A STUDY TO COMPARE THE ANALGESIC EFFICACY OF DEXAMETHASONE AND DEXMEDETOMIDINE AS AN ADJUVANT TO BUPIVACAINE FOR BILATERAL SUPERFICIAL CERVICAL PLEXUS BLOCK IN PATIENTS UNDERGOING THYROID SURGERIES – A RANDOMISED CLINICAL TRIAL

By

DR. VANISHREE DESHPANDE

Dissertation submitted to

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In partial fulfilment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

ANESTHESIOLOGY

Under the guidance of

Dr. VIJAY. V. KATTI

PROFESSOR

DEPARTMENT OF ANESTHESIOLOGY

B.L.D.E. (Deemed to be University)

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE, VIJAYAPUR

KARNATAKA

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DATE: 01-07-2024

PLACE: VIJAYAPUR

DR. VANISHREE DESHPANDE
DEPARTMENT OF ANESTHESIOLOGY
B.L.D.E (DEEMED TO BE) UNIVERSITY
SHRI B. M. PATIL MEDICAL COLLEGE
HOSPITAL AND RESEARCH CENTRE,
VIJAYAPUR, KARNATAKA

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DATE: 01-07-2024
PLACE: VIJAPURA

DR. VIJAY. V. KATTI
PROFESSOR
DEPARTMENT OF ANESTHESIOLOGY
B.L.D.E (DEEMED TO BE) UNIVERSITY
SHRI B. M. PATIL MEDICAL COLLEGE
HOSPITAL AND RESEARCH CENTRE,
VIJAYAPUR, KARNATAKA

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DATE: 01-07-2024
PLACE: VIJAYAPUR

DR. RENUKA HOLYACHI
HEAD OF THE DEPARTMENT
DEPARTMENT OF ANESTHESIOLOGY
B.L.D.E (DEEMED TO BE) UNIVERSITY
SHRI B. M. PATIL MEDICAL COLLEGE
HOSPITAL AND RESEARCH CENTRE,
VIJAYAPUR, KARNATAKA

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DATE: 01-07-2024
PLACE: VIJAYAPUR

DR. ARAVIND PATIL
PRINCIPAL
DEPARTMENT OF ANESTHESIOLOGY
B.L.D.E (DEEMED TO BE) UNIVERSITY
SHRI B. M. PATIL MEDICAL COLLEGE
HOSPITAL AND RESEARCH CENTRE,
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B.L.D.E (DEEMED TO BE) UNIVERSITY
SHRI B. M. PATIL MEDICAL COLLEGE
HOSPITAL AND RESEARCH CENTRE,
VIJAYAPUR, KARNATAKA

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ABBREVIATIONS:

ASA- American Society of Anaesthesiologists

BSCPB- bilateral superficial cervical plexus block

VAS- visual analogue score

SBP- Systolic blood pressure

DBP- Diastolic blood pressure

MAP- Mean arterial pressure

HR- Heart rate

PONV- Post operative nausea vomiting

ETT- endotracheal tube

US- ultrasound, USG- ultrasonogram

GA- general anaesthesia

NRS- numerical rating scale

H- hour

Min- minutes

SCM- sternocleidomastoid

LA- local anaesthetic

mg- milligram

mcg- microgram

Kg-kilogram

ACLS- advanced cardiac life support

IV- intravenous

IM- intramuscular

S.D- standard deviation

ECG- electrocardiogram

GRE- glucocorticoid response elements

L- litres

ml- millilitres

ICU- intensive care unit

CVS- cardiovascular system

CNS- central nervous system

NIBP- Non-invasive blood pressure

SPO2: oxygen saturation

ABSTRACT

BACKGROUND AND AIMS:

Thyroid surgeries being one of the most common endocrine surgical procedures carried out throughout the world. Pain control is one of the many challenges faced by the perioperative physicians in post thyroid surgeries patients which when untreated proceeds to become chronic pain. The goal in the initial postoperative period is to provide good analgesia and better quality of recovery along with eliminating the side effects of systemic analgesics. Regional anaesthesia techniques have become a popular tool in achieving this goal. BSCPB is one of the simple and easy locoregional techniques used in managing pain in post-thyroid surgery patients.

This study aims at comparing the analgesic efficacy of dexmedetomidine and dexamethasone as an adjuvant with bupivacaine for BSCPB in patients undergoing thyroid surgeries.

METHODOLOGY:

- Written informed consent obtained.
- Nil by mouth status confirmed.
- IV access was secured 20 Gauge cannula.
- Patients underwent thorough Pre-anaesthetic evaluation with detailed history, airway examination, systemic examination. Patient was explained about the BSCPB procedure and sensitized about Visual analogue scale. Routine blood investigations were done along with thyroid profile.

- General anaesthesia was given. Before the incision, BSCPB was given with 10ml 0.5% bupivacaine either with 50mcg dexmedetomidine in Group A or 8mg dexamethasone in Group B. Patients were monitored for 24 hours for postoperative pain.

RESULTS:

- Age and gender were comparable and statistically insignificant.
- Intraoperative hemodynamic parameters (SBP, DBP, MAP, HR) monitored at specific time intervals were significantly lower in Group A.
- VAS scores were significantly better up to 8 hours in Group A.
- The time taken for first analgesic dose request is significantly longer in Group A than Group B.
- The total postoperative analgesic consumption was significantly lower in Group A than in Group B.
- Group B had significantly lower incidence of postoperative nausea and vomiting.

CONCLUSION:

In conclusion, with all the above findings which are statistically significant, Dexmedetomidine performs better than dexamethasone as an adjuvant to bupivacaine for BSCPB for post operative analgesia in patients undergoing thyroid surgeries.

KEYWORDS: Bilateral superficial cervical plexus block, Dexmedetomidine, Dexamethasone, Bupivacaine, PONV.

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INTRODUCTION

"For all the happiness mankind can gain is not in pleasure but in rest from pain"
-John Dryden.

Surgical interventions carried out to reduce human suffering results in inevitable consequences such as pain and distress to the patient. Controlling acute pain that follows tissue injury after surgery is important in the immediate postoperative period as well as in preventing chronic postsurgical pain, which can develop in as many as 10% of patients.[1]

Ineffective pain management can result in negative clinical and psychological outcomes such as restlessness causing hypoxemia, coronary ischemia, myocardial infarction, poor wound healing, insomnia, decreased quality of life and demoralization which further increases morbidity and mortality.[2]

Thyroidectomy is one of the common surgical procedures performed for various thyroid conditions that causes mild to moderate pain in the first 24 h after surgery hence requires adequate postoperative pain relief to augment patient recovery and satisfaction.[3,4]

Thyroid surgeries are generally carried out under general anaesthesia requiring relatively deeper anaesthesia due to combined effects of surgery and frequent tracheal stimulation due to movements of ETT during surgery and can lead to complications such as discomfort while swallowing, sore throat, nausea and vomiting along with pain.[5]

Management of postoperative pain is usually by either administration of nonsteroidal anti-inflammatory drugs which may be ineffective in pain relief and increase the risk of postoperative bleeding or opioids which have side effects like nausea, vomiting, sedation and respiratory depression worsening the clinical condition of the patient.[6]

Loco-regional methods of anaesthesia such as local wound infiltration, bilateral superficial cervical plexus block (BSCPB), bilateral combined superficial and deep cervical plexus block can alleviate the post operative pain and prevent sensitization of the central and peripheral nervous system due to longer duration of action thus preventing development of chronic pain without the side effects of systemic analgesics and superior patient satisfaction. [7, 8, 9]

The introduction of ultrasound (US) guidance in anaesthesia has permitted an indirect vision of internal structures (muscle, vessels, nerves) and ultrasonography has become an indispensable tool for anaesthesiologist and a gold standard for truncal and peripheral blocks, as recommended by several international guidelines.

