sensory block was graded as:
- 0 = normal sensation
- 1 = decreased sensation to pinprick
- 2 = loss of sensation to pinprick

2. **Motor Block**:

- Assessed using the modified Bromage scale for upper limb:
  - 0 = normal motor function
  - 1 = decreased motor strength with ability to move the fingers only
  - 2 = complete motor block with inability to move the fingers

3. **Hemodynamic Parameters**:

- Hypotension: Decrease in systolic blood pressure by more than 20% from baseline or systolic blood pressure < 90 mmHg.
- Bradycardia: Heart rate < 50 beats per minute.
- Respiratory depression: Respiratory rate < 10 breaths per minute or SpO_2 < 90%.

4. **Ramsay Sedation Scale**:

- 1 = anxious, agitated, or restless
- 2 = cooperative, oriented, and tranquil
- 3 = responsive to commands only
- 4 = brisk response to light glabellar tap or loud auditory stimulus
- 5 = sluggish response to light glabellar tap or loud auditory stimulus
- 6 = no response to light glabellar tap or loud auditory stimulus

Postoperative Management:

All patients were monitored in the postoperative recovery room for the first 6 hours and then in the ward for the next 18 hours. Vital parameters were recorded at regular intervals. Pain was assessed using the VAS and NRS scales. Rescue analgesia in the form of intravenous diclofenac sodium 75 mg was administered when the VAS score was ≥ 4 or when the patient requested analgesia. The time of first rescue analgesia and the total number of rescue analgesic doses in the first 24 hours were recorded.

Any adverse effects such as hypotension, bradycardia, respiratory depression, nausea, vomiting, or any other complications were recorded and managed appropriately. Hypotension was treated with intravenous fluids and incremental doses of ephedrine if required. Bradycardia was treated with intravenous atropine 0.6 mg.

STATISTICAL ANALYSIS:

The data were analysed using SPSS version 20.0 software. Continuous variables were presented as mean ± standard deviation (SD). Categorical variables were presented as frequency and percentage. The normality of data was tested using the Kolmogorov-Smirnov test.

For normally distributed continuous variables, the independent t-test was used for comparing the two groups. For non-normally distributed continuous variables, the Mann-Whitney U test was used. For categorical variables, the chi-square test or Fisher's exact test was used as appropriate.

The primary outcome measure was the duration of analgesia, defined as the time from the onset of sensory block to the first request for rescue analgesia. Secondary outcome measures included the onset times of sensory and motor blocks, durations of sensory and motor blocks, and adverse effects.

A p-value < 0.05 was considered statistically significant.

RESULTS

RESULTS

This is a study of ultrasound guided supraclavicular brachial plexus block using clonidine versus dexmedetomidine as adjuvants to ropivacaine for post-operative analgesia in upper limb surgeries conducted on ASA I and ASA II patients with upper limb injuries for elective and/or emergency surgeries at R.L.Jalappa Hospital and Research Centre Tamaka Kolar for a period of 18 months.

Table 1: Age Categories

Age Group (years)	**Group A (Clonidine) (n=53)**	**Group B (Dexmedetomidine) (n=53)**	**p-value**
18-20	2 (3.8%)	3 (5.7%)	0.19
21-40	27 (50.9%)	35 (66.03%)	
41-60	24 (45.3%)	15 (28.3%)	
Total	**53 (100%)**	**53 (100%)**	
Mean ± SD	38.25 ± 13.41	35.34 ± 11.40	0.232

This table shows the age distribution of patients in both groups. Group A (Clonidine) had 53 patients with 3.8% aged 18-20 years, 50.9% aged 21-40 years, and 45.3% aged 41-60 years. Group B (Dexmedetomidine) also had 53 patients with 5.7% aged 18-20 years, 66.03% aged 21-40 years, and 28.3% aged 41-60 years. The mean age was 38.25 ± 13.41 years for Group A and 35.34 ± 11.40 years for Group B. With a p-value

of 0.232 for the mean age comparison, there was no statistically significant difference between the two groups, indicating they were comparable in terms of age distribution.

Graph 1: Age Categories

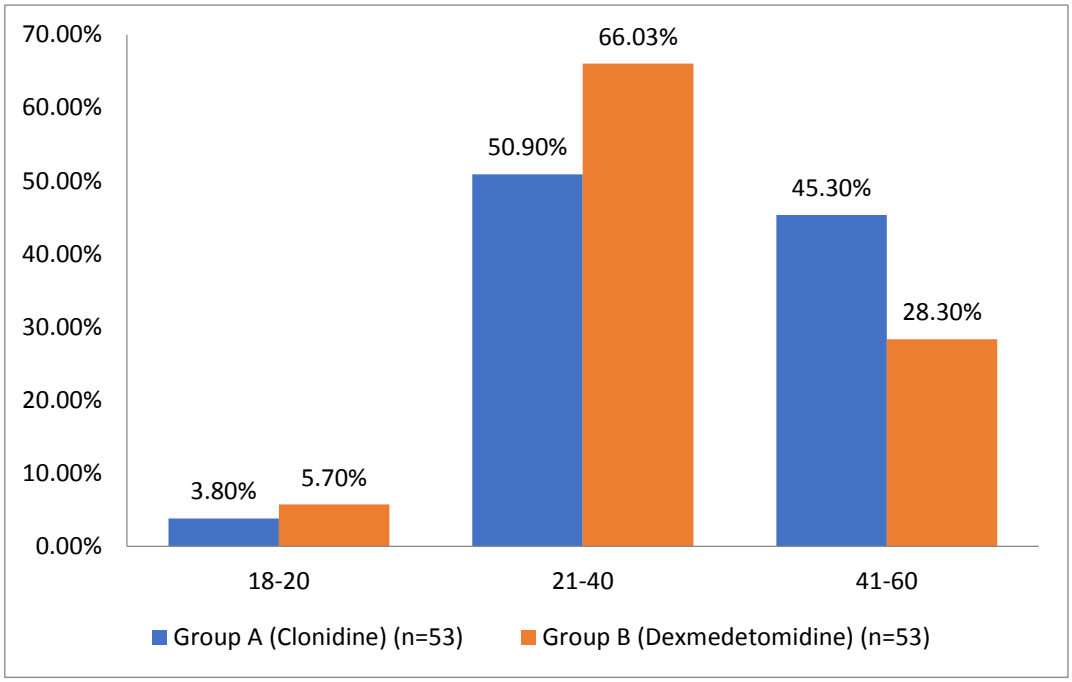


Table 2: Gender Distribution

Gender	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Male	33 (62.3%)	36 (67.9%)	0.68
Female	20 (37.7%)	17 (32.1%)	
Total	53 (100%)	53 (100%)	

This table presents the gender distribution across both groups. In Group A (Clonidine), 62.3% (33 patients) were male and 37.7% (20 patients) were female. In Group B (Dexmedetomidine), 67.9% (36 patients) were male and 32.1% (17 patients) were female. The p-value of 0.68 indicates no statistically significant difference in gender distribution between the two groups, suggesting both groups were comparable in terms of gender composition.

Graph 2: Gender Distribution

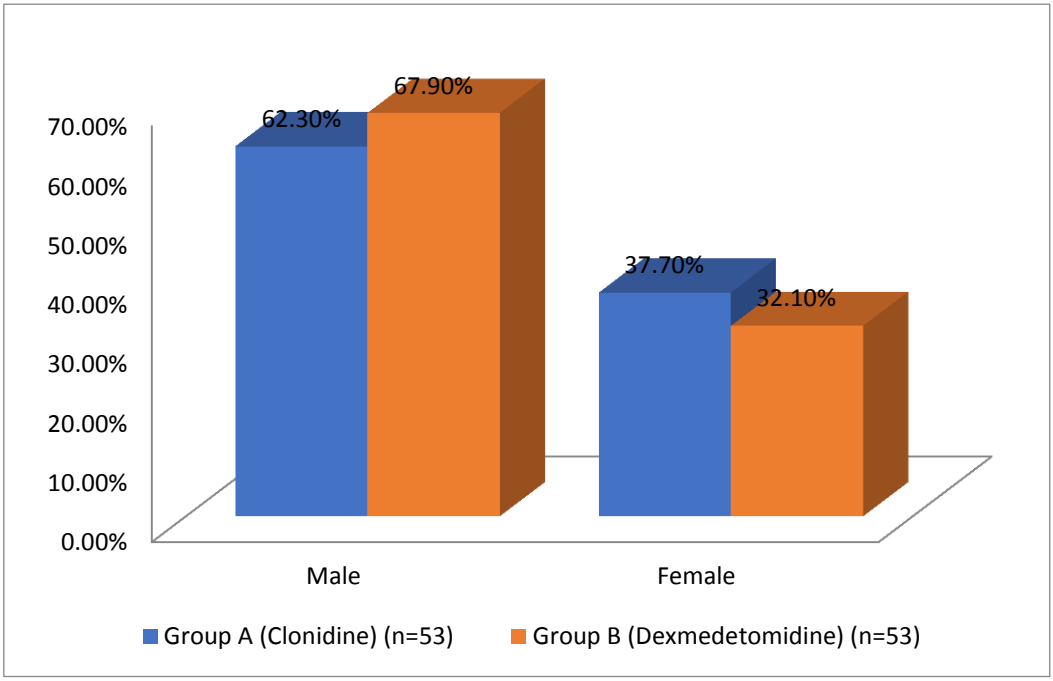


Table 3: BMI Distribution

BMI (kg/m²)	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Mean ± SD	24.27 ± 5.87	22.86 ± 5.98	0.226

This table shows the Body Mass Index (BMI) distribution between the two groups. The mean BMI for Group A (Clonidine) was 24.27 ± 5.87 kg/m², while for Group B (Dexmedetomidine) it was 22.86 ± 5.98 kg/m². With a p-value of 0.226, there was no statistically significant difference in BMI between the two groups, indicating that both groups were comparable in terms of BMI.

Graph 3: BMI Distribution

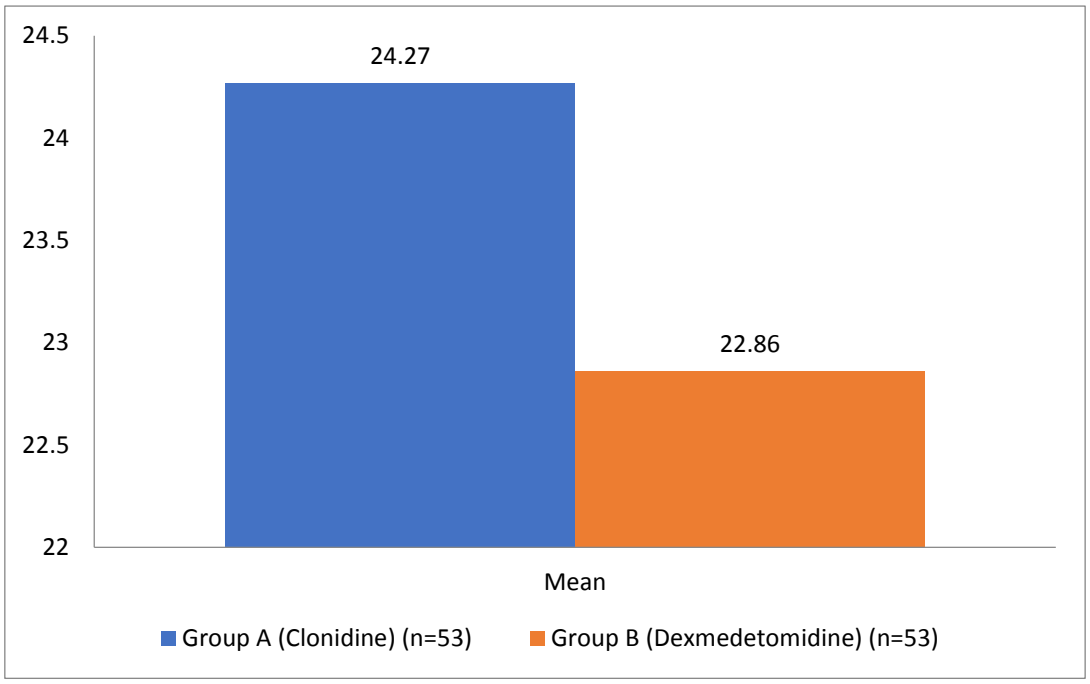


Table 4: ASA Grade Distribution

ASA Grade	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
I	40 (75.5%)	38 (71.7%)	0.826
II	13 (24.5%)	15 (28.3%)	
Total	**53 (100%)**	**53 (100%)**	

This table displays the American Society of Anaesthesiologists (ASA) physical status classification distribution. In Group A (Clonidine), 75.5% (40 patients) were ASA grade I and 24.5% (13 patients) were ASA grade II. In Group B (Dexmedetomidine), 71.7% (38 patients) were ASA grade I and 28.3% (15 patients) were ASA grade II. The p-value of 0.826 shows no statistically significant difference in ASA grade distribution between the groups, indicating they were comparable in terms of physical status.

Graph 4: ASA Grade Distribution

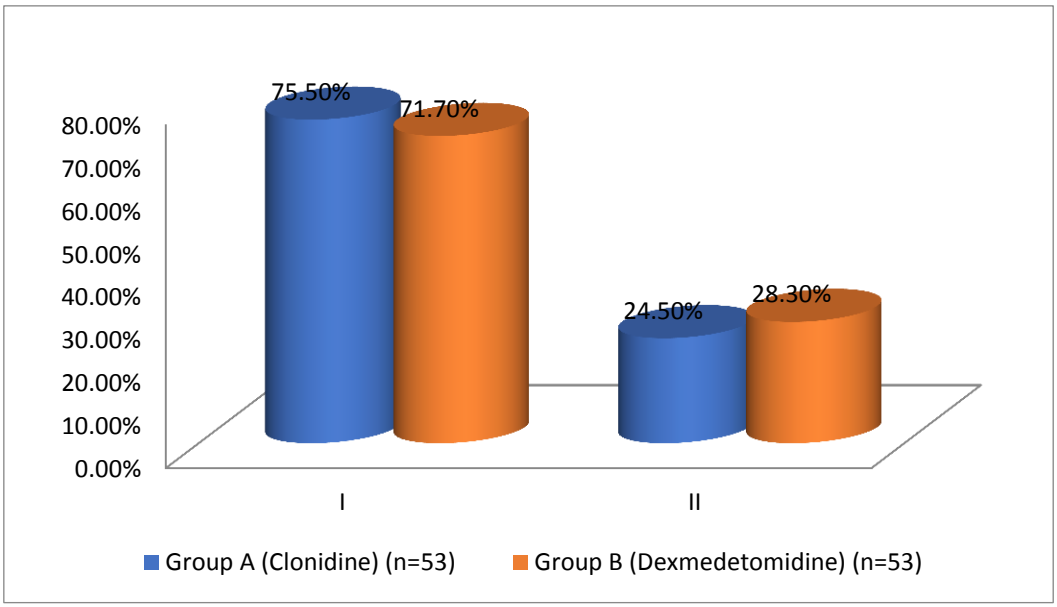


Table 5: Surgery Duration

Surgery Duration (min)	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Mean ± SD	86.42 ± 20.97	85.00 ± 22.77	0.740

This table presents the duration of surgery for both groups. The mean surgery duration for Group A (Clonidine) was 86.42 ± 20.97 minutes, while for Group B (Dexmedetomidine) it was 85.00 ± 22.77 minutes. The p-value of 0.740 indicates no statistically significant difference in surgery duration between the two groups, suggesting that both groups underwent comparable surgical times.

Graph 5: Surgery Duration

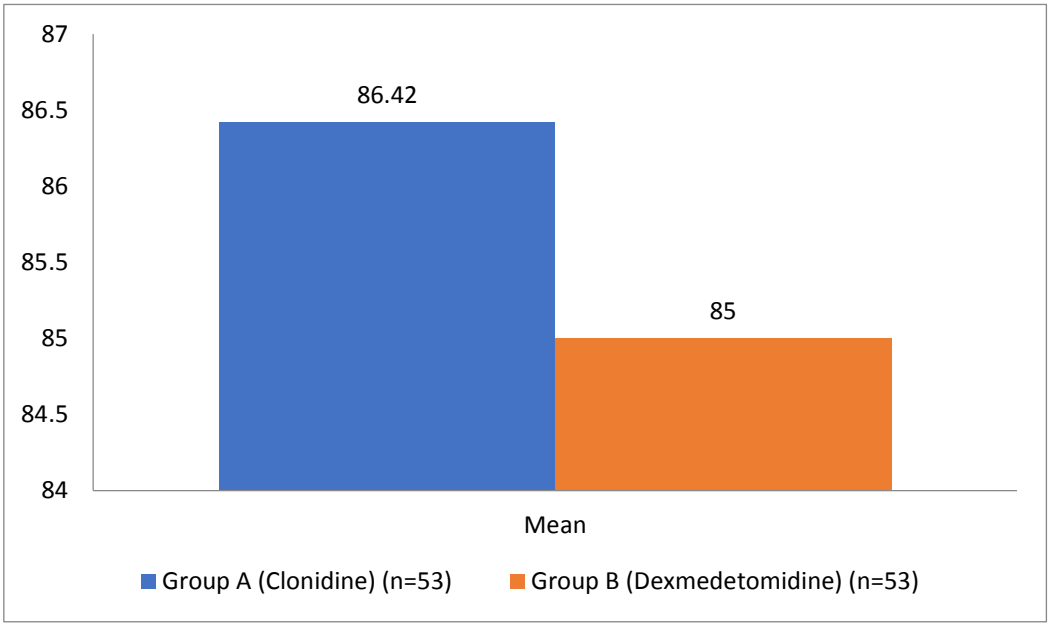


Table 6: Sensory Block characteristics

Parameters (Mean ± SD)	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Onset Time of Sensory Block (min)	9.28 ± 1.80	6.95 ± 1.47	**<0.001***
Duration of Sensory Block	551.81 ± 43.93	668.26 ± 41.65	**<0.001***

*Statistically significant

This table shows two important parameters related to sensory block. The onset time of sensory block was 9.28 ± 1.80 minutes for Group A (Clonidine) and 6.95 ± 1.47 minutes for Group B (Dexmedetomidine). The duration of sensory block was 551.81 ± 43.93 minutes for Group A and 668.26 ± 41.65 minutes for Group B. The p-value of <0.001 for both parameters indicates statistically significant differences. Group B (Dexmedetomidine) had a faster onset of sensory block and a longer duration of sensory block compared to Group A (Clonidine).

Graph 6: Sensory Block characteristics

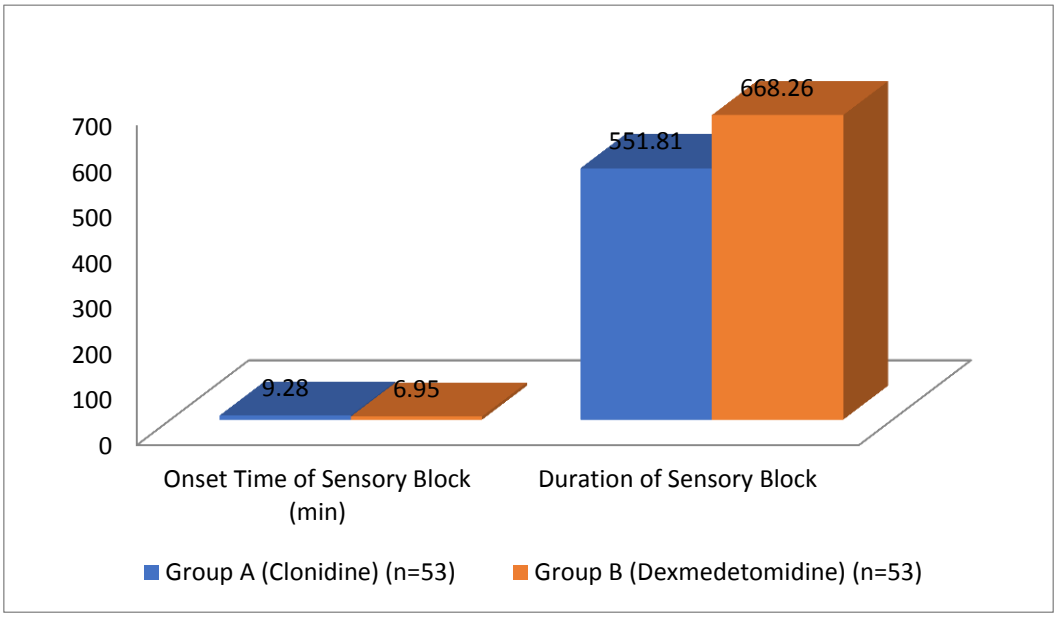


Table 7: Motor Block characteristics

Parameters	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Onset Time of Motor Block (min)	12.08 ± 2.40	8.70 ± 2.00	**<0.001***
Duration of Motor Block (min)	497.94 ± 46.61	595.06 ± 42.37	**<0.001***

*Statistically significant

This table presents motor block characteristics across both groups. The onset time of motor block was 12.08 ± 2.40 minutes for Group A (Clonidine) and 8.70 ± 2.00 minutes for Group B (Dexmedetomidine). The duration of motor block was 497.94 ± 46.61 minutes for Group A and 595.06 ± 42.37 minutes for Group B. The p-value of <0.001 for both parameters indicates statistically significant differences. Group B (Dexmedetomidine) demonstrated a faster onset of motor block and a longer duration of motor block compared to Group A (Clonidine).

Graph 7: Motor Block characteristics

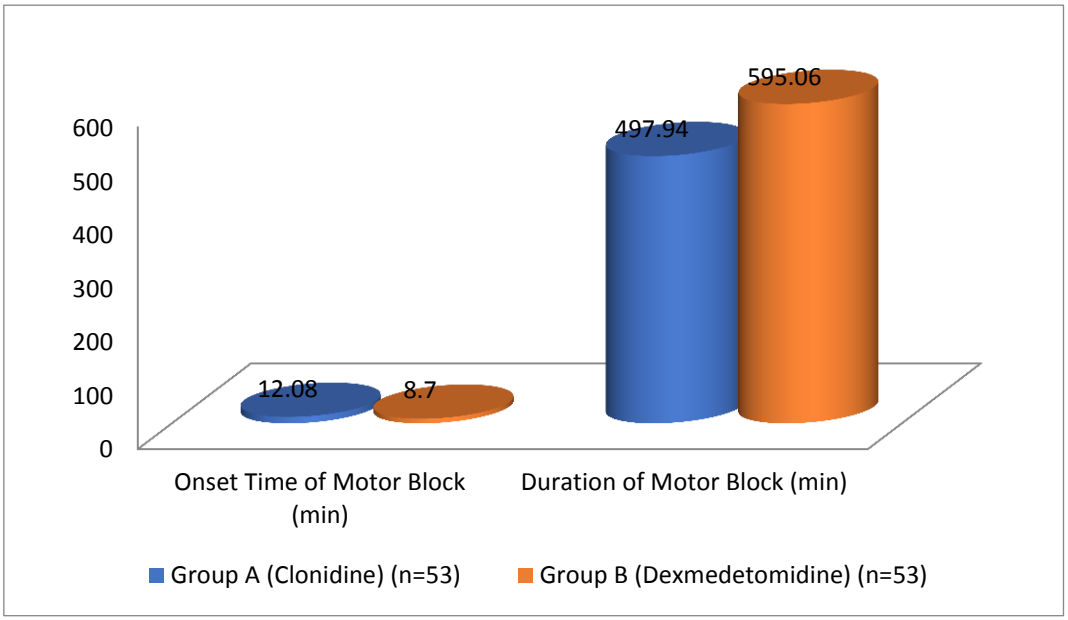


Table 8: Time to First Rescue Analgesia

Time to First Rescue Analgesia (min)	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Mean ± SD	579.15 ± 57.30	716.45 ± 76.88	**<0.001***

*Statistically significant

This table shows when patients first required rescue analgesia after surgery. The mean time to first rescue analgesia was 579.15 ± 57.30 minutes for Group A (Clonidine) and 716.45 ± 76.88 minutes for Group B (Dexmedetomidine). The p-value of <0.001 indicates a statistically significant difference, with Group B patients experiencing a longer pain-free period before requiring additional pain medication compared to Group A.

Graph 8: Time to First Rescue Analgesia

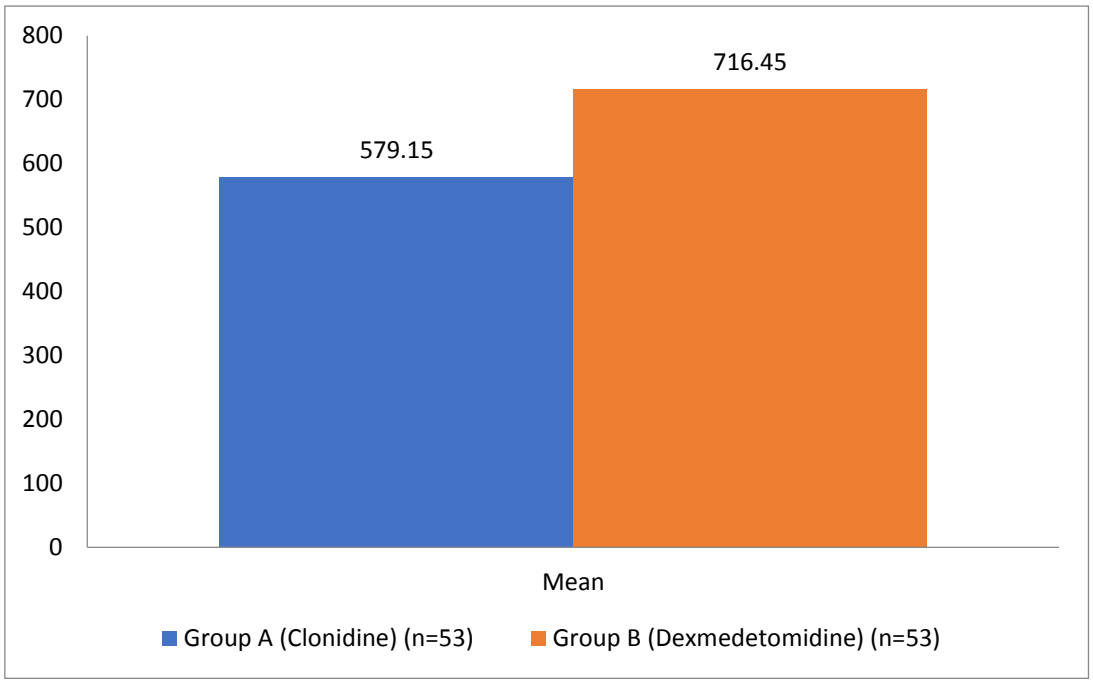


Table 9: VAS Score at Rescue Analgesia

VAS Score at Rescue Analgesia	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Mean ± SD	4.51 ± 1.09	3.28 ± 1.10	**<0.001***

*Statistically significant

This table displays the Visual Analog Scale (VAS) pain scores at the time of rescue analgesia. The mean VAS score was 4.51 ± 1.09 for Group A (Clonidine) and 3.28 ± 1.10 for Group B (Dexmedetomidine). The p-value of <0.001 indicates a statistically significant difference, with Group B patients reporting lower pain scores at the time they required rescue analgesia compared to Group A.

Graph 9: VAS Score at Rescue Analgesia

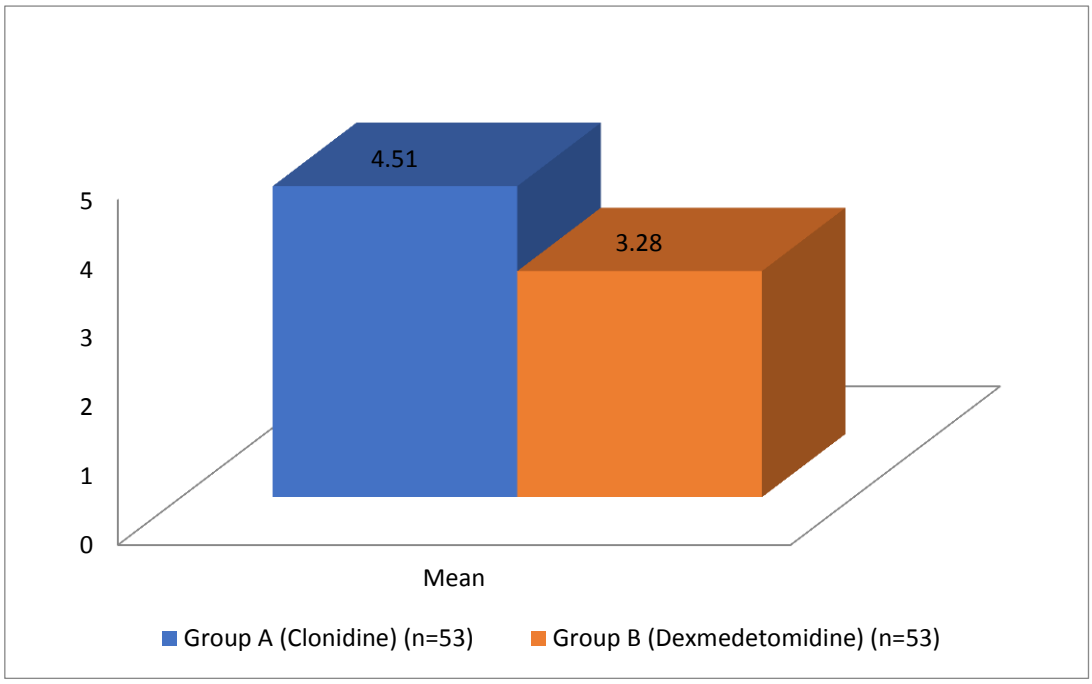


Table 10: Number of Rescue Analgesics in 24h

Number of Rescue Analgesics in 24h	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Mean ± SD	3.51 ± 1.15	1.92 ± 0.85	**<0.001***

*Statistically significant

This table presents the number of rescue analgesics required during the first 24 hours after surgery. Group A (Clonidine) patients required a mean of 3.51 ± 1.15 doses, while Group B (Dexmedetomidine) patients required 1.92 ± 0.85 doses. The p-value of <0.001 indicates a statistically significant difference, with Group B patients requiring significantly fewer rescue analgesics in the first 24 hours compared to Group A.