Recently introduced alpha-2 agonist which is highly selective is dexmedetomidine which has been evaluated as an adjuvant in peripheral nerve block is reported to be safe and effective in prolonging the action of the peripheral blocks. [10, 11]

Glucocorticoids have a prerequisite to bind to ligands within the cell and be transported into the nucleus, where they have their effect on DNA transcription and cause anti-inflammatory action. Dexamethasone is proven to potentiate the action of local anaesthetics through modulation of the function of potassium channels in the excitable cells which halts the transmission along with causes local vasoconstriction hence, prolong the duration of nerve blocks. [12, 13]

Our study thus, was aimed at evaluating the analgesic effect, duration of action of BSCPB, postoperative visual analogue scores and advantages along with complications of adding dexmedetomidine and dexamethasone to bupivacaine as adjuvants for the block in patients undergoing thyroid surgeries under general anaesthesia. Our study thus is, was aimed at evaluating the analgesic effect, duration of action of BSCPB, postoperative visual analogue scores and advantages along with complications of adding dexmedetomidine and dexamethasone to bupivacaine as adjuvants for the block in patients undergoing thyroid surgeries under general anaesthesia.

AIMS AND OBJECTIVES

Primary objective:

1. Assessment of postoperative pain by visual analogue scale (VAS) scores for both groups at 2, 6, 8, 10 and 12 hours after surgery, patient satisfaction score at 24h
2. Assessment of duration of block.

Secondary objective:

1. To Monitor intraoperative hemodynamic stability
2. To study requirement of total dose of rescue analgesia, time to first rescue analgesia
3. To study post operative nausea vomiting
4. To study any complications during first 24H of block

REVIEW OF LITERATURE

In a study conducted by Pham MQ *et al*. in 60 patients undergoing thyroid surgery divided into study group (received BSCPB) and control group (received normal saline) it was discovered that in the study group who received ultrasound guided BSCPB postoperative pain was alleviated up to 24 hours with reduced need for analgesics and decreased incidence of PONV. (4)

Similarly, in a trial carried out by Aweke Z *et al*. in 66 patients undergoing thyroid surgeries out of which 33 patients were administered with BSCPB after induction with 0.25% bupivacaine, it was found that BSCPB is an effective and useful method of postoperative analgesia for thyroid surgeries patients. They also advocated for BSCPB to be considered as a primary analgesic modality for such patients. (5)

Goulart TF *et al.* (2019) - this study included 100 patients undergoing total thyroidectomies with one group receiving only general anaesthesia and second group receiving general anaesthesia along with BSCPB. It was proved that GA with the said block is a safe and efficient method to control pain and to achieve better patient outcomes. (6)

In 162 patients posted for thyroid surgeries Shihi ML *et al.* studied the analgesic efficacy BSCPB. Patients were divided into 3 groups in which group A received normal saline, group B received 0.5% bupivacaine and group C received levobupivacaine. The study inferred that BSCPB brought down the need of general anaesthetics intraoperatively and remarkable reduced the postoperative pain severity for the first 24 H and shortened the hospital stay. (7)

A prospective double blinded study was conducted in 60 patients undergoing surgery for thyroid disorders by Santosh BS *et al*. The patients were randomized into 2 groups of 30 among which group A received 20ml of 0.5% ropivacaine and group B received 20ml of 0.5% ropivacaine with 0.5mcg/kg dexmedetomidine. Group B exhibited notably prolonged and higher quality pain relief in postoperative period than group A (11).

Kumar MS *et al*. - In their study which was done among 80 patients undergoing thyroid surgeries under general anaesthesia, divided randomly into 2 groups, receiving BSCPB with 0.25% bupivacaine 20ml with dexamethasone in Group A and plain local anaesthetic in Group B. It was concluded that dexamethasone

when added to local anaesthetic for the block it prolonged the duration of analgesia of the block and decreased PONV compared to bupivacaine alone. [15]

Woldegerima B *et al*. - their study including 74 patients assessed analgesic efficacy of BSCPB. Out of 74, half received the block with 10ml of 0.25% bupivacaine while the other half did not. BSCPB was recommended as easy, safe and effective mode of pain control for first 24 postoperative hours in patients who underwent thyroid surgeries as it decreases pain scores, reduces opioid requirement and lengthens the time for first analgesic dose. [16]

In a systemic review and meta-analysis carried out by Cai, Y *et al.* which included 18 studies with 1265 patients, it was observed that BSCPB significantly reduced VAS scores, pain in post-operative period, reduced opioid and antiemetic requirement and PONV incidence. [17]

In another study undertaken by Senapathi *et al*. in 36 patients divided into 2 groups of 18 having thyroidectomy procedure, compared ultrasound guided technique (US group) and landmark technique (LM group) of BSCPB. It was interpreted that ultrasound guided BSCPB was more effective than landmark technique in reducing pain scores in postoperative period as the VAS scores and

postoperative opioid required in US group were significantly low than in LM group. [18]

In a study that aimed at comparing the post operative effects of BSCPB with 0.75% ropivacaine and intravenous lidocaine at the dose of 1.5mg/kg for 10mins followed by 1.5mg/kg/hr in thyroidectomy patients by Yang X *et al*. Group N, Group L and Group C received the block, IV lidocaine and Normal saline respectively. All three groups were assessed for quality of recovery using QoR-40, NRS, hemodynamic stability, opioid requirement and adverse effects. It was observed that Group N had higher QoR-40 total scores compared to group L and C. group N had lower NRS scores and less changes in the hemodynamic parameters thus concluded that BSCPB improved the quality of recovery in patients recovering from thyroidectomies. [19]

In a systemic review conducted by Betancourt. C *et al.* in order to assess the postoperative analgesic effect of BSCPB, 34 RCTs were included which either compared block to placebo or block to no block. It was concluded that BSCPB reduced the analgesic requirement in first 24 hours and extended the period before first rescue dose of analgesia. [20]

In order to evaluate the effects of adding dexmedetomidine as adjuvant to levobupivacaine for BSCPB, El Bendary HM *et al.* carried out a trial including 80 patients who underwent tracheal stenosis repair, bifurcated randomly into 2 groups. Group L received 0.5% levobupivacaine plain 10ml while Group D received 0.5% bupivacaine 10 ml along with 0.5mcg/kg dexmedetomidine. The addition of dexmedetomidine resulted in significant reduction of fentanyl consumption in postoperative period in Group D than in Group L (p value< 0.001). It was observed dexmedetomidine also increased the duration of action of BSCPB. [21]