Graph 10: Number of Rescue Analgesics in 24h

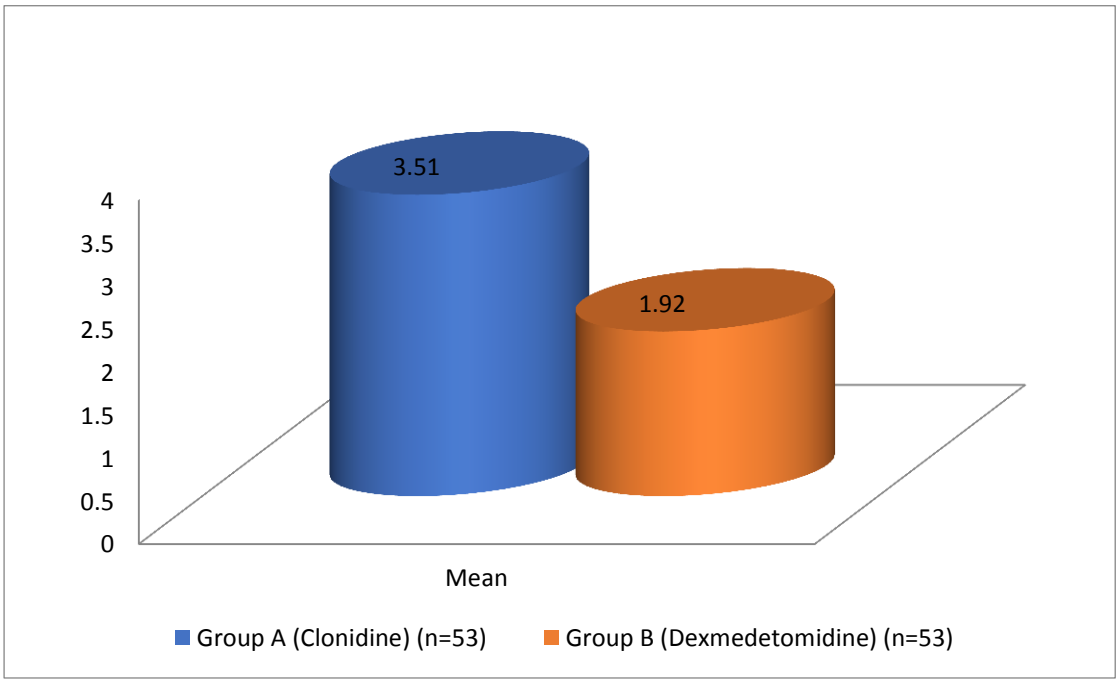


Table 11: Ramsay Sedation Score

Ramsay Sedation Score (max)	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Mean ± SD	2.11 ± 0.32	2.55 ± 0.64	**<0.001***

*Statistically significant

This table shows the maximum Ramsay Sedation Score observed in each group. The mean maximum sedation score was 2.11 ± 0.32 for Group A (Clonidine) and 2.55 ± 0.64 for Group B (Dexmedetomidine). The p-value of <0.001 indicates a statistically significant difference, with Group B patients experiencing a higher level of sedation compared to Group A.

Graph 11: Ramsay Sedation Score

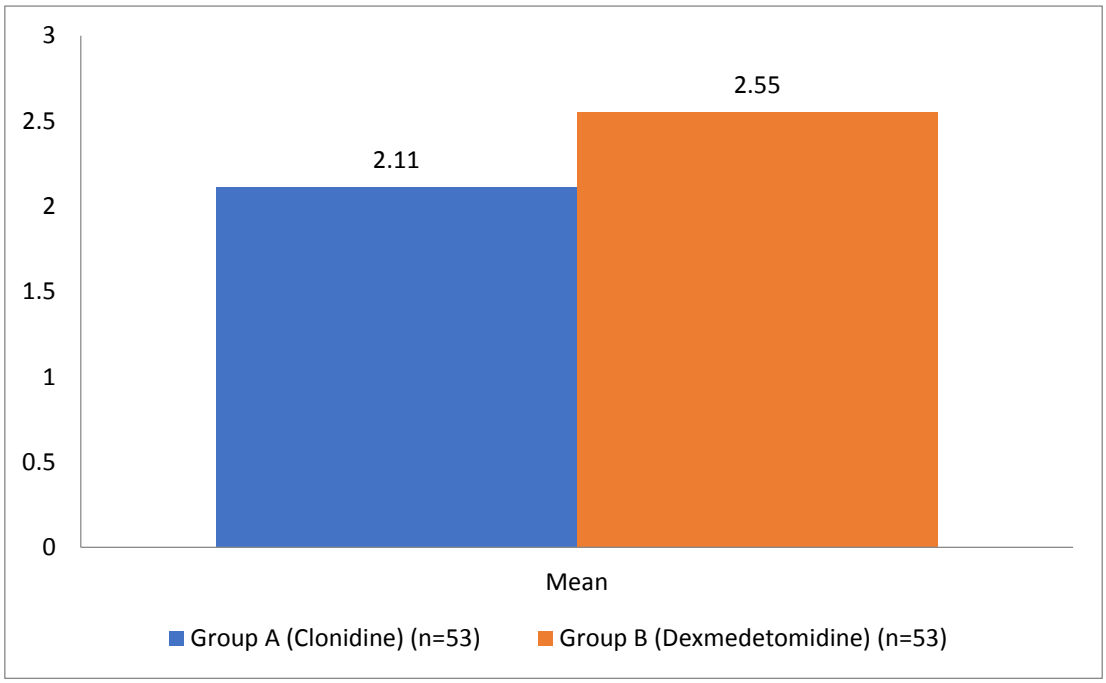


Table 12: Heart Rate at Different Time Intervals

Time (minutes)	Group A (Clonidine)	Group B (Dexmedetomidine)	p-value
Baseline	76.68 ± 8.68	77.91 ± 7.27	0.432
5 min	74.20 ± 7.42	75.60 ± 7.56	0.328
15 min	72.85 ± 7.29	73.90 ± 7.39	0.452
30 min	71.50 ± 7.15	72.30 ± 7.23	0.566
60 min	70.95 ± 7.10	71.50 ± 7.15	0.682
90 min	70.40 ± 7.04	70.80 ± 7.08	0.765
120 min	69.80 ± 6.98	70.20 ± 7.02	0.762
150 min	69.30 ± 6.93	69.75 ± 6.98	0.730

This table presents heart rate measurements at different time points during the procedure. The baseline heart rates were 76.68 ± 8.68 beats per minute (bpm) for Group A (Clonidine) and 77.91 ± 7.27 bpm for Group B (Dexmedetomidine). Throughout the monitoring period (5, 15, 30, 60, 90, 120, and 150 minutes), heart rates gradually decreased in both groups, but all p-values were >0.05, indicating no statistically significant differences in heart rate between the two groups at any time point.

Graph 12: Heart Rate at Different Time Intervals

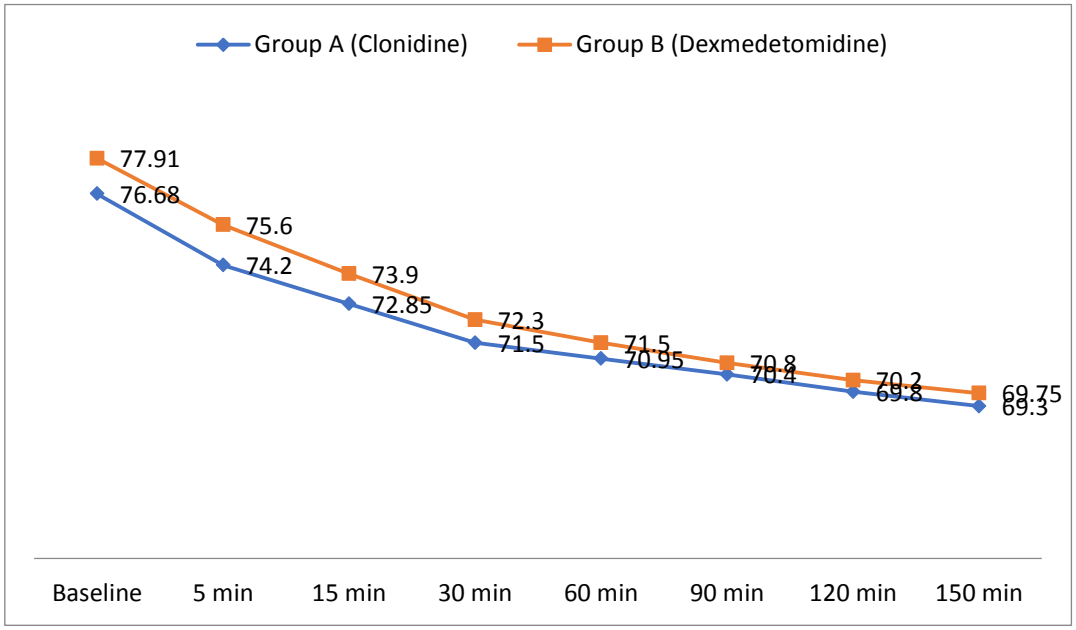


Table 13: Systolic Blood Pressure at Different Time Intervals

Time (minutes)	Group A (Clonidine)	Group B (Dexmedetomidine)	p-value
Baseline	125.34 ± 11.06	123.70 ± 9.96	0.424
5 min	123.50 ± 12.35	122.40 ± 12.24	0.642
15 min	121.20 ± 12.12	120.10 ± 12.01	0.634
30 min	119.80 ± 11.98	118.60 ± 11.86	0.595
60 min	118.40 ± 11.84	117.30 ± 11.73	0.624
90 min	117.60 ± 11.76	116.50 ± 11.65	0.628
120 min	116.90 ± 11.69	115.80 ± 11.58	0.620
150 min	116.20 ± 11.62	115.20 ± 11.52	0.653

This table shows systolic blood pressure measurements throughout the procedure. Baseline systolic blood pressures were 125.34 ± 11.06 mmHg for Group A (Clonidine) and 123.70 ± 9.96 mmHg for Group B (Dexmedetomidine). Both groups showed a gradual decrease in systolic blood pressure over time, but all p-values were >0.05, indicating no statistically significant differences between the groups at any time point.

Graph 13: Systolic Blood Pressure at Different Time Intervals

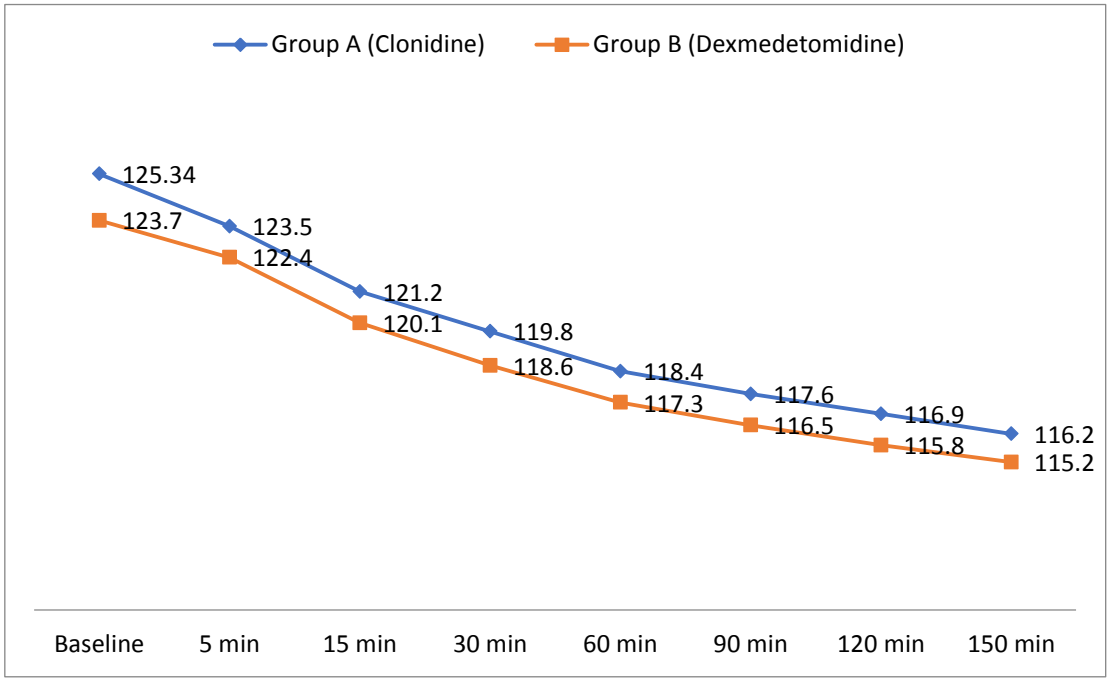


Table 14: Diastolic Blood Pressure at Different Time Intervals

Time (minutes)	Group A (Clonidine)	Group B (Dexmedetomidine)	p-value
Baseline	78.21 ± 6.65	78.87 ± 5.73	0.585
5 min	76.30 ± 7.63	75.50 ± 7.55	0.576
15 min	75.10 ± 7.51	74.40 ± 7.44	0.615
30 min	74.20 ± 7.42	73.60 ± 7.36	0.668
60 min	73.60 ± 7.36	73.10 ± 7.31	0.712
90 min	73.20 ± 7.32	72.70 ± 7.27	0.715
120 min	72.80 ± 7.28	72.30 ± 7.23	0.708
150 min	72.40 ± 7.24	72.00 ± 7.20	0.765

This table presents diastolic blood pressure measurements during the procedure. Baseline diastolic blood pressures were 78.21 ± 6.65 mmHg for Group A (Clonidine) and 78.87 ± 5.73 mmHg for Group B (Dexmedetomidine). Both groups showed slight decreases in diastolic blood pressure over the monitored period, but all p-values were >0.05, indicating no statistically significant differences between the groups at any time point.

Graph 14: Diastolic Blood Pressure at Different Time Intervals

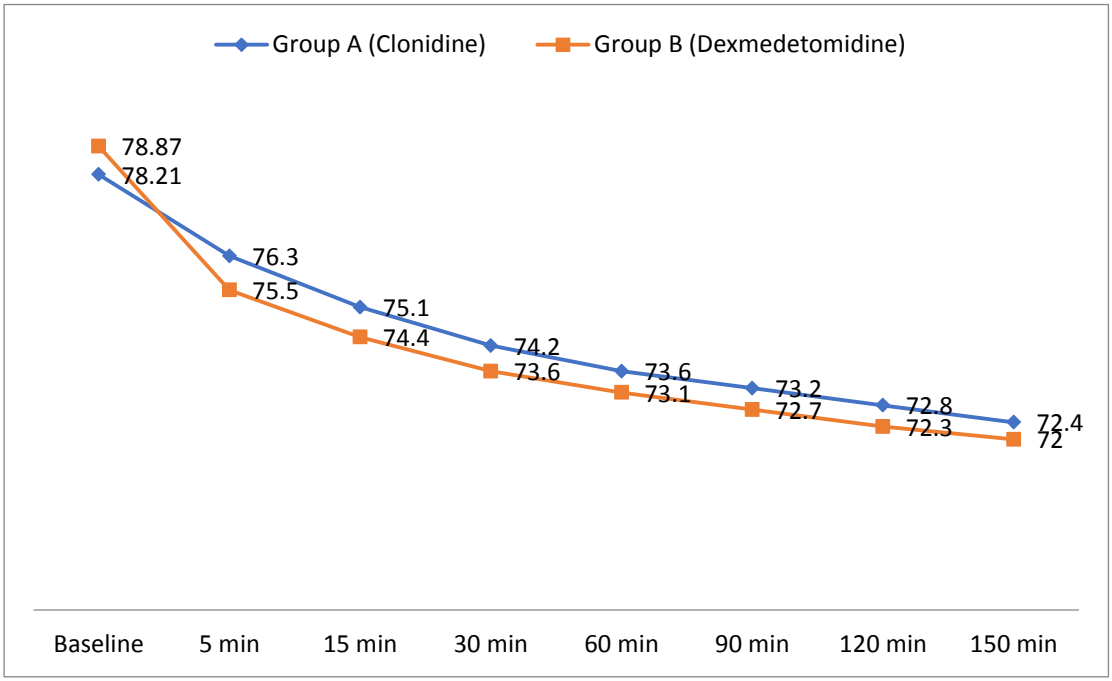


Table 15: Mean Arterial Pressure at Different Time Intervals

Time (minutes)	Group A (Clonidine)	Group B (Dexmedetomidine)	p-value
Baseline	94.15 ± 9.41	93.21 ± 9.32	0.596
5 min	92.03 ± 9.20	91.13 ± 9.11	0.605
15 min	90.47 ± 9.05	89.63 ± 8.96	0.624
30 min	89.40 ± 8.94	88.60 ± 8.86	0.632
60 min	88.53 ± 8.85	87.83 ± 8.78	0.670
90 min	88.00 ± 8.80	87.30 ± 8.73	0.662
120 min	87.50 ± 8.75	86.80 ± 8.68	0.668
150 min	87.00 ± 8.70	86.40 ± 8.64	0.705

This table displays mean arterial pressure (MAP) measurements throughout the procedure. Baseline MAP values were 94.15 ± 9.41 mmHg for Group A (Clonidine) and 93.21 ± 9.32 mmHg for Group B (Dexmedetomidine). Both groups showed gradual decreases in MAP over time, but all p-values were >0.05, indicating no statistically significant differences between the groups at any time point.

Graph 15: Mean Arterial Pressure at Different Time Intervals

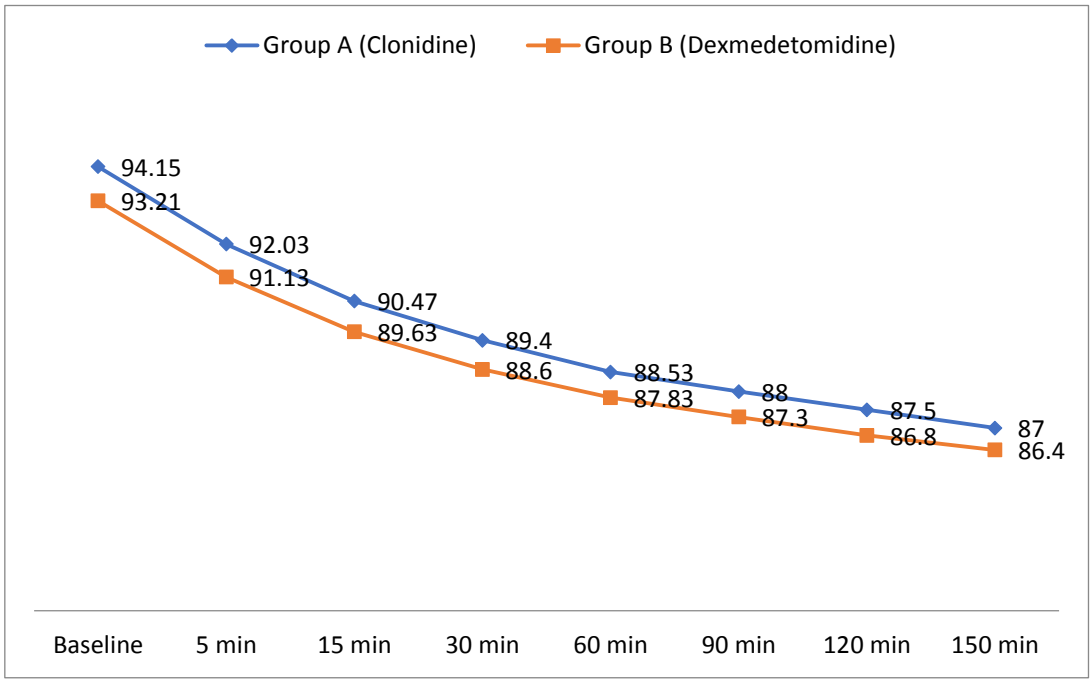


Table 16: Oxygen Saturation at Different Time Intervals

Time (minutes)	Group A (Clonidine)	Group B (Dexmedetomidine)	p-value
Baseline	99.10 ± 0.99	99.15 ± 0.99	0.782
5 min	99.05 ± 0.99	99.10 ± 0.99	0.764
15 min	99.15 ± 0.99	99.20 ± 0.99	0.760
30 min	99.20 ± 0.99	99.25 ± 0.99	0.775
60 min	99.25 ± 0.99	99.30 ± 0.99	0.765
90 min	99.20 ± 0.99	99.25 ± 0.99	0.774
120 min	99.15 ± 0.99	99.20 ± 0.99	0.761
150 min	99.10 ± 0.99	99.15 ± 0.99	0.758

This table shows oxygen saturation levels during the procedure. Baseline oxygen saturation levels were 99.10 ± 0.99% for Group A (Clonidine) and 99.15 ± 0.99% for Group B (Dexmedetomidine). Both groups maintained high oxygen saturation levels throughout the monitored period, and all p-values were >0.05, indicating no statistically significant differences between the groups at any time point.

Graph 16: Oxygen Saturation at Different Time Intervals

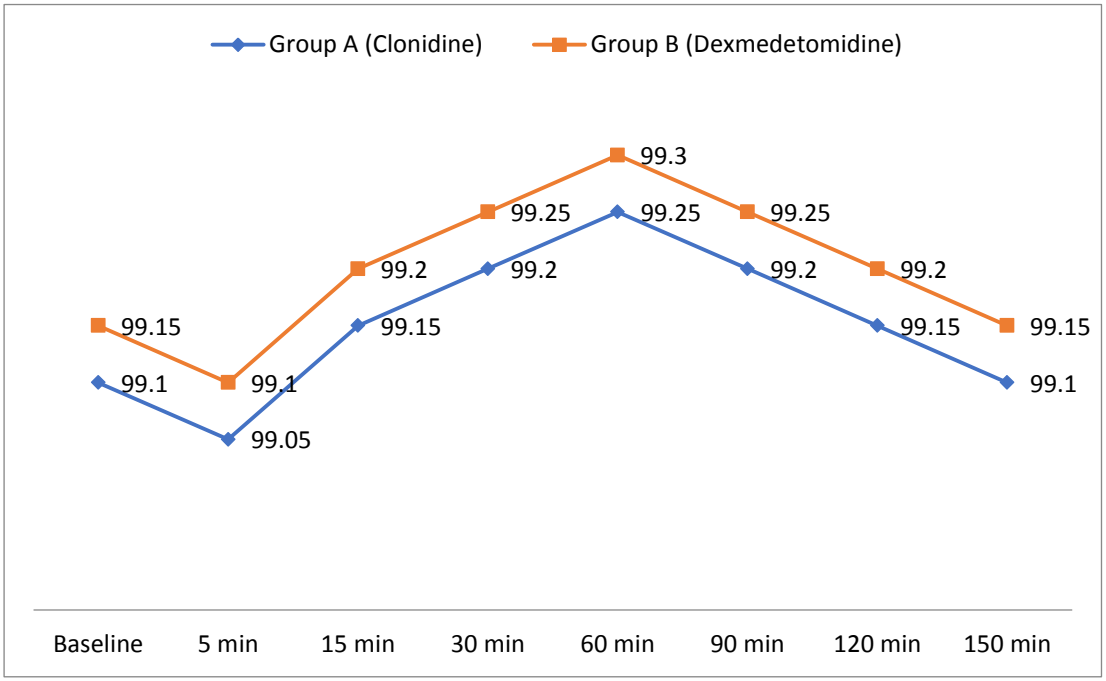
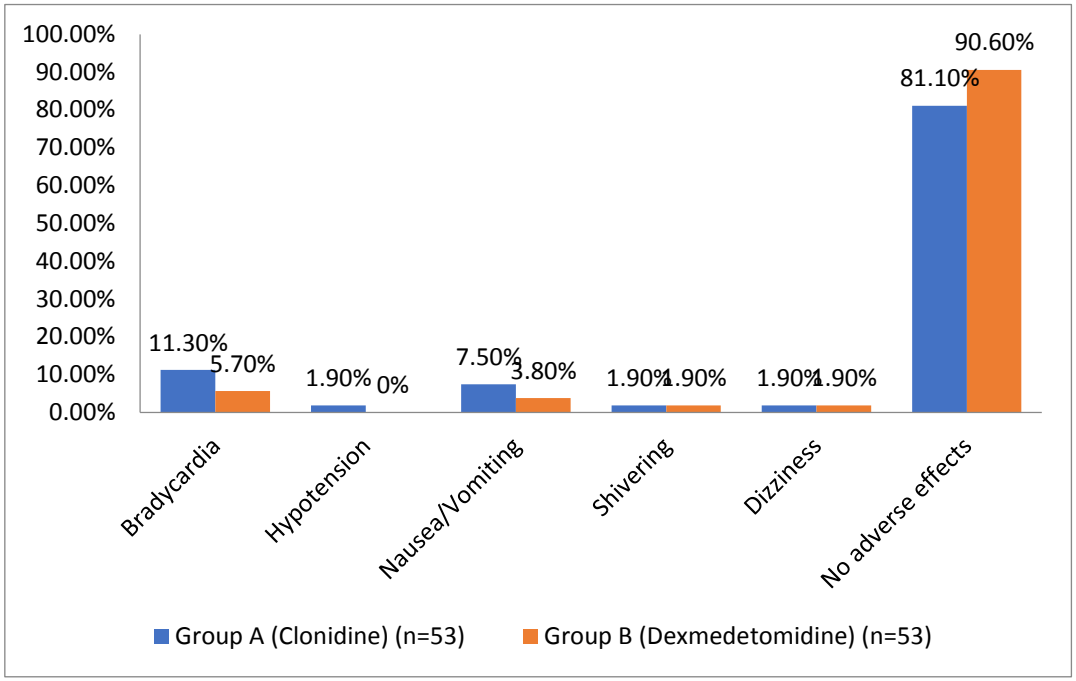


Table 17: Adverse Effects

Adverse Effect	Group A (Clonidine) (n=53)	Group B (Dexmedetomidine) (n=53)	p-value
Bradycardia	6 (11.3%)	3 (5.7%)	0.488
Hypotension	1 (1.9%)	0 (0%)	1.000
Nausea/Vomiting	4 (7.5%)	2 (3.8%)	0.678
Shivering	1 (1.9%)	1 (1.9%)	1.000
Dizziness	1 (1.9%)	1 (1.9%)	1.000
No adverse effects	43 (81.1%)	48 (90.6%)	0.156

This table presents the occurrence of adverse effects in both groups. In Group A (Clonidine), 11.3% experienced bradycardia, 1.9% had hypotension, 7.5% had nausea/vomiting, 1.9% had shivering, 1.9% experienced dizziness, and 81.1% had no adverse effects. In Group B (Dexmedetomidine), 5.7% had bradycardia, 0% had hypotension, 3.8% had nausea/vomiting, 1.9% had shivering, 1.9% had dizziness, and 90.6% had no adverse effects. All p-values were >0.05, indicating no statistically significant differences in the occurrence of adverse effects between the two groups, though Group B tended to have fewer adverse effects overall.

Figure 17: Adverse Effects



DISCUSSION

DISCUSSION

Regional anaesthesia techniques have evolved considerably over the past few decades, with ultrasound-guided peripheral nerve blocks becoming the standard of care for many surgical procedures. The supraclavicular approach to brachial plexus block is widely utilized for upper limb surgeries due to its high success rate and reliable anaesthesia. While local anaesthetics alone provide effective analgesia, their relatively short duration of action necessitates the use of adjuvants to prolong the analgesic effect. Alpha-2 adrenergic receptor agonists, specifically clonidine and dexmedetomidine, have emerged as valuable adjuvants to local anaesthetics in peripheral nerve blocks. These agents not only extend the duration of analgesia but also improve the quality of block and reduce the requirement for rescue analgesics in the postoperative period. The present study aimed to compare the efficacy of clonidine and dexmedetomidine as adjuvants to ropivacaine in ultrasound-guided supraclavicular brachial plexus block for patients undergoing upper limb surgeries.

Demographic Profile:

In our study, both Group A (clonidine) and Group B (dexmedetomidine) were comparable in terms of demographic characteristics including age, gender distribution, BMI, and ASA physical status. This demographic homogeneity strengthens the validity of our findings by minimizing potential confounding factors that could influence the outcomes.

Das A et al. conducted a similar study comparing clonidine and dexmedetomidine as adjuvants to ropivacaine in supraclavicular brachial plexus block, and reported comparable demographic profiles between their study groups.[61]

Their sample consisted of 50 patients in each group with mean ages of 37.16 ± 13.64 years in the clonidine group and 36.92 ± 14.08 years in the dexmedetomidine group, which aligns closely with our study population (38.25 ± 13.41 years for Group A and 35.34 ± 11.40 years for Group B).

Similarly, Mangal V et al. reported no significant differences in demographic parameters between their clonidine and dexmedetomidine groups in a randomized double-blind study of 60 patients undergoing upper limb surgeries under ultrasound-guided supraclavicular block.[62]Their gender distribution was also comparable to our study, with male predominance in both groups, reflecting the typical pattern of traumatic upper limb injuries requiring surgical intervention.

Onset and Duration of Sensory Block:

One of the most significant findings of our study was the difference in onset time and duration of sensory block between the two groups. The dexmedetomidine group demonstrated a significantly faster onset of sensory block (6.95 ± 1.47 minutes) compared to the clonidine group (9.28 ± 1.80 minutes), with a p-value of <0.001. Additionally, the duration of sensory block was significantly longer in the dexmedetomidine group (668.26 ± 41.65 minutes) compared to the clonidine group (551.81 ± 43.93 minutes).