With the aim to assess the analgesic efficacy of BSCPB in patients undergoing various head and neck surgeries along with assessment of intraoperative and postoperative systemic analgesics requirement, total analgesia duration of the block, hemodynamic variations and complications if any, Patel. H *et al.* conducted a study in 60 patients divided randomly into 2 groups that is Group A and Group B undergoing mandibular surgeries, tympanomastoid and clavicular surgeries. Group A received GA with systemic analgesics and Group B was received GA and SCPB with 0.25% 10ml on each side. Observed results were that the intraoperative and postoperative analgesic requirement was higher in Group A than Group B. Group B was observed to have significantly longer duration of analgesia. Hence the conclusion was that SCPB can give better

perioperative analgesia along with reduction in the systemic analgesic requirement and side effects associated with them in various head and neck surgeries. (22)

Jain, Neena *et al.* in 2023 compared efficacy of dexmedetomidine via two routes, parenteral and perineural. They conducted the trial among 60 ASA I and II thyroidectomy patients belonging to age 18 to 65 years. Patients were divided into 2 groups randomly. Group A (n=30) was administered with BSCPB with 0.25% ropivacaine 10ml on each side along with IV infusion of dexmedetomidine at the dose of 0.5mcg/kg. Group B (n=30) received 10ml of 0.25% ropivacaine with dexmedetomidine as adjuvant for BSCPB on each side. It was observed that time for the first analgesic dose request for significantly prolonged (p value < 0.001) and total analgesic consumption was reduced (p value < 0.001) in Group B than in Group A. Hence the study concluded that perineural dexmedetomidine as adjuvant with ropivacaine is better for BSCPB. (23)

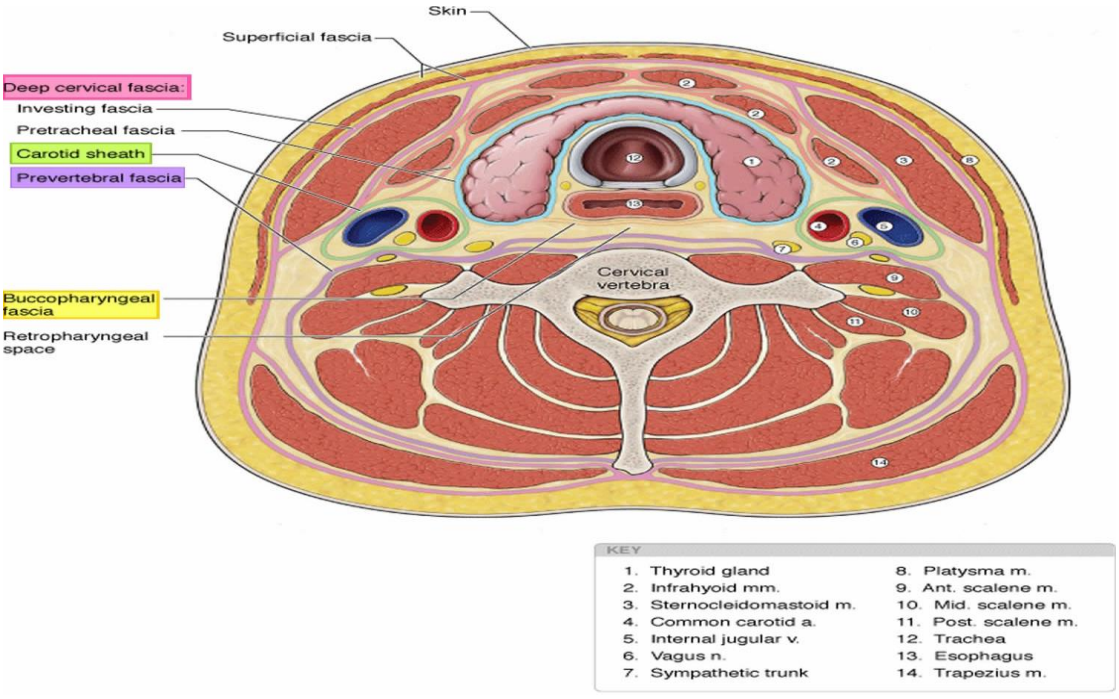
CLINICAL ANATOMY

CERVICAL FASCIA:

Cervical fascia is a resistant structure that consists of two layers namely superficial and deep as described initially by Burns [24].

Recently the cervical fascia has been described to have 2 layers namely superficial/subcutaneous and deep layers [25]. Deep layers are additionally divided into 3 layers. They are as follows:

a. Investing layer- also called masticator fascia, submandibular or sternocleidomastoid-trapezius fascia is the superficial layer
b. Strap muscle fascia or visceral fascia- middle layer.
c. The deep layer is the prevertebral fascia



FigFIG.1 – LAYERS OF CERVICAL FASCIA

NOMENCLATURE OF CERVICAL PLEXUS BLOCK [26]- is as follows:

a. Blocks given above the subcutaneous layer of deep cervical fascia are named as SUPERFICIAL or SUBCUTANEOUS CERVICAL PLEXUS BLOCK
b. Blocks given below the subcutaneous layer but above the prevertebral layer are named as INTERMEDIATE PLEXUS BLOCK- as suggested by Telford and Stoncham.
c. Blocks given deeper to prevertebral layer are called DEEP CERVICAL PLEXUS BLOCK

CLINICAL ANATOMY OF THYROID [27, 28, 29]

The thyroid is a H-shaped endocrine gland with a very rich blood supply. The gland extends from C5 to T1 vertebral levels in the anterior part of the neck.

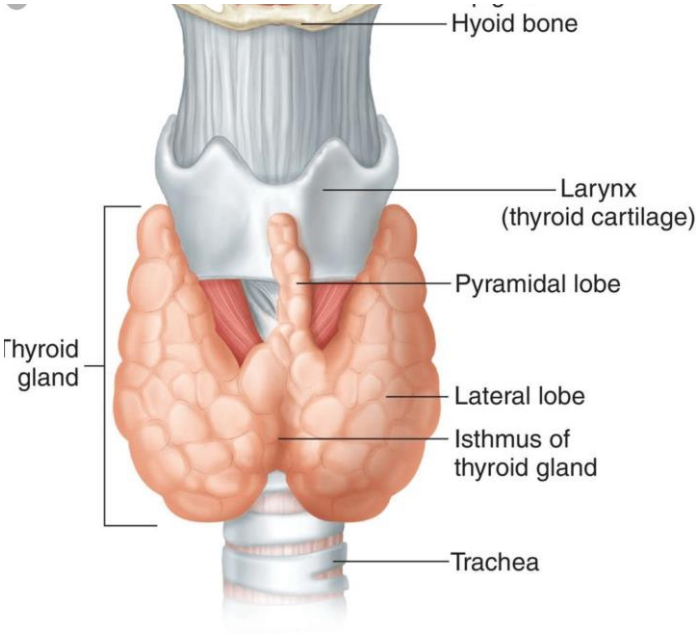
Weight of the gland: 15-20gm on average.

The gland is made of two lobes i.e. right and left lobes connected to each other by isthmus.

Dimensions of the lobe: 4x2x2 cms

Dimensions of isthmus: 2x2x2-6cm

Pyramidal lobe or also called Morgagni's or Lalouete's pyramid may be present in about 50% of the population.