These findings are consistent with those reported by Swami SS et al., who compared dexmedetomidine and clonidine as adjuvants to bupivacaine in supraclavicular brachial plexus block.[63] They found that dexmedetomidine provided significantly faster onset (13.2 ± 1.8 vs. 15.6 ± 1.5 minutes) and longer duration (413.2 ± 31.5 vs. 227.3 ± 47.7 minutes) of sensory block compared to clonidine.

While the absolute values differ from our study due to the use of a different local anaesthetic (bupivacaine vs. ropivacaine in our study), the relative advantage of dexmedetomidine over clonidine is consistently observed.

Esmaoglu A et al. investigated the effects of adding dexmedetomidine to levobupivacaine for axillary brachial plexus block and reported a significant prolongation of sensory block duration.[64] They attributed this effect to the peripheral action of dexmedetomidine on α2A-adrenoceptors, which inhibits the release of norepinephrine and enhances the local anaesthetic action.

The superior effect of dexmedetomidine over clonidine can be explained by its higher selectivity for α2-adrenergic receptors (α2:α1 ratio of 1620:1 for dexmedetomidine vs. 220:1 for clonidine).[65] This higher selectivity translates to more potent effects on peripheral nerve block characteristics, as demonstrated in our study.

Zhang Y et al. conducted a meta-analysis of randomized controlled trials comparing dexmedetomidine and clonidine as adjuvants to local anaesthetics in brachial plexus block.[66] Their analysis of 14 trials involving 868 patients concluded that dexmedetomidine significantly shortened the sensory block onset time and prolonged the duration of sensory block compared to clonidine, which aligns with our findings.

Onset and Duration of Motor Block:

Similar to sensory block characteristics, our study demonstrated significant differences in motor block parameters between the two groups. The onset time of motor block was significantly faster in the dexmedetomidine group (8.70 ± 2.00 minutes) compared to the clonidine group (12.08 ± 2.40 minutes) with a p-value of <0.001. The duration of motor block was also significantly longer in the dexmedetomidine group (595.06 ± 42.37 minutes) compared to the clonidine group

(497.94 ± 46.61 minutes).

These findings are supported by Hussain N et al., who performed a systematic review and meta-analysis of perineural dexmedetomidine as an adjuvant to local anaesthetics in various nerve blocks.[67] They analyzed 32 randomized controlled trials and found that dexmedetomidine consistently accelerated the onset and prolonged the duration of motor block across different types of nerve blocks and local anaesthetics. Kathuria S et al. compared dexmedetomidine and clonidine as adjuvants to ropivacaine 0.75% in ultrasound-guided supraclavicular block and reported motor block onset times of 10.5 ± 2.1 minutes in the dexmedetomidine group and 14.2 ± 2.3 minutes in the clonidine group.[68] They also found longer motor block duration with dexmedetomidine (486 ± 41 minutes) compared to clonidine (310 ± 31 minutes). While their absolute values differ from our study, possibly due to variations in drug dosages and assessment methods, the relative advantage of dexmedetomidine over clonidine remains consistent."

The prolonged motor blockade with dexmedetomidine may be attributed to its action on α2-adrenoceptors in the peripheral nervous system. Brummett CM et al. demonstrated in an animal study that perineural dexmedetomidine added to ropivacaine enhances the duration of sensory and motor blockade by blocking the hyperpolarization-activated cation current (Ih current), which prevents the nerve from returning to its resting membrane potential.[69]

Duration of Analgesia and Rescue Analgesic Requirements:

A key objective in regional anaesthesia is to provide prolonged postoperative analgesia, reducing the need for systemic analgesics and their associated side effects. Our study demonstrated that patients in the dexmedetomidine group had significantly

longer time to first rescue analgesia (716.45 ± 76.88 minutes) compared to the clonidine group (579.15 ± 57.30 minutes), with a p-value of <0.001. Moreover, patients in the dexmedetomidine group required significantly fewer rescue analgesics in the first 24 hours postoperatively (1.92 ± 0.85) compared to the clonidine group (3.51 ± 1.15).

These findings are consistent with those reported by Gandhi R et al., who compared dexmedetomidine, clonidine, and butorphanol as adjuvants to ropivacaine in supraclavicular brachial plexus block.[70]

They found that dexmedetomidine provided significantly longer postoperative analgesia (773.80 ± 58.80 minutes) compared to clonidine (574.80 ± 60.96 minutes) and butorphanol (410.60 ± 44.22 minutes). They also reported reduced rescue analgesic consumption in the dexmedetomidine group during the first 24 hours postoperatively.

Singh AP et al. compared clonidine and dexmedetomidine as adjuvants to bupivacaine in supraclavicular brachial plexus block and reported that the time to first rescue analgesia was significantly longer in the dexmedetomidine group (456 ± 97 minutes) compared to the clonidine group (289 ± 62 minutes).[71]They also found that the total number of rescue analgesics required in the first 24 hours was significantly lower in the dexmedetomidine group.

The enhanced analgesic efficacy of dexmedetomidine can be attributed to both peripheral and central mechanisms. Peripherally, dexmedetomidine reduces norepinephrine release and prevents nerve impulse propagation by hyperpolarization of post-synaptic dorsal horn neurons.[72] Centrally, it activates α2-adrenoceptors in the locus coeruleus, leading to inhibition of adenylyl cyclase and reducing pain

transmission.

Pain Intensity at Rescue Analgesia:

Our study showed that patients in the dexmedetomidine group had significantly lower VAS scores at the time of rescue analgesia (3.28 ± 1.10) compared to those in the clonidine group (4.51 ± 1.09), with a p-value of <0.001.

This finding suggests that dexmedetomidine not only prolongs the time to first analgesic requirement but also provides better quality of analgesia during the transition period from effective block to the need for supplementary analgesia.

Similar findings were reported by El-Boghdadly K et al. in their systematic review and meta-analysis on perineural dexmedetomidine.[73]

They found that dexmedetomidine was associated with reduced pain scores and analgesic consumption in the early postoperative period compared to control groups. Although they did not directly compare dexmedetomidine with clonidine, their findings support the superior analgesic efficacy of dexmedetomidine as an adjuvant in peripheral nerve blocks.

Agarwal S et al. compared dexmedetomidine and clonidine as adjuvants to bupivacaine in supraclavicular brachial plexus block and found that pain scores at the time of first analgesic request were significantly lower in the dexmedetomidine group compared to the clonidine group.[74] They attributed this to the higher α2-selectivity of dexmedetomidine and its enhanced penetration into the nerve membrane.

Sedation:

Sedation is a known effect of α2-adrenergic receptor agonists due to their action on central α2-receptors. In our study, the maximum Ramsay Sedation Score was

significantly higher in the dexmedetomidine group (2.55 ± 0.64) compared to the clonidine group (2.11 ± 0.32), with a p-value of <0.001, indicating a greater degree of sedation with dexmedetomidine.

This finding is consistent with that of Tripathi A et al., who compared dexmedetomidine and clonidine as adjuvants to ropivacaine in supraclavicular brachial plexus block and found that patients in the dexmedetomidine group had higher sedation scores compared to those in the clonidine group.[75]

They reported that the sedation was arousable and did not cause respiratory depression, similar to our observations.

The sedative effect of perineural dexmedetomidine is likely due to its systemic absorption and subsequent action on central α2-receptors in the locus coeruleus. The higher degree of sedation observed with dexmedetomidine compared to clonidine can be attributed to its higher α2-selectivity and potency.

Importantly, the sedation observed in our study was mild to moderate (Ramsay Sedation Score of 2-3), which is considered beneficial in the perioperative setting as it reduces patient anxiety without causing respiratory depression.

Hemodynamic Parameters:

Our study showed no statistically significant differences in heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure between the two groups at any time point during the 150-minute observation period. Both groups showed a gradual decrease in hemodynamic parameters from baseline, which is a known effect of sympathetic blockade associated with brachial plexus block.

These findings differ somewhat from those reported by Das A et al., who found a

greater reduction in heart rate and blood pressure in their dexmedetomidine group compared to the clonidine group.[61] They attributed this to the higher α2-selectivity of dexmedetomidine and its more potent sympatholytic effect.

The absence of significant hemodynamic differences between our study groups may be explained by the use of lower doses of both adjuvants (1 μg/kg for dexmedetomidine and 2 μg/kg for clonidine), which minimized their systemic effects while preserving their local anaesthetic-enhancing properties. Additionally, the use of ultrasound guidance in our study allowed precise deposition of the local anaesthetic mixture around the brachial plexus, reducing the risk of systemic absorption and associated hemodynamic effects.

Vorobeichik L et al. conducted a systematic review and meta-analysis on the effects of perineural versus intravenous dexmedetomidine as an adjuvant for peripheral nerve blocks.[76] They found that perineural administration of dexmedetomidine was associated with less pronounced hemodynamic effects compared to intravenous administration, supporting our approach of using perineural dexmedetomidine to maximize local effects while minimizing systemic side effects.

Adverse Effects:

In our study, there were no statistically significant differences in the incidence of adverse effects between the two groups, although the dexmedetomidine group tended to have fewer adverse effects overall (90.6% of patients with no adverse effects compared to 81.1% in the clonidine group). The most common adverse effect in both groups was bradycardia (11.3% in the clonidine group vs. 5.7% in the dexmedetomidine group), followed by nausea/vomiting (7.5% in the clonidine group vs. 3.8% in the dexmedetomidine group).

These findings are similar to those reported by Mangal V et al., who found no significant differences in adverse effects between their clonidine and dexmedetomidine groups, with a slight trend towards fewer side effects in the dexmedetomidine group.[62] They attributed this to the higher α2-selectivity of dexmedetomidine, which may result in more targeted effects with fewer off-target side effects.

Chinnappa J et al. compared clonidine and dexmedetomidine as adjuvants to levobupivacaine in supraclavicular brachial plexus block and reported minor adverse effects such as bradycardia, hypotension, and sedation, with no statistically significant differences between the groups.[77] They concluded that both adjuvants were safe when used in appropriate doses for peripheral nerve blocks.

The relatively low incidence of adverse effects in our study can be attributed to several factors, including the use of ultrasound guidance for precise needle placement, careful dose selection of adjuvants, and exclusion of patients with significant comorbidities (ASA III and above) who might be more susceptible to adverse hemodynamic effects.

LIMITATIONS

Limitations and Future Directions:

Our study had several limitations that should be acknowledged. First, we used fixed doses of adjuvants (1 μg/kg for dexmedetomidine and 2 μg/kg for clonidine) based on body weight. Future studies could explore different dose ratios to identify the optimal dose of each adjuvant that maximizes analgesic efficacy while minimizing side effects.

Second, our follow-up period was limited to 24 hours postoperatively. Longer follow-up periods would be valuable to assess the potential impact of these adjuvants on chronic pain development and long-term outcomes.

Third, we did not measure plasma concentrations of the adjuvants, which would have provided insights into their systemic absorption and correlation with clinical effects. Future pharmacokinetic studies could help elucidate the relationship between plasma levels and clinical efficacy/safety of these adjuvants.

Finally, our study focused on ultrasound-guided supraclavicular brachial plexus block for upper limb surgeries. The findings may not be generalizable to other approaches to brachial plexus block (e.g., infraclavicular, axillary) or other types of peripheral nerve blocks.

Future research directions could include:

1. Dose-finding studies to determine the optimal doses of clonidine and dexmedetomidine as adjuvants to different local anaesthetics.
2. Comparison of different administration routes (perineural vs. intravenous vs. oral premedication) to identify the most effective and safe method of administration.

3. Investigation of the molecular mechanisms underlying the differential effects of clonidine and dexmedetomidine on peripheral nerves.
4. Exploration of potential synergistic effects between α2-adrenergic receptor agonists and other adjuvants (e.g., opioids, dexamethasone) in peripheral nerve blocks.
5. Assessment of the impact of these adjuvants on chronic postsurgical pain development through long-term follow-up studies.

CONCLUSION

CONCLUSION:

Our study demonstrates that both clonidine and dexmedetomidine are effective adjuvants to ropivacaine in ultrasound-guided supraclavicular brachial plexus block for upper limb surgeries. However, dexmedetomidine offers several advantages over clonidine, including faster onset and longer duration of sensory and motor block, prolonged postoperative analgesia, reduced rescue analgesic requirements, and lower pain scores at the time of rescue analgesia. These benefits are likely attributable to the higher α2-selectivity of dexmedetomidine compared to clonidine.

The hemodynamic stability observed with both adjuvants, along with the low incidence of adverse effects, supports their safety profile when used in appropriate doses for peripheral nerve blocks. The mild to moderate sedation associated with dexmedetomidine may be considered beneficial in the perioperative setting as it enhances patient comfort without causing respiratory depression.

Based on our findings, dexmedetomidine appears to be a superior adjuvant to ropivacaine compared to clonidine for ultrasound-guided supraclavicular brachial plexus block in patients undergoing upper limb surgeries. However, the choice between these adjuvants should consider individual patient factors, surgical requirements, and cost considerations in clinical practice.

This randomized controlled trial comparing clonidine and dexmedetomidine as adjuvants to ropivacaine in ultrasound-guided supraclavicular brachial plexus block for upper limb surgeries demonstrates the superior efficacy of dexmedetomidine over clonidine in several clinically relevant parameters.

Dexmedetomidine significantly accelerated the onset of both sensory and motor blockade compared to clonidine, providing faster surgical readiness and improving

operating room efficiency. The duration of sensory and motor blockade was substantially prolonged in the dexmedetomidine group, offering extended postoperative analgesia that translated to improved patient comfort during the critical early recovery period.

The time to first rescue analgesia was significantly longer in the dexmedetomidine group, and the total analgesic consumption within the first 24 hours was markedly reduced compared to the clonidine group. Importantly, patients in the dexmedetomidine group reported lower pain scores at the time of rescue analgesia, indicating a smoother transition from effective regional anaesthesia to systemic analgesia. These findings reflect the superior analgesic efficacy of dexmedetomidine, which can be attributed to its higher selectivity for α2-adrenergic receptors compared to clonidine.

The hemodynamic parameters remained stable in both groups throughout the observation period, with no significant differences between the groups, establishing the cardiovascular safety of both adjuvants at the doses used in this study. Though the dexmedetomidine group exhibited higher sedation scores, the level of sedation was clinically acceptable and potentially beneficial in the perioperative setting by reducing patient anxiety without causing respiratory depression.

The incidence of adverse effects was low in both groups, with a trend toward fewer side effects in the dexmedetomidine group, though this difference did not reach statistical significance. This favourable safety profile, combined with the enhanced efficacy, positions dexmedetomidine as an excellent adjuvant option for brachial plexus blocks.

Based on these findings, we conclude that dexmedetomidine (1 μg/kg) is superior to clonidine (2 μg/kg) as an adjuvant to ropivacaine (0.5%) in ultrasound-guided supraclavicular brachial plexus block for patients undergoing upper limb surgeries. Dexmedetomidine provides faster onset, longer duration of analgesia, better quality of pain relief, and a favourable side effect profile, making it the preferred adjuvant for improving postoperative pain management in these patients.