FigFIG.2 – LOCATION OF THYROID GLAND

RELATIONS:

Deep cervical fascia surrounds the gland with its layers.

Anteriorly it is bounded by strap muscles. Anterio-laterally it is related to SCM muscle on each side.

The gland is divided into lobes and lobules by the septae formed by the tightly adherent true thyroid capsule which is also called visceral fascia.

Medially it is related to RLN, trachea, oesophagus.

Posterior-laterally it is related to vagus nerve, common carotid artery, internal jugular vein.

SENSORY LIGAMENT OF BERRY: it is the condensation of middle layer of the deep cervical fascia which is present posteriorly. This brings contact between thyroid lobes with cricoid cartilage and first two tracheal rings.

Parathyroid glands are located in the posterior surface of lateral lobes.

BLOOD SUPPLY:

Two or sometimes three arteries supply the thyroid gland.

1. Superior thyroidal artery- a branch of external carotid artery.
2. Inferior thyroidal artery- a branch of subclavian artery.
3. Thyroid IMA artery also called Neubauer's artery usually originates from common carotid.

VENOUS DRAINAGE:

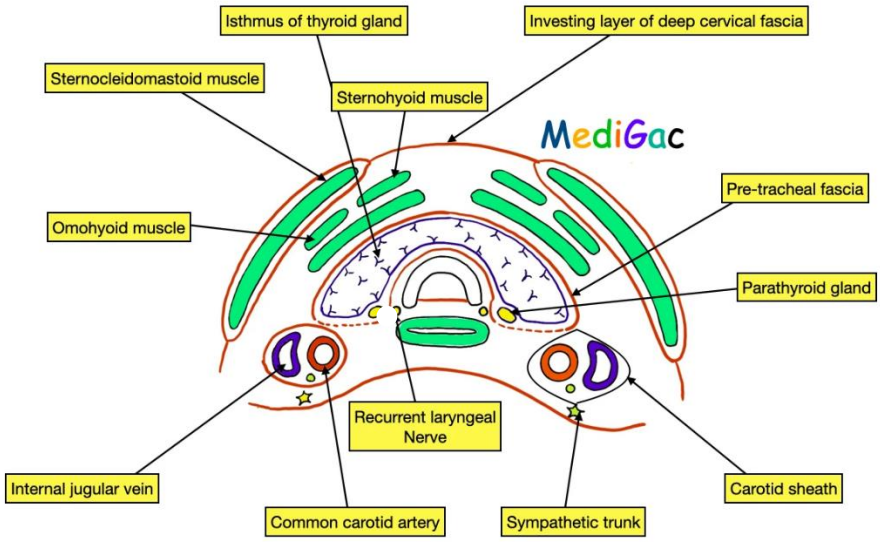
Thyroid is drained by 3 veins namely superior, middle and inferior thyroid veins.

Superior and middle thyroid veins drain into external jugular vein whereas inferior thyroid vein drains into brachiocephalic vein.

NERVE SUPPLY: Thyroid has autonomic nerve supply

Sympathetic supply is by cervical sympathetic chain

Parasympathetic supply is from Vagus.



FigFIG.3 – RELATIONS OF THYROID GLAND

FUNCTIONS OF THYROID GLAND:

The thyroid gland synthesizes and secretes thyroid hormones which play various roles in organ development and in the homeostatic control of fundamental physiological mechanisms such as body growth and energy expenditure.

Follicles are the functional units of the gland. Follicular cells, the major cell type, form a single layer epithelium that surrounds a central cavity which consists of thyroid hormone reserve. Other cells which are present interstitially are neuroendocrine cells, fibroblasts and other stromal cells.

The genes responsible for raising thermogenesis and metabolic rate are activated when the thyroid hormone attaches to its intranuclear receptor.

The thyroid hormones have their effect on all the organ systems of the body. The physiological effects of thyroid hormones are summarized below:

1. The basal metabolic rate is raised by thyroid hormone. It raises the body temperature, respiration rate, and oxygen consumption in several tissues via increasing the Na+/K+ ATPase gene expression. Lipid synthesis or lipolysis may be induced, depending on the state of metabolism. The anabolism of proteins and the metabolism of carbohydrates are both accelerated by thyroid hormones. In excessive amounts, thyroid hormones

can also cause the breakdown of proteins. Although they can enhance glucose oxidation, gluconeogenesis, glycogen synthesis, and reabsorption, thyroid hormones have little effect on blood glucose levels.

2. Catecholamines are influenced positively by thyroid hormones. In order to raise heart rate, stroke volume, cardiac output, and contractility, it enhances the expression of beta-receptors.
3. Thyroid hormones stimulate the respiratory centers and lead to increased oxygenation because of increased perfusion.
4. Thyroid hormones cause increased development of type II muscle fibers. These are fast-twitch muscle fibers capable of fast and powerful contractions.
5. In children, thyroid hormones act synergistically with growth hormone to stimulate bone growth. It induces chondrocytes, osteoblasts, and osteoclasts. Thyroid hormone also helps with brain maturation by axonal growth and the formation of the myelin sheath.

The physiological effects of thyroid hormones [31] are listed below:

- Increases the basal metabolic rate
- Depending on the metabolic status, it can induce lipolysis or lipid synthesis.
- Stimulate the metabolism of carbohydrates

- Anabolism of proteins. Thyroid hormones can also induce catabolism of proteins in high doses.
- Permissive effect on catecholamines
- In children, thyroid hormones act synergistically with growth hormone to stimulate bone growth.
- The impact of thyroid hormone on CNS is important. During the prenatal period, it is needed for the maturation of the brain. In adults, it can affect mood. Hyperthyroidism can lead to hyperexcitability and irritability. Hypothyroidism can cause impaired memory, slowed speech, and sleepiness.
- Thyroid hormone affects fertility, ovulation, and menstruation.

PHYSIOLOGY OF PAIN [30, 31, 32]

An unpleasant sensory and emotional experience which has association with an ongoing or potential tissue damage is called Pain [30]. Experience of pain in subjective thus difficult to measure.

The characteristic response to any surgical or traumatic injury is as follows:

a. Flare i.e. increased blood flow at the site of injury
b. Wheal i.e. tissue edema
c. Hyperalgesia i.e. peripheral receptor sensitization.

Hyperalgesia is alteration of sense of pain. Here discomfort is markedly increased with recurrent painful stimulus.

Primary hyperalgesia occurs within minutes of injury characterized by hyperresponsiveness to touch, heat and mechanical stimuli. This represents increased sensitivity of C and Aδ fibres or receptors.

Primary hyperalgesia leads to increased wound sensitivity, prolonged discomfort and delayed wound healing due to decreased regional blood flow.

Secondary hyperalgesia is seen in the surrounding area of the injured site. It is a delayed variation in pain sensitivity. This is mediated by central sensitization i.e. in the limbic cortex, brain stem, spinal cord.

Secondary hyperalgesia leads to increased incident pain, muscle splinting and prolonged disability. In addition to secondary hyperalgesia neural and glial remodelling leads to development of chronic pain.