SUMMARY

SUMMARY

This prospective, randomized, double-blind study was conducted on 106 ASA physical status I and II patients aged 18-60 years undergoing elective or emergency upper limb surgeries under ultrasound-guided supraclavicular brachial plexus block at R.L. Jalappa Hospital and Research Centre, Tamaka, Kolar over a period of 18 months. Patients were randomly allocated into two groups of 53 each: Group A received 30 ml of 0.5% ropivacaine with clonidine 2 μg/kg, and Group B received 30 ml of 0.5% ropivacaine with dexmedetomidine 1 μg/kg.

The demographic characteristics including age, gender, BMI, and ASA physical status were comparable between the two groups, as was the duration of surgery, establishing a homogeneous study population. The onset of sensory block was significantly faster in the dexmedetomidine group (6.95 ± 1.47 minutes) compared to the clonidine group (9.28 ± 1.80 minutes). Similarly, the onset of motor block was more rapid in the dexmedetomidine group (8.70 ± 2.00 minutes) than in the clonidine group (12.08 ± 2.40 minutes).

The duration of sensory block was significantly longer in the dexmedetomidine group (668.26 ± 41.65 minutes) compared to the clonidine group (551.81 ± 43.93 minutes). The motor block duration was also extended in the dexmedetomidine group (595.06 ± 42.37 minutes) compared to the clonidine group (497.94 ± 46.61 minutes).

The time to first rescue analgesia was substantially prolonged in the dexmedetomidine group (716.45 ± 76.88 minutes) compared to the clonidine group (579.15 ± 57.30 minutes). The visual analog scale (VAS) pain score at the time of rescue analgesia was significantly lower in the dexmedetomidine group (3.28 ± 1.10) compared to the clonidine group (4.51 ± 1.09).

Patients in the dexmedetomidine group required fewer rescue analgesics in the first 24 hours (1.92 ± 0.85) compared to those in the clonidine group (3.51 ± 1.15).

The Ramsay sedation score was higher in the dexmedetomidine group (2.55 ± 0.64) compared to the clonidine group (2.11 ± 0.32), indicating a greater degree of sedation, though within clinically acceptable limits. Hemodynamic parameters including heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and oxygen saturation were stable in both groups throughout the observation period, with no significant differences between the groups.

The incidence of adverse effects was low in both groups, with bradycardia being the most common (11.3% in the clonidine group vs. 5.7% in the dexmedetomidine group), followed by nausea/vomiting (7.5% in the clonidine group vs. 3.8% in the dexmedetomidine group). Although there was a trend toward fewer adverse effects in the dexmedetomidine group (90.6% of patients with no adverse effects) compared to the clonidine group (81.1%), this difference was not statistically significant.

In summary, dexmedetomidine proved to be a superior adjuvant to ropivacaine compared to clonidine in ultrasound-guided supraclavicular brachial plexus block for upper limb surgeries, providing faster onset and longer duration of sensory and motor blockade, prolonged postoperative analgesia, reduced analgesic requirements, and a favourable safety profile.

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ANNEXURE

ANNEXURE

PROFORMA

"Ultrasound guided Supraclavicular brachial plexus block using clonidine versus dexmedetomidine as adjuvants to ropivacaine for post-operative analgesia in upper limb surgeries – A Randomised control trial."

Name Of Patient:

Age:

Gender:

UHID :

Group A – Patients receiving 0.5% ropivacaine 30mL along with clonidine 1mcg/kg diluted with normal saline reconstituted to a total volume of 32mL.

Group B – Patients receiving 0.5% ropivacaine 30mL along with dexmedetomidine 1mcg/kg diluted with normal saline reconstituted to a total volume of 32mL.

General physical examination :

Local examination:

Pallor-

Icterus-

Cyanosis-

Clubbing-

Lymphadenopathy-

Oedema-

Systemic Examination

CVS-

RS-

CNS-

P/A-

Investigations

Hb-

RBC-

WBC-

PLATELETS-

PT-

APTT-

INR-

UREA-

CREATININE-

SODIUM-

POTASSIUM-

CXR-

ECG-

INTRA-OPERATIVE MONITORING

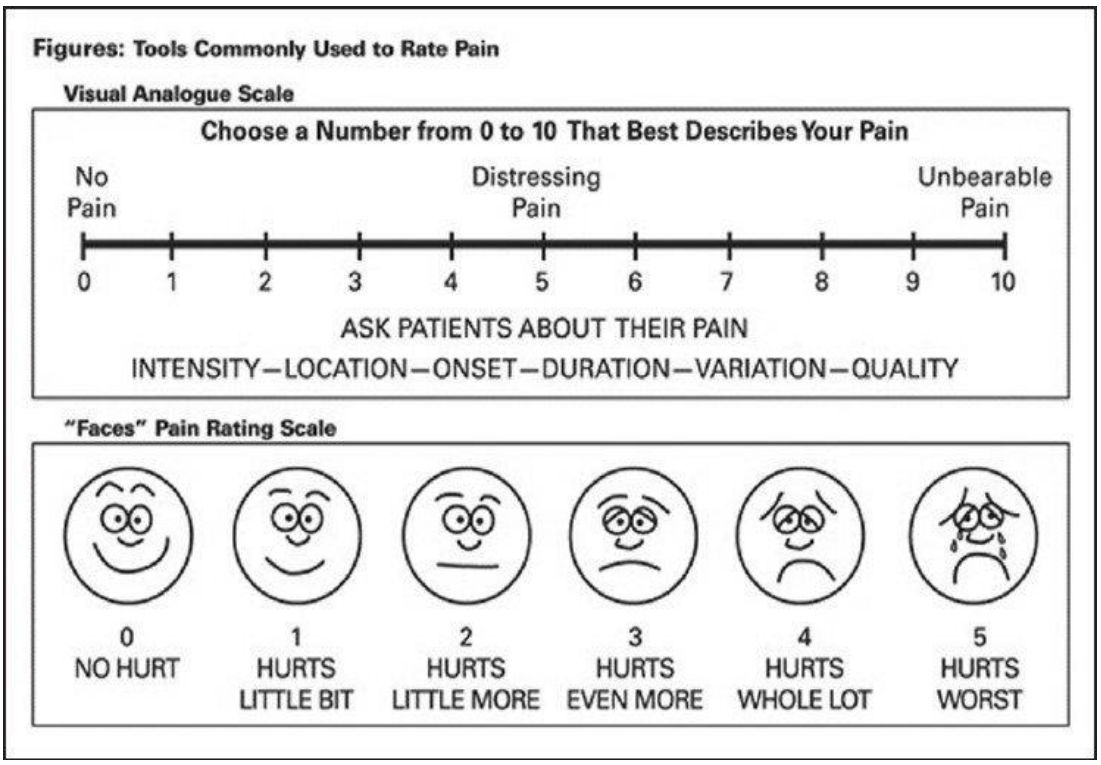
TIME	BASELINE	5’	15’	30’	60’	90’	120’	150’
BP(mm of Hg)								
HR (bpm)								
MAP								
SpO2(%)								
RR(cpm)								

Parameters	Time of onset	Duration of block	
Sensory blockade			
Motor blockade			

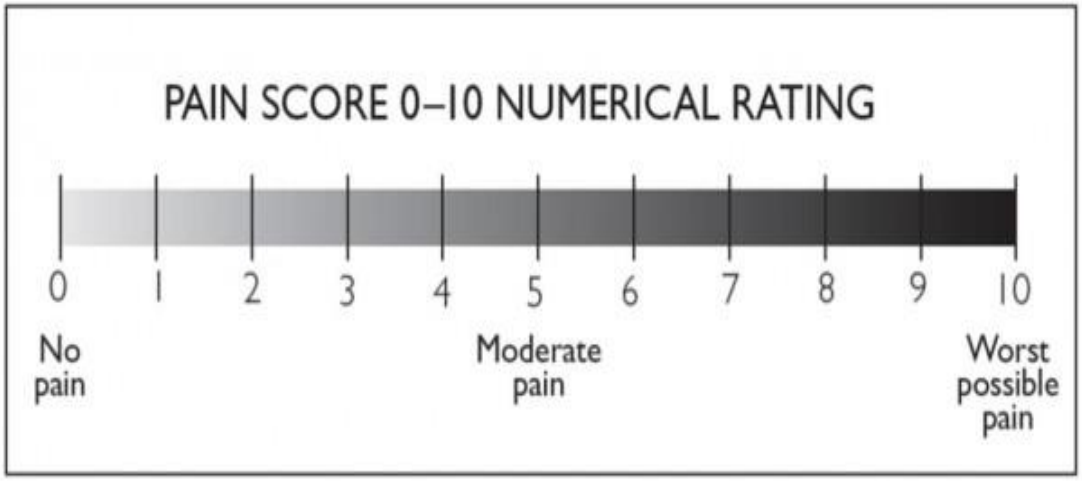
POST OPERATIVE MONITORING

Duration of first call for rescue analgesia post-operatively in the first 24 hours :

VAS SCORE at the time of rescue analgesia:



NRS(Numerical Pain Rating Scale) score:



Ramsay sedation score:

Scores	Responses
1	Anxious or restless or both
2	Cooperative, oriented, and tranquil
3	Responding to commands
4	Brisk response to stimulus
5	Sluggish response to stimulus
6	No response to stimulus

Scores 1, 2, and 3–awake; scores 4, 5 and 6–asleep.

The number of times patient asked for rescue analgesia in first 24 hours of post-operative period :

Analgesia administered :

"ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK USING CLONIDINE VERSUS DEXMEDETOMIDINE AS ADJUVANTS TO ROPIVACAINE FOR POST-OPERATIVE ANALGESIA IN UPPER LIMB SURGERIES – A RANDOMISED CONTROL TRIAL."

INFORMED CONSENT FORM

Date:

I, age/gender , UHID have

been explained in an understandable language about the purpose of the study **Dexmedetomidine or clonidine as adjuvants to ropivacaine in supraclavicular brachial plexus block during upper limb surgery. To Evaluate the efficacy of clonidine or dexmedetomidine as adjuvants to ropivacaine with respect to the onset and duration of sensory and motor blockade, duration of post-operative analgesia**.

Hence, hereby I give my valid written informed consent without any force or prejudice to be part of the study.

I have been explained about the side effects of dexmedetomidine and clonidine such as hypotension, and bradycardia (that will be treated with fluids and Inj.Atropine) were explained to the patient pre operatively.

The nature and risks involved have been explained to me to my understanding and satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I understand throughout the study if any form of payment/purchase is necessary, it will be

completely borne by the investigator. I will not be charged any extra cost throughout the study.

I consent voluntarily to take part as a participant in this research. I hereby give my full valid consent to provide my history, undergo physical examination, undergo the procedure, undergo investigations and provide its results and documents etc to the doctor / institute etc. For academic and scientific purpose, the operation / procedure etc. may be videographed or photographed.

All the data may be published or used for any academic purpose. I will not hold the doctors / institute etc. responsible for any untoward consequences during the procedure / study.

A copy of this Informed Consent Form and Patient Information Sheet has been provided to the participant.

Patient's consent (Signature/Thumb impression): Name of patient:

Witness 1 (Signature/Thumb impression): _

Name of witness 1: _

Relationship to patient: _

Research/Study conducting Doctor's signature: _

Name of Doctor:

Dr. Tarun Kumar R (Principal investigator)

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

"ಅಲ್ಟ್ರಾಸೌಂಡ್ ಗೈಡೆಡ್ ಸುಪ್ರಾಕ್ಲಾವಿಕ್ಯುಲರ್ ಬ್ರಾಚಿಯಲ್ ಪ್ಲೆಕ್ಸಸ್ ಬ್ಲಾಕ್ ಅನ್ನು ಕ್ಲೋನಿಡೈನ್ ವರ್ಸಸ್ ಡೆಕ್ಸ್ಮೆಡೆಟೊಮಿಡಿನ್ ಅನ್ನು ರೋಪಿವಕೈನ್‌ಗೆ ಸಹಾಯಕವಾಗಿ ಮೇಲಿನ ಅಂಗ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಗಳಲ್ಲಿ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ ನೋವು ನಿವಾರಕವಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ - ಎ ಯಾದೃಚ್ಛಿಕ ನಿಯಂತ್ರಣ ಪ್ರಯೋಗ."

ನಾನು, ವಯಸ್ಸು/ಲಿಂಗ , UHID ಹೊಂದಿದ್ದೇನೆ ಮೇಲಿನ ಅಂಗ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ಸಮಯದಲ್ಲಿ ಸುಪ್ರಾಕ್ಲಾವಿಕ್ಯುಲರ್ ಬ್ರಾಚಿಯಲ್ ಪ್ಲೆಕ್ಸಸ್ ಬ್ಲಾಕ್‌ನಲ್ಲಿ ರೋಪಿವಕೈನ್‌ಗೆ ಸಹಾಯಕವಾಗಿ ಡೆಕ್ಸ್ಮೆಡೆಟೊಮಿಡಿನ್ ಅಥವಾ ಕ್ಲೋನಿಡಿನ್ ಅಧ್ಯಯನದ ಉದ್ದೇಶದ ಬಗ್ಗೆ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ. ಸಂವೇದನಾ ಮತ್ತು ಮೋಟಾರು ದಿಗ್ಬಂಧನದ ಪ್ರಾರಂಭ ಮತ್ತು ಅವಧಿಗೆ ಸಂಬಂಧಿಸಿದಂತೆ ರೋಪಿವಕೈನ್‌ಗೆ ಸಹಾಯಕವಾಗಿ ಕ್ಲೋನಿಡಿನ್ ಅಥವಾ ಡೆಕ್ಸ್ಮೆಡೆಟೊಮಿಡಿನ್‌ನ ಪರಿಣಾಮಕಾರಿತ್ವವನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲು, ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ ನೋವು ನಿವಾರಕ ಅವಧಿ.

ಆದ್ದರಿಂದ, ಈ ಮೂಲಕ ನಾನು ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯನ್ನು ನೀಡುತ್ತೇನೆ.