EFFECTS OF PAIN ON ORGAN SYSTEM:

Increased release of catecholamines via sympathetic stimulation leads to decreased peripheral perfusion and tachycardia, hypertension and thus compensatory increase blood flow to vital organs like heart and brain.

Increased peripheral vascular resistance leading to increased myocardial contractility and demand can precipitate myocardial ischemia and infarction in high-risk patients.

Decreased regional blood flow and increased cortisol levels delays wound healing.

In chronic untreated pain there is increased catabolism and decreased anabolism due to variation in the neuroendocrine functions leading to lipolysis and proteolysis which results in decreased immunoglobulin synthesis and impaired phagocytosis leading to reduced immunocompetence.

To conclude, consequences of poorly controlled pain is as follows:

- Reduced functional capacity
- Sleep disturbance
- Delayed wound healing
- Decreased quality of life
- Lengthened hospital stays and increased cost of care.

Therefore, in addition to providing anaesthesia, anaesthesiologists also play a significant role in providing pain management and understanding the details of pain physiology is vital in management of pain.

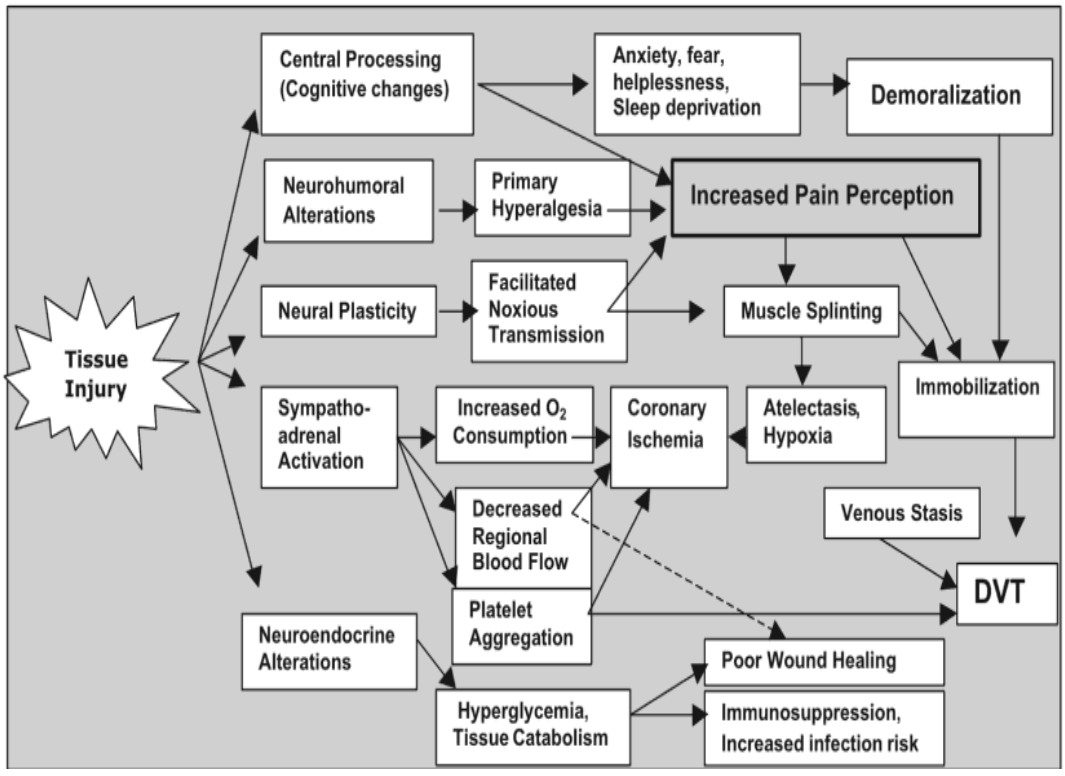


FIG.4 – EFFECTS OF PAIN

PAIN ASSESSMENT

Assessment of pain is a necessary component to achieve adequate pain control in the post operative period. Few of the pain evaluation scales are used in an attempt to assess pain. Most of these scales can be used by the patients themselves to evaluate pain when the patient can express and communicate what pain feels like.

VISUAL ANALOGUE SCALE (VAS):

Visual analogue scale in measurement was introduced in 1966 before which it was used in psychology to measure mood disorder. Since then, it has become a standard and a popular tool for pain assessment. It consists of a line, typically 100 mm long, with anchor descriptions like "no pain" and "worst pain imaginable" (in the context of pain). The distance in millimetres between the patient's mark and the left endpoint is measured after the patient creates a mark that represents their perception.

The WONG-BAKER pain rating scale and Visual Analogue Scale facial expressions: It is a pictorial self-assessment tool which includes six faces. Each face conveys different emotions which range from a face with a cheerful smile to a face with a crying one. It is popular among the population such as younger patients, elderly patients or patients with disorientation or even in patients who cannot comprehend local language or any sort of difficulty in communication.

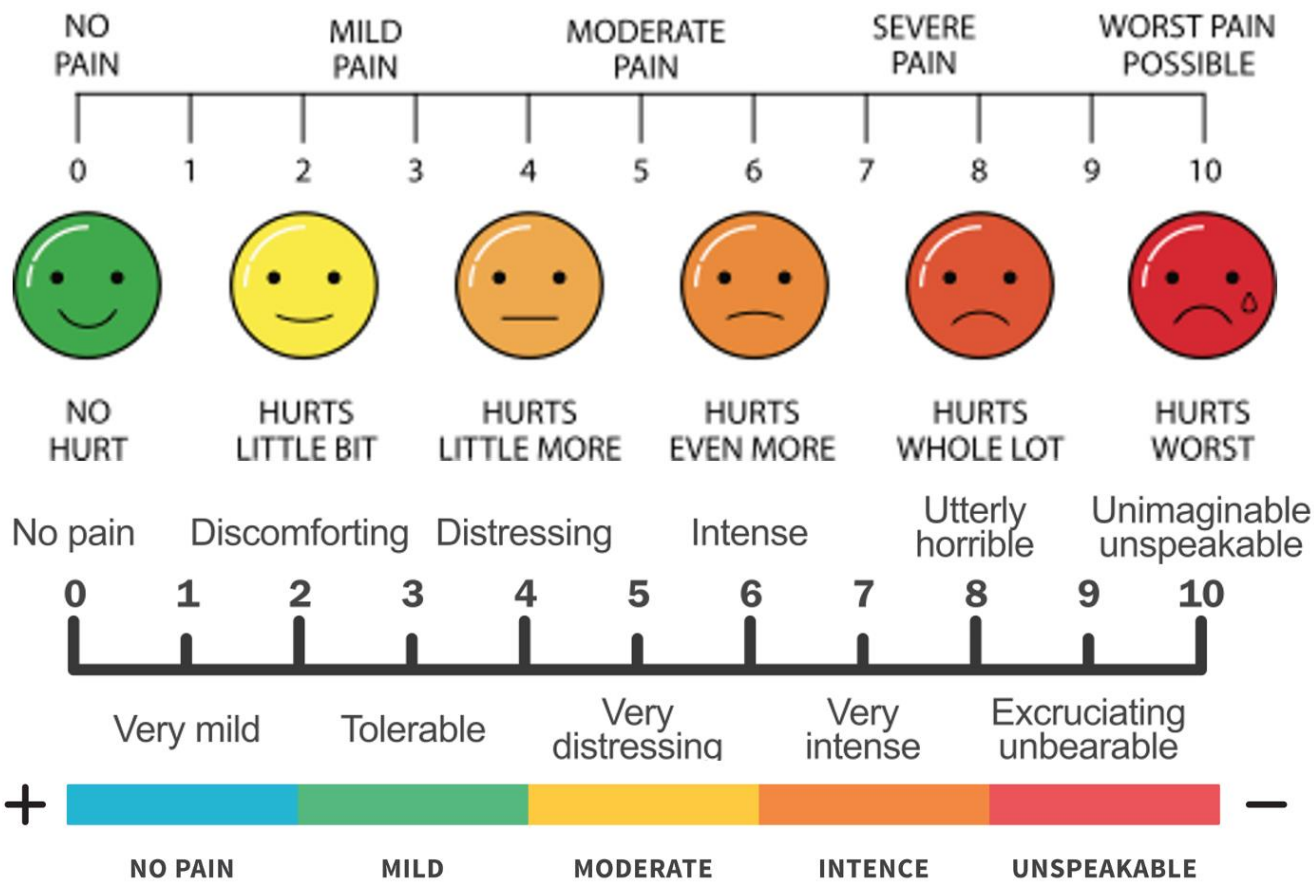


FIG.5 – VISUAL ANALOGUE SCALE

Superficial cervical plexus block [33, 34, 35, 36]

HISTORY:

Cervical plexus blocks were first performed by Halsted in 1884.

Two main approaches of cervical plexus anaesthesia were introduced in the early 20th century.

Posterior approach to cervical plexus was described for the first time in 1923 by Kapis which targeted the nerves at the point of their emergence from the vertebral column.

In 1914, lateral approach was described by Heidenhein which became the basis for development of present techniques of cervical plexus block.

In 1920, Victor Pauchet added to the description of lateral technique and recommended it over posterior technique.

The lateral approach was restudied by Winnie 1975 and described a simple single injection technique.

Currently, the most commonly performed is the lateral approach of cervical plexus block.

Anatomy of cervical plexus

The superficial and deep set of branches forms cervical plexus

The superficial branches are sensory to skin, and the deep branches form motor supply to muscles. The cervical plexus is formed by ventral or also called anterior rami of C1, C2, C3 and C4 cervical nerves.

Superficial cervical plexus includes the four sensory terminal branches of the cervical plexus which include:

1. Lesser occipital nerve with root values C2- supplies occipital region and upper neck. Lesser occipital nerve sometimes is a branch of greater occipital nerve.
2. Greater auricular nerve with root values C2 and C3 supplies skin over parotid gland and posterior auricle
3. Transverse cervical nerve with root values C2 and C3 supplies skin of anterior triangle of neck
4. Supraclavicular nerves with root values C3 and C4 supplies skin over shoulder and upper pectoral region.

These superficial branches emerge at lateral edge of the sternocleidomastoid muscle and lie posterior to the same.