ಹೈಪೊಟೆನ್ಷನ್ ಮತ್ತು ಬ್ರಾಡಿಕಾರ್ಡಿಯಾದಂತಹ ಡೆಕ್ಸ್ಮೆಡೆಟೊಮಿಡಿನ್ ಮತ್ತು ಕ್ಲೋನಿಡಿನ್‌ನ ಅಡ್ಡಪರಿಣಾಮಗಳ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ, ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಗೆ ಮುನ್ನ ರೋಗಿಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಒಳಗೊಂಡಿರುವ ಸ್ವಭಾವ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನ್ನ ತಿಳುವಳಿಕೆ ಮತ್ತು ತೃಪ್ತಿಗೆ ವಿವರಿಸಲಾಗಿದೆ. ನಡೆಸುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಯಾವುದೇ ರೀತಿಯ ಪಾವತಿ/ಖರೀದಿ ಅಗತ್ಯವಿದ್ದಲ್ಲಿ ಅದನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ತನಿಖಾಧಿಕಾರಿಯೇ ಭರಿಸಬೇಕಾಗುತ್ತದೆ ಎಂದು ನಾನು ಅಧ್ಯಯನದ ಉದ್ದಕ್ಕೂ ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಅಧ್ಯಯನದ ಉದ್ದಕ್ಕೂ ನನಗೆ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚವನ್ನು ವಿಧಿಸಲಾಗುವುದಿಲ್ಲ.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವರಾಗಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸುತ್ತೇನೆ. ನನ್ನ ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಕಾರ್ಯವಿಧಾನಕ್ಕೆ

ಒಳಗಾಗಲು, ತನಿಖೆಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳಿಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ನನ್ನ ಸಂಪೂರ್ಣ ಮಾನ್ಯ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ಶೈಕ್ಷಣಿಕ ಮತ್ತು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ, ಕಾರ್ಯಾಚರಣೆ / ಕಾರ್ಯವಿಧಾನ ಇತ್ಯಾದಿಗಳನ್ನು ವೀಡಿಯೊಗ್ರಾಫ್ ಮಾಡಬಹುದು ಅಥವಾ ಛಾಯಾಚಿತ್ರ.

ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಬಹುದು. ಕಾರ್ಯವಿಧಾನ / ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಅಹಿತಕರ ಪರಿಣಾಮಗಳಿಗೆ ನಾನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳನ್ನು ಹೊಣೆಗಾರರನ್ನಾಗಿ ಮಾಡುವುದಿಲ್ಲ.

ಈ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆ ನಮೂನೆಯ ಪ್ರತಿಯನ್ನು ಮತ್ತು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಭಾಗವಹಿಸುವವರಿಗೆ ಒದಗಿಸಲಾಗಿದೆ.

ರೋಗಿಯ ಸಮ್ಮತಿ (ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು):

ರೋಗಿಯ ಹೆಸರು:

ಸಾಕ್ಷಿ 1 (ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು):_

ಸಾಕ್ಷಿಯ ಹೆಸರು 1:_

ರೋಗಿಗೆ ಸಂಬಂಧ:_

ಸಂಶೋಧನೆ/ಅಧ್ಯಯನ ನಡೆಸುವ ವೈದ್ಯರ ಸಹಿ: _

ವೈದ್ಯರ ಹೆಸರು:

ಡಾ. ತರುಣ್ ಕುಮಾರ್ ಆರ್ (ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ)

PATIENT INFORMATION SHEET

STUDY TITLE: "**Dexmeditomidine or clonidine for adjuvants to ropivacaine in supraclavicular brachial plexus block during upper limb surgery – a randomised control trial."**

STUDY SITE: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

This is to inform you that,

This study will be conducted on patients requiring upper limb surgeries without cardiac abnormalities/ neurological diseases/complication and matching inclusion criteria, undergoing neuraxial anaesthesia for elective surgeries at R. L. Jalappa Hospital, Tamaka, Kolar are included in the study. Patients who meet exclusion criteria will be excluded from the study. This study aims to assess the efficacy of the block properties of ropivacaine utilising either clonidine or dexmedetomidine as a perineural adjuvant.

Patient and the attenders will be completely explained about the procedure being done under ultrasound guidance. Throughout the study if any form of payment/purchase is necessary, it will be completely borne by the investigator. No extra cost will charged for patient throughout the study.

Please read the above mentioned information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect required information and relevant history will be taken. The collected information will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. There is no compulsion to agree to

this study. The care you will get will not change if you do not wish to participate.

You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact:

Dr. Tarun Kumar R

(Principal investigator)

Contact number: 9900920007

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನ: ಸುಪ್ರಾಕ್ಲಾವಿಕ್ಯುಲರ್ ಬ್ರಾಚಿಯಲ್ ಪ್ಲೆಕ್ಸಸ್ ಬ್ಲಾಕ್ ಡುರಿಮ್ಸ್ ಮೇಲಿನ ಅಂಗ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯಲ್ಲಿ ರೋಪಿವಾಕೈನ್‌ಗೆ ಸಹಾಯಕವಾಗಿ ಡೆಕ್ಸ್‌ಮೆಡಿಟೊಮಿಡಿನ್ ಅಥವಾ ಕ್ಲೋನಿಡಿನ್ - ತುಲನಾತ್ಮಕ ಅಧ್ಯಯನ.

ತನಿಖಾಧಿಕಾರಿಗಳು: ಡಾ. ತರುಣ್ ಕುಮಾರ್ .ಆರ್. / ಡಾ ರವಿ ಎಂ

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್ ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರವು ಶ್ರೀ ದೇವರಾಜರ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು, ಟಮಕ, ಕೋಲಾರ.

ವಿವರಗಳು: ಈ ಅಧ್ಯಯನವನ್ನು ಹೃದಯ ವೈಪರೀತ್ಯಗಳು / ನರವೈಜ್ಞಾನಿಕ ಕಾಯಿಲೆಗಳು / ತೊಡಕುಗಳು ಮತ್ತು ಹೊಂದಾಣಿಕೆಯ ಸೇರ್ಪಡೆ ಮಾನದಂಡಗಳಿಲ್ಲದ ಮೇಲ್ಭಾಗದ ಅಂಗಗಳ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ಅಗತ್ಯವಿರುವ ರೋಗಿಗಳ ಮೇಲೆ ನಡೆಸಲಾಗುವುದು, ಆರ್. ಹೊರಗಿಡುವ ಮಾನದಂಡಗಳನ್ನು ಪೂರೈಸುವ ರೋಗಿಗಳನ್ನು ಅಧ್ಯಯನದಿಂದ ಹೊರಗಿಡಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನವು ಕ್ಲೋನಿಡೈನ್ ಅಥವಾ ಡೆಕ್ಸ್ಮೆಡೆಟೊಮಿಡಿನ್ ಅನ್ನು ಬಳಸಿಕೊಂಡು ರೋಪಿವಕೈನ್ನ ಬ್ಲಾಕ್ ಗುಣಲಕ್ಷಣಗಳ ಪರಿಣಾಮಕಾರಿತ್ವವನ್ನು ನಿರ್ಣಯಿಸುವ ಗುರಿಯನ್ನು ಹೊಂದಿದೆ.

ರೋಗಿಯು ಮತ್ತು ಹಾಜರಾದವರಿಗೆ ಅಲ್ಟ್ರಾಸೌಂಡ್ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಕಾರ್ಯವಿಧಾನದ ಬಗ್ಗೆ ಸಂಪೂರ್ಣವಾಗಿ ವಿವರಿಸಲಾಗುವುದು. ಅಧ್ಯಯನದ ಉದ್ದಕ್ಕೂ ಯಾವುದೇ ರೀತಿಯ ಪಾವತಿ/ಖರೀದಿ ಅಗತ್ಯವಿದ್ದರೆ, ಅದನ್ನು ತನಿಖಾಧಿಕಾರಿಯು ಸಂಪೂರ್ಣವಾಗಿ ಭರಿಸಬೇಕಾಗುತ್ತದೆ. ಅಧ್ಯಯನದ ಉದ್ದಕ್ಕೂ ರೋಗಿಗೆ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚವನ್ನು ವಿಧಿಸಲಾಗುವುದಿಲ್ಲ

ದಯವಿಟ್ಟು ಮೇಲಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದರೆ ನಾವು ಅಗತ್ಯವಿರುವ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ ಮತ್ತು ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳುತ್ತೇವೆ. ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿಯು ಬದಲಾಗುವುದಿಲ್ಲ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸಿದರೆ ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ಸಂಪರ್ಕಿಸಿ:

ಡಾ.ತರುಣ್ ಕುಮಾರ್ ಆರ್

(ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ)

ಸಂಪರ್ಕ ಸಂಖ್ಯೆ: 9900920007

MASTERCHART

MASTER CHART

"ULTRASOUND GUIDED SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK USING CLONIDINE VERSUS DEXMEDETOMIDINE AS ADJUVANTS TO ROPIVACAINE FOR POST-OPERATIVE ANALGESIA IN UPPER LIMB SURGERIES"

Sl No	Age (years)	Gender (M/F)	Weight (kg)	Height (cm)	BMI	ASA Grade (I/II)	Group (A/B)	Surgery Type	Surgery Duration (min)	Onset Time of Sensory Block (min)	Onset Time of Motor Block (min)	Duration of Sensory Block (min)	Duration of Motor Block (min)	Time to First Rescue Analgesia (min)	VAS Score at Rescue Analgesia	Number of Rescue Analgesics in 24h	Ramsay Sedation Score (max)	HR Baseline (bpm)	HR Minimum (bpm)	SBP Baseline (mmHg)	SBP Minimum (mmHg)	DBP Baseline (mmHg)	DBP Minimum (mmHg)	SpO2 Minimum (%)	Bradycardia (Y/N)	Hypotension (Y/N)	Nausea/Vomiting (Y/N)	Other Adverse Effects (text)
1	21	F	62.1	141.9	30.8	I	A	Left Ulna Fracture	75	8.1	9.8	573	450	551	5	5	2	83	74	125	119	70	66	97	N	N	N	None
2	25	M	60.8	181.5	18.5	I	A	Hand Surgery	73	10.2	11.8	517	526	649	3	4	2	69	64	120	104	81	71	97	N	N	N	None
3	27	M	62.5	160.8	24.2	I	A	Wrist Surgery	57	6.6	9.9	456	514	560	3	4	2	89	84	138	120	79	71	98	N	N	Y	None
4	31	F	61.6	158.3	24.6	I	A	Ulna Fracture	67	8.9	11.1	623	464	522	3	3	2	53	49	126	112	78	68	99	Y	N	Y	None
5	36	F	70.5	163.2	26.5	I	A	Ulna Fracture	115	9.4	11.1	565	554	660	5	2	3	85	77	128	119	69	65	97	N	N	N	None
6	31	M	70.9	158.3	28.3	II	A	Wrist Surgery	79	8.7	10.3	610	546	651	4	4	2	66	58	117	100	87	80	98	Y	N	N	None
7	23	F	75.5	159	29.9	I	A	Elbow Surgery	99	5.1	16	543	575	515	6	2	2	68	60	114	102	78	68	100	N	N	N	None
8	47	M	53.9	168.5	19	II	A	Elbow Surgery	98	11	11.2	542	532	646	6	3	2	70	62	119	113	80	74	97	N	N	N	Shivering
9	20	M	56.2	168.9	19.7	I	A	Wrist Surgery	109	8.2	5.9	584	522	626	6	4	2	84	76	123	110	84	74	97	N	N	N	None
10	44	M	81.7	177.3	26	I	A	Ulna Fracture	111	9.4	13.6	525	508	606	5	4	2	88	76	126	111	78	70	100	N	N	N	None
11	57	M	79.4	171.1	27.1	I	A	Elbow Surgery	110	8.9	13.5	542	472	573	5	4	3	82	73	129	111	87	82	100	N	N	N	None
12	23	M	62.9	177.4	20	II	A	Ulna Fracture	89	10.4	14.4	561	583	585	4	2	2	83	77	101	95	76	65	97	N	N	N	None
13	40	M	53.1	179.9	16.4	I	A	Elbow Surgery	85	9.1	9.7	483	503	611	6	3	2	85	76	129	114	81	74	100	N	N	Y	None
14	53	M	89	159.7	34.9	II	A	Elbow Surgery	57	11.2	9.2	539	443	619	4	4	2	94	85	135	123	78	67	97	N	N	N	None
15	31	F	59.4	156.4	24.3	I	A	Hand Surgery	107	8.5	9.2	502	528	541	3	2	2	71	63	119	103	69	62	97	N	N	N	None
16	20	F	57.8	162	22	I	A	Wrist Surgery	82	9.5	13.7	522	582	541	4	3	2	69	62	153	143	72	65	100	N	N	N	None
17	40	F	73.3	146.4	34.2	I	A	Elbow Surgery	54	9.3	15.6	454	458	594	3	2	2	68	61	129	117	83	76	100	N	N	N	None
18	49	M	65.6	165.1	24.1	I	A	Elbow Surgery	75	8.8	8.9	555	475	449	3	2	2	76	68	135	127	85	76	99	N	N	N	None
19	57	F	68.6	161.7	26.2	II	A	Hand Surgery	120	11.2	16	502	543	625	4	5	2	64	56	134	120	80	70	100	Y	N	N	None
20	21	F	75.2	149.4	33.7	II	A	Ulna Fracture	66	8.4	8.5	474	441	635	5	5	2	65	57	133	115	87	78	100	Y	N	N	None
21	21	M	60.2	171.4	20.5	I	A	Elbow Surgery	108	5	13.6	578	472	508	4	5	3	67	63	132	120	76	66	97	N	N	N	None
22	22	M	45.1	172.7	15.1	I	A	Ulna Fracture	117	8.6	12.1	614	400	543	6	2	2	79	69	144	134	67	63	100	N	N	N	None
23	44	M	45.6	177.6	14.5	I	A	Ulna Fracture	73	12.4	10.3	594	461	630	6	5	2	75	66	122	107	69	61	98	N	N	N	None
24	46	M	62.3	170.4	21.5	I	A	Ulna Fracture	46	7.1	9.1	572	530	658	6	2	2	76	69	126	114	92	84	100	N	N	N	None
25	29	F	54.9	156.8	22.3	II	A	Wrist Surgery	97	9.8	15.5	514	519	547	5	2	2	67	61	127	110	82	73	99	N	N	N	None
26	34	M	56.7	160.9	21.9	I	A	Radius Fracture	78	6	10.7	558	522	527	4	3	2	84	75	129	117	75	70	100	N	N	Y	None
27	49	M	67	168.1	23.7	I	A	Wrist Surgery	109	12	14.1	586	621	635	5	5	2	82	72	126	112	78	67	98	N	N	N	None
28	59	F	67.5	159.5	26.5	I	A	Radius Fracture	59	8.3	12.4	576	491	503	5	4	2	66	56	110	94	69	64	97	Y	N	N	None
29	60	M	45.3	166.2	16.4	I	A	Hand Surgery	86	6.4	13.9	591	443	647	3	3	2	77	67	138	123	80	68	98	N	N	N	None
30	54	F	78.7	160.8	30.4	II	A	Ulna Fracture	68	5.7	13.6	586	436	587	6	2	2	70	66	128	114	84	76	97	N	N	N	None
31	45	M	65.2	156.3	26.7	I	A	Radius Fracture	120	10.2	12	564	441	560	5	5	2	83	78	115	107	76	67	96	N	N	N	None
32	24	M	72.7	168	25.8	II	A	Elbow Surgery	58	10.4	13.5	591	505	687	5	5	2	73	69	127	118	84	79	97	N	N	N	None
33	37	M	44	155.5	18.2	I	A	Radius Fracture	85	8.6	12.1	609	526	599	4	4	2	85	77	94	88	80	76	98	N	Y	N	None
34	46	M	55.5	170.7	19	I	A	Wrist Surgery	106	7.7	15.6	524	460	548	5	5	2	75	68	124	110	85	76	99	N	N	N	None
35	59	M	74.3	173.4	24.7	II	A	Elbow Surgery	66	8.9	10.8	612	496	630	5	2	2	69	62	142	130	74	64	98	N	N	N	None
36	46	M	55.6	162.9	21	I	A	Wrist Surgery	113	9.6	12	577	475	579	3	5	2	70	64	115	99	68	60	98	N	N	N	None
37	24	M	69.1	154.7	28.9	II	A	Ulna Fracture	110	12.1	14.9	579	491	493	3	4	2	80	71	139	119	88	76	99	N	N	N	None
38	19	F	62.3	165.1	22.9	I	A	Radius Fracture	99	12.1	13.5	503	448	575	6	4	3	89	79	112	100	83	76	99	N	N	N	None
39	31	M	48.1	176	15.5	I	A	Wrist Surgery	74	9.3	16	542	493	539	4	3	2	93	80	113	105	86	75	98	N	N	N	None