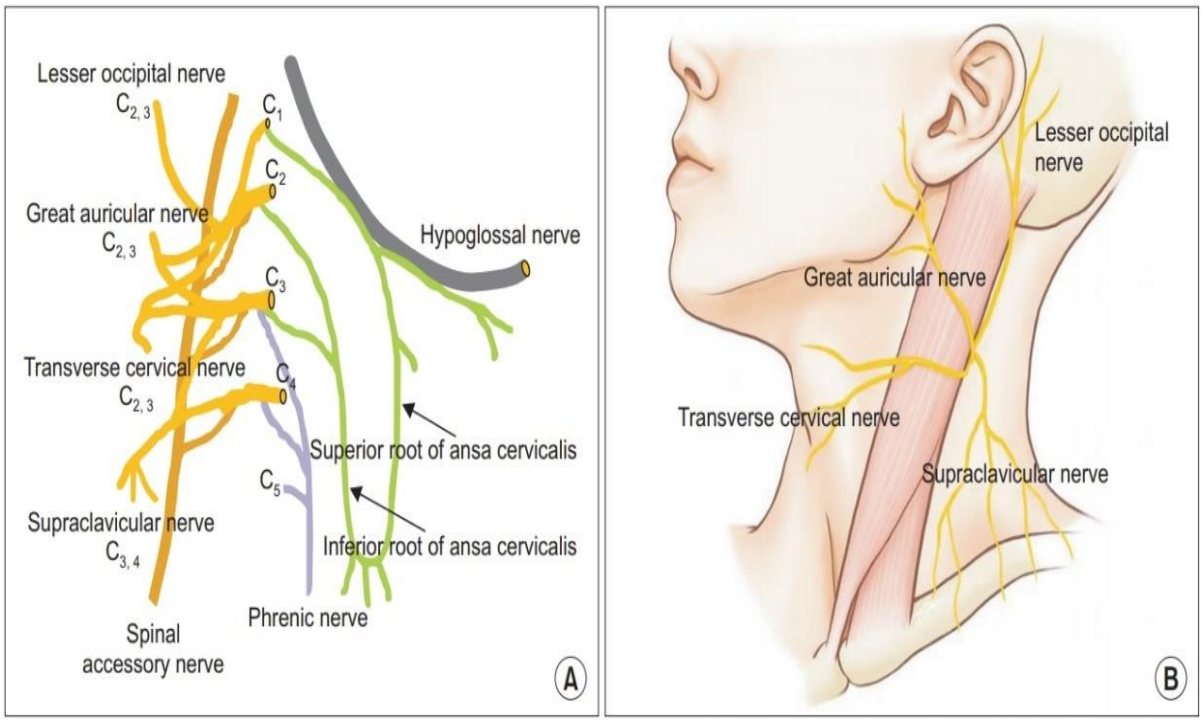


FIG.6(A) – FORMATION OF CERVICAL PLEXUS

(B) – DISTRIBUTION OF SUPERFICIAL CERVICAL PLEXUS

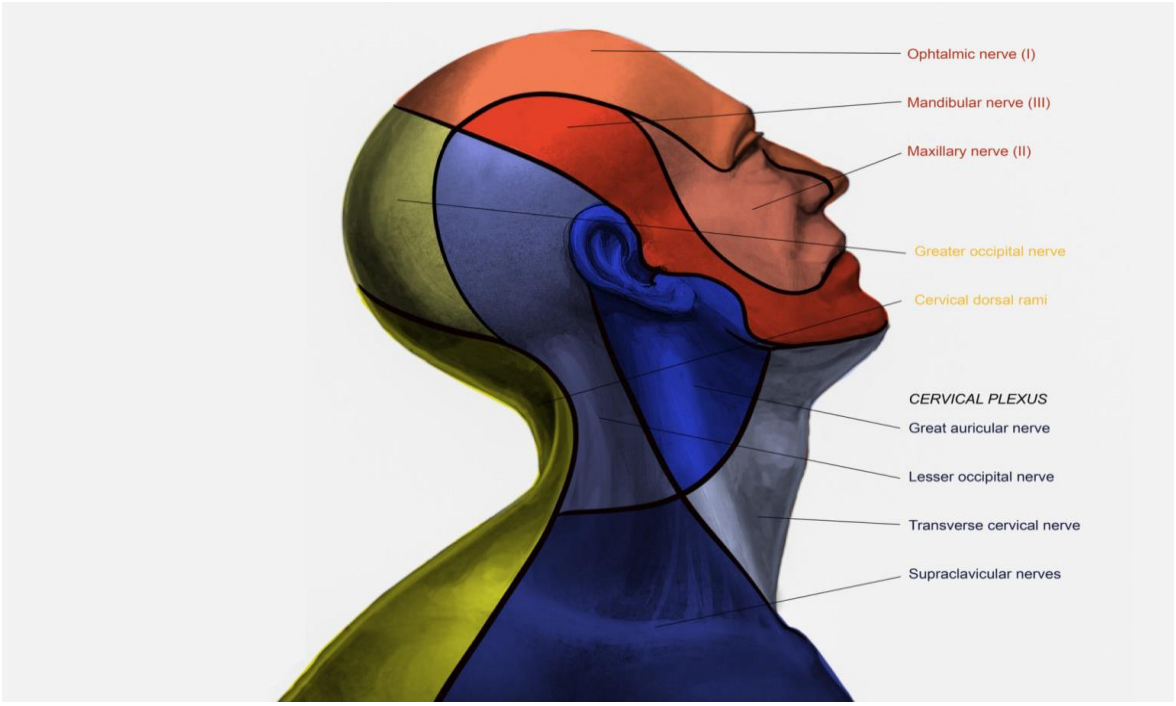


FIG.7 – CUTANEOUS NERVE SUPPLY OF NECK

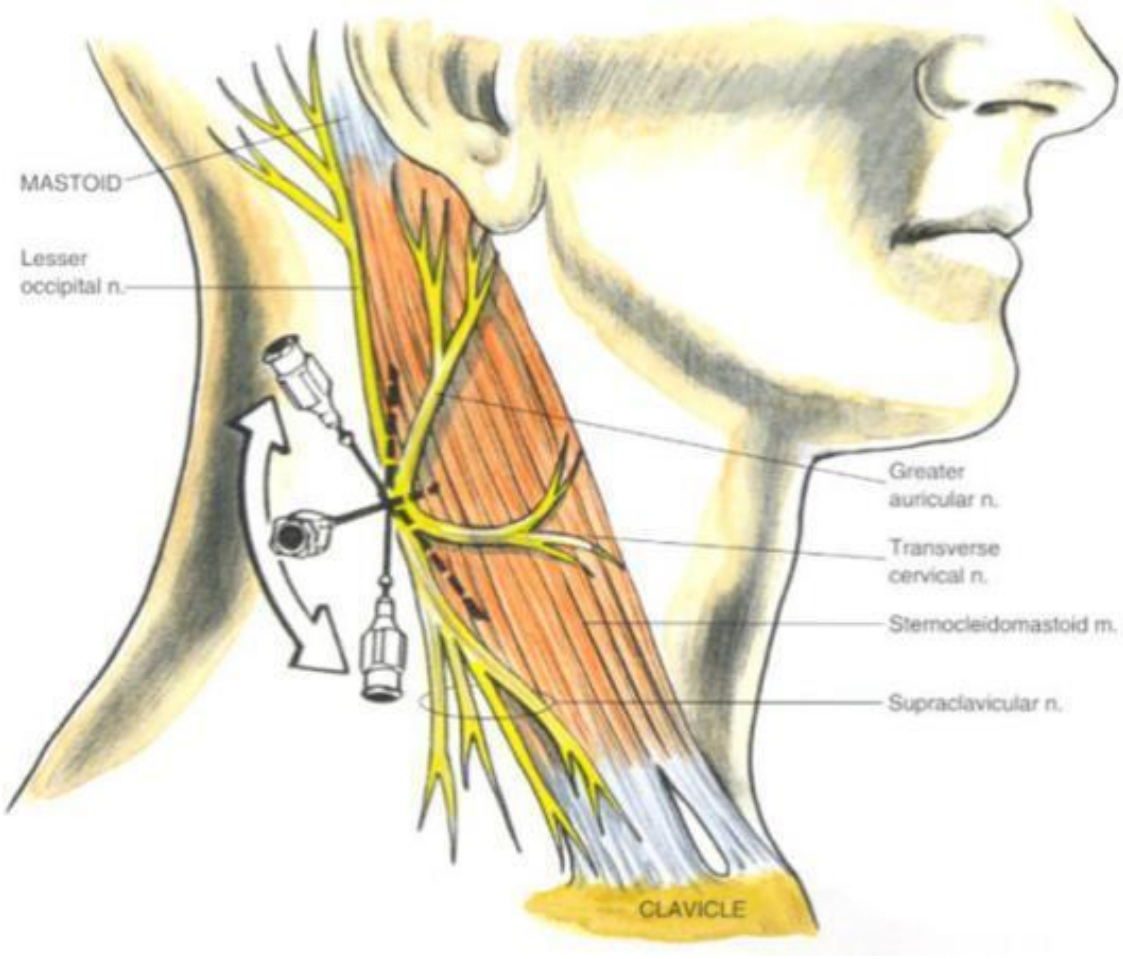


FIG.8 – SCPB (LANDMARK TECHNIQUE)

Indications:

SCPB is used as a single block or alongside deep cervical plexus block for complete anaesthesia in various procedure including:

1. Carotid end arterectomy
2. Lymph node biopsy
3. Internal jugular cannulation.
4. For thyroid and parathyroid surgeries in high-risk patients.

As an analgesic modality in:

1. Carotid surgeries
2. Thyroid surgeries
3. Tracheostomy
4. Mastoid and ear surgeries
5. As a supplement to brachial block in shoulder surgeries.

For chronic pain management in conditions like:

1. Cervical radiculopathy
2. Cervicogenic headache

Benefits of Superficial Cervical Plexus Block:

a. SCPB is an excellent modality foe analgesia for neck and shoulder surgeries.
b. As the analgesia is taken care of, the use of opioids is reduced hence minimizing the adverse effects of opioids such as respiratory depression.
c. It augments patient comfort thus avoiding the need of GA in many procedures.

Technique of SCPB

Landmark technique:

Landmarks:

a. Posterior border of clavicular head of sternocleidomastoid muscle
b. Cricoid cartilage (C6) or midpoint of SCM.

Position of the patient: supine with the head turned to opposite side of the block.

Landmarks as described above are identified.

A small-gauge needle is inserted at the midpoint of posterior border of SCM muscle and directed superficially to the investing fascia of the neck.

Aspiration is performed to confirm the needle is not in any vascular compartment.

Local anaesthetic is injected in a fan shaped in subcutaneous plane along with the posterior border of the SCM muscle. 10-15ml of LA is adequate to block superficial sensory branches.

USG GUIDED SCPB

Position: supine or semi recumbent position with patient's head turned to contralateral.

Skin is prepared and cleaned

Over the lateral side of neck transducer is placed horizontally or in transverse orientation at the midpoint of posterior border of SCM muscle or at the level of cricoid cartilage. Carotid artery, IJV and SCM muscle are located. Tapering end of SCM muscle is identified and is focused in the centre of the screen.