Sl No	Age (years)	Gender (M/F)	Weight (kg)	Height (cm)	BMI	ASA Grade (I/II)	Group (A/B)	Surgery Type	Surgery Duration (min)	Onset Time of Sensory Block (min)	Onset Time of Motor Block (min)	Duration of Sensory Block (min)	Duration of Motor Block (min)	Time to First Rescue Analgesia (min)	VAS Score at Rescue Analgesia	Number of Rescue Analgesics in 24h	Ramsay Sedation Score (max)	HR Baseline (bpm)	HR Minimum (bpm)	SBP Baseline (mmHg)	SBP Minimum (mmHg)	DBP Baseline (mmHg)	DBP Minimum (mmHg)	SpO2 Minimum (%)	Bradycardia (Y/N)	Hypotension (Y/N)	Nausea/Vomiting (Y/N)	Other Adverse Effects (text)
40	50	F	88.1	178.9	27.5	I	A	Radius Fracture	53	10.8	10.3	509	522	497	5	3	2	76	68	123	110	82	72	100	N	N	N	None
41	24	F	48.6	172.2	16.4	II	A	Hand Surgery	98	11.3	8.4	561	520	471	4	4	2	69	62	127	109	67	63	94	N	N	N	None
42	51	M	75.1	166.1	27.2	I	A	Radius Fracture	100	10.5	11.5	603	463	654	5	5	2	74	66	145	133	77	69	98	N	N	N	None
43	48	F	55.1	157.4	22.2	I	A	Radius Fracture	94	10.3	9.7	563	444	485	3	2	3	76	70	133	120	75	71	98	N	N	N	None
44	55	F	80.3	147.5	36.9	I	A	Radius Fracture	107	10.9	9.7	610	481	617	5	4	2	80	71	124	112	64	57	97	N	N	N	None
45	22	F	60.8	161.3	23.4	I	A	Elbow Surgery	100	8.4	13.4	470	514	601	4	4	3	88	83	124	108	75	68	96	N	N	N	None
46	32	F	79.1	166.8	28.4	II	A	Elbow Surgery	68	8	10.8	521	504	624	4	5	2	76	65	113	102	69	62	100	N	N	N	Dizziness
47	50	M	54	173	18	I	A	Elbow Surgery	77	7.6	12.4	607	528	543	6	2	2	81	69	129	118	80	75	100	N	N	N	None
48	60	M	88.2	147.3	40.7	I	A	Hand Surgery	58	8.5	13.4	544	587	530	3	3	2	70	66	118	103	75	67	97	N	N	N	None
49	36	M	65.6	161.5	25.2	I	A	Hand Surgery	118	11.8	13.2	477	428	590	3	2	2	83	75	114	99	86	77	100	N	N	N	None
50	56	M	59.4	161	22.9	I	A	Wrist Surgery	59	11.3	10.1	559	526	552	6	4	2	84	78	112	101	78	67	100	N	N	N	None
51	19	M	58.1	177.4	18.5	I	A	Hand Surgery	83	10.7	16.7	561	517	531	6	2	2	88	75	114	104	82	77	99	N	N	N	None
52	34	M	59.6	175.4	19.4	I	A	Radius Fracture	82	10.7	14	529	459	572	4	5	2	81	75	139	120	69	59	99	N	N	N	None
53	45	F	75	149.7	33.5	I	A	Elbow Surgery	83	9.9	11.3	590	449	674	4	4	2	66	58	132	121	88	77	100	Y	N	N	None
54	32	M	75.1	180.4	23.1	II	B	Wrist Surgery	79	6.3	7.2	735	598	819	4	3	2	75	70	127	117	84	79	98	N	N	N	None
55	34	F	67.6	160.4	26.3	I	B	Ulna Fracture	115	6.2	11.5	731	594	762	6	3	4	78	72	128	110	80	70	97	N	N	N	None
56	30	M	63	164.6	23.3	II	B	Radius Fracture	57	5.5	6.8	629	589	559	4	3	3	68	63	128	119	78	69	100	N	N	N	None
57	30	M	88.2	177.7	27.9	I	B	Wrist Surgery	49	7.8	10.3	682	644	808	4	2	3	82	75	114	100	68	59	100	N	N	N	None
58	25	M	46	170	15.9	II	B	Elbow Surgery	116	7.2	8.9	652	692	770	4	1	2	64	57	129	110	80	73	99	Y	N	N	None
59	44	F	75.8	153.4	32.2	I	B	Wrist Surgery	99	7.4	11.4	720	549	791	4	3	3	97	89	120	110	90	79	97	N	N	N	None
60	55	M	50.6	171	17.3	II	B	Elbow Surgery	112	7.9	9.8	681	649	667	3	2	3	88	81	130	119	87	81	100	N	N	N	None
61	45	F	89.9	153	38.4	I	B	Wrist Surgery	86	7.6	7.7	721	556	754	3	3	2	73	68	111	103	69	64	97	N	N	N	None
62	54	M	53.8	188.5	15.1	I	B	Wrist Surgery	112	9.7	8.4	629	614	556	5	2	2	73	66	126	116	79	73	100	N	N	N	None
63	45	F	59.7	167.6	21.3	I	B	Radius Fracture	57	5.9	9.4	695	635	736	3	2	2	69	62	127	112	83	74	97	N	N	N	None
64	60	F	61.3	152.8	26.3	I	B	Radius Fracture	102	7.9	6.3	647	621	659	6	2	2	89	81	114	98	82	72	98	N	N	N	None
65	32	M	44	159.3	17.3	II	B	Elbow Surgery	54	5.2	11.7	638	592	683	4	2	3	81	76	132	112	79	71	99	N	N	N	None
66	29	M	40.3	158.7	16	I	B	Ulna Fracture	62	4.6	5.7	667	603	663	3	2	4	73	68	123	105	81	75	97	N	N	N	None
67	26	M	42.8	167.8	15.2	I	B	Radius Fracture	94	5.6	9.2	607	647	770	3	1	3	88	79	133	126	78	71	99	N	N	N	None
68	33	F	50.6	159.6	19.9	I	B	Hand Surgery	112	7.1	8.2	659	599	764	5	2	2	77	66	124	116	84	76	97	N	N	N	None
69	40	M	33.9	173.3	11.3	II	B	Elbow Surgery	61	4.6	8.9	638	572	724	5	1	3	87	75	136	121	85	73	94	N	N	N	None
70	38	M	68.3	161.9	26.1	I	B	Ulna Fracture	89	7.2	9.3	727	600	574	4	1	3	67	58	144	133	81	75	99	Y	N	N	None
71	34	M	58.1	163.6	21.7	I	B	Ulna Fracture	102	7.7	6.9	635	578	611	3	2	2	83	71	109	95	84	77	98	N	N	N	None
72	26	F	54.7	163.5	20.5	I	B	Elbow Surgery	99	6.3	10.3	717	630	738	5	3	2	73	69	120	107	78	68	98	N	N	N	None
73	55	F	78.5	141.9	39	I	B	Ulna Fracture	55	7	12.8	628	666	725	4	2	3	69	60	125	112	84	74	97	N	N	N	None
74	25	M	70.5	164.2	26.1	I	B	Ulna Fracture	96	7.7	5.3	674	627	780	4	2	2	82	72	104	93	74	65	98	N	N	N	None
75	37	M	81.6	170.8	28	II	B	Wrist Surgery	118	7.1	7.8	632	501	695	6	2	2	68	62	117	104	79	69	99	N	N	N	None
76	25	M	69.4	178.5	21.8	I	B	Radius Fracture	101	10.1	11.2	670	543	678	5	1	3	76	67	107	99	76	66	94	N	N	N	None
77	32	F	62.8	157.6	25.3	II	B	Wrist Surgery	90	7	7.2	715	606	773	4	3	2	72	67	118	108	87	78	97	N	N	N	None
78	54	F	47	159.2	18.5	II	B	Wrist Surgery	57	10.4	10.5	692	574	667	5	1	2	80	69	134	117	81	75	98	N	N	N	None

Sl No	Age (years)	Gender (M/F)	Weight (kg)	Height (cm)	BMI	ASA Grade (I/II)	Group (A/B)	Surgery Type	Surgery Duration (min)	Onset Time of Sensory Block (min)	Onset Time of Motor Block (min)	Duration of Sensory Block (min)	Duration of Motor Block (min)	Time to First Rescue Analgesia (min)	VAS Score at Rescue Analgesia	Number of Rescue Analgesics in 24h	Ramsay Sedation Score (max)	HR Baseline (bpm)	HR Minimum (bpm)	SBP Baseline (mmHg)	SBP Minimum (mmHg)	DBP Baseline (mmHg)	DBP Minimum (mmHg)	SpO2 Minimum (%)	Bradycardia (Y/N)	Hypotension (Y/N)	Nausea/Vomiting (Y/N)	Other Adverse Effects (text)
79	58	M	62.3	166.2	22.6	II	B	Radius Fracture	52	6.2	11.1	647	586	788	3	2	2	78	71	122	108	88	83	100	N	N	N	None
80	30	F	46.5	155.4	19.3	I	B	Elbow Surgery	83	4.9	7.9	685	580	664	4	1	2	82	78	112	101	79	72	99	N	N	N	None
81	21	M	80.1	169.5	27.9	I	B	Radius Fracture	61	6.1	8.2	600	672	801	4	1	3	72	68	136	128	81	76	98	N	N	N	None
82	59	M	73.3	176.4	23.6	I	B	Hand Surgery	71	5.8	6.1	590	605	691	6	1	2	89	81	129	121	77	68	99	N	N	N	Dizziness
83	23	M	79.1	162.1	30.1	I	B	Ulna Fracture	85	6.6	10	689	590	621	6	3	2	86	75	109	99	81	76	97	N	N	N	None
84	21	F	60.3	164.3	22.3	II	B	Hand Surgery	73	5.6	4.4	632	584	778	5	3	2	76	67	126	119	68	65	98	N	N	N	None
85	53	M	77.9	166.6	28.1	I	B	Elbow Surgery	110	6	6.9	602	563	514	3	1	3	65	58	124	117	69	61	98	Y	N	N	None
86	19	M	40.8	173.7	13.5	I	B	Ulna Fracture	97	5.2	7.5	753	649	790	3	1	2	69	62	129	120	71	61	98	N	N	Y	None
87	32	M	46.7	178.4	14.7	I	B	Ulna Fracture	82	5.8	6.8	588	607	694	3	3	3	87	81	139	125	76	69	99	N	N	N	None
88	39	M	68.2	168.3	24.1	I	B	Radius Fracture	119	5.8	11	710	523	634	6	1	3	82	73	122	111	75	67	98	N	N	N	None
89	40	M	52.3	175.5	17	I	B	Hand Surgery	71	7.3	10.6	635	541	682	5	1	2	86	80	133	121	69	64	100	N	N	N	None
90	26	M	56.8	166.6	20.5	II	B	Radius Fracture	113	10.8	10.4	723	594	641	4	3	2	78	73	126	119	77	68	99	N	N	N	None
91	36	F	59.6	158.6	23.7	I	B	Radius Fracture	96	6.1	9.9	638	668	801	6	3	2	74	64	112	100	71	63	100	N	N	N	None
92	22	M	58.3	165.9	21.2	I	B	Elbow Surgery	115	7.1	8.6	617	608	723	6	1	2	70	64	120	107	83	78	97	N	N	N	None
93	25	M	60.7	157.8	24.4	I	B	Wrist Surgery	113	9.5	4.4	706	603	774	3	1	2	75	65	132	123	77	71	100	N	N	N	None
94	30	M	61.2	174.1	20.2	I	B	Ulna Fracture	104	9.1	6.8	719	569	647	3	2	4	73	67	121	105	84	72	98	N	N	N	None
95	44	M	85.6	166.6	30.8	I	B	Elbow Surgery	67	7.2	6.9	622	615	656	3	3	3	77	67	115	105	70	63	100	N	N	N	None
96	41	F	85.2	154.6	35.6	I	B	Radius Fracture	54	8.9	8.4	667	637	749	4	2	3	70	62	129	113	79	72	99	N	N	N	None
97	26	M	65.2	177.4	20.7	I	B	Wrist Surgery	57	6.4	9.9	684	491	748	3	1	2	74	67	134	127	80	73	99	N	N	Y	Shivering
98	33	F	53.3	169	18.7	I	B	Hand Surgery	51	6.8	8.9	710	565	733	5	1	3	76	65	152	137	86	78	99	N	N	N	None
99	32	F	51.1	165.8	18.6	II	B	Wrist Surgery	113	8.1	8.2	645	523	864	4	1	3	82	76	116	103	71	67	94	N	N	N	None
100	33	M	65.6	179.8	20.3	II	B	Radius Fracture	79	8.4	11.1	677	585	747	3	3	3	83	73	118	104	74	69	98	N	N	N	None
101	25	M	74.4	174.3	24.5	I	B	Wrist Surgery	70	5.3	8.7	668	561	749	4	1	3	85	78	128	121	86	73	97	N	N	N	None
102	49	F	57.6	147.6	26.4	I	B	Radius Fracture	58	6.9	12.2	708	552	830	6	1	2	88	75	131	122	86	79	98	N	N	N	None
103	46	M	57.7	183.9	17.1	II	B	Hand Surgery	52	8	10.5	687	634	641	6	3	3	78	70	101	90	85	76	97	N	N	N	None
104	24	M	61.5	176.2	19.8	I	B	Hand Surgery	83	6	7.7	696	586	835	5	3	2	83	72	120	105	78	68	100	N	N	N	None
105	25	M	73.5	155.3	30.5	I	B	Radius Fracture	103	6.3	9.3	636	597	718	5	3	4	77	72	119	110	73	68	97	N	N	N	None
106	21	M	69.7	179	21.8	I	B	Wrist Surgery	99	5.1	6.2	663	571	733	3	1	2	82	75	121	112	75	64	99	N	N	N	None