Needle is introduced from the lateral side of the probe through skin and platysma and advanced in the guidance of ultrasound ensuring that the tip of needle is beneath the investing fascia of SCM.

Once the needle tip placement is confirmed with the negative aspiration, 10-15ml of local anaesthetic is injected and spread of same is observed.

Complications of superficial cervical plexus block:

a. Local anaesthetic toxicity: intravascular accidental deposition of local anaesthetic can lead to systemic toxicity.
b. Nerve injury: rare but chances of nerve injury are present with improper needle placement.
c. Formation of hematoma: accidental vascular puncture can cause hematoma at injection site.
d. Infection: if proper aseptic precautions are not taken it is possible to introduce infection as it is an invasive procedure.

Advantages of USG:

a. Easy to perform
b. Improved accuracy and increased success rate of the block
c. Improved safety: blood vessels can be identified on USG and avoided hence decreasing the risk of intravascular LA injection.
d. USG ensures spread of LA in effective location hence requires less volume of local anaesthetic.
e. Reduced complications

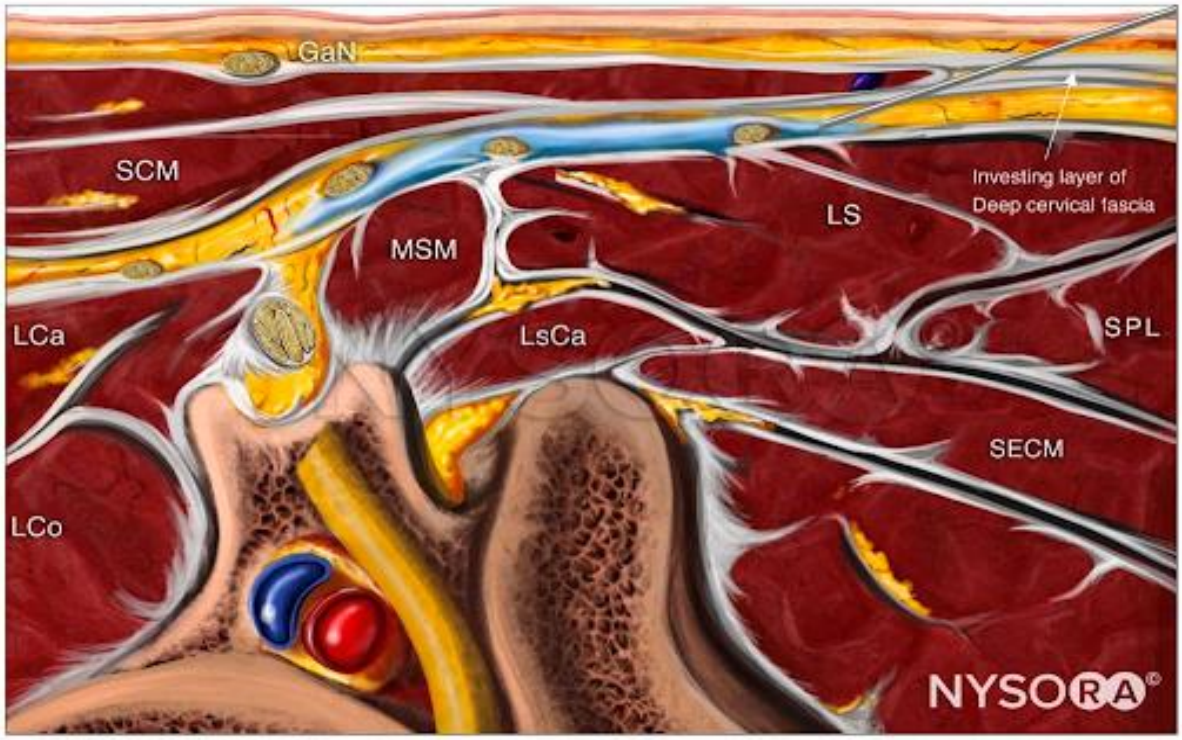


FIG.9 – SCHEMATIC REPRESENTATION OF SCPB

FIG.10- SONOANATOMY OF SUPERFICIAL CERVICAL PLEXUS

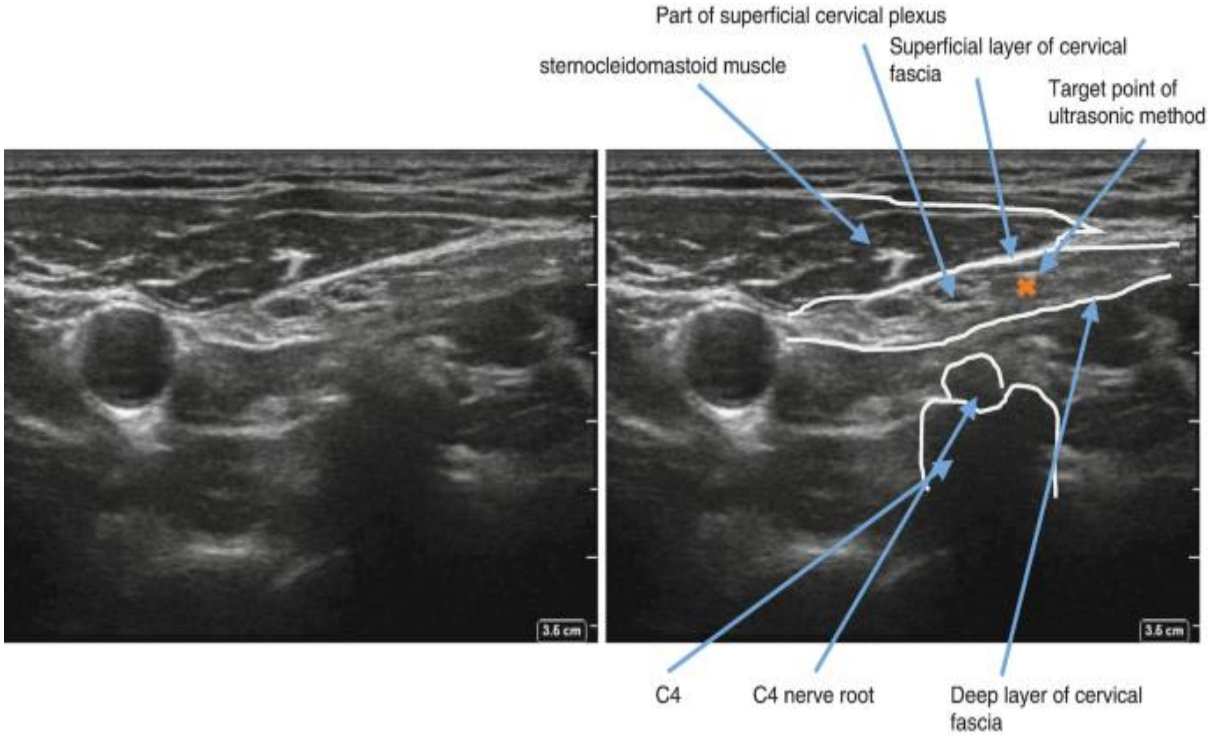


FIG. 11- POSITIONING OF THE PATIENT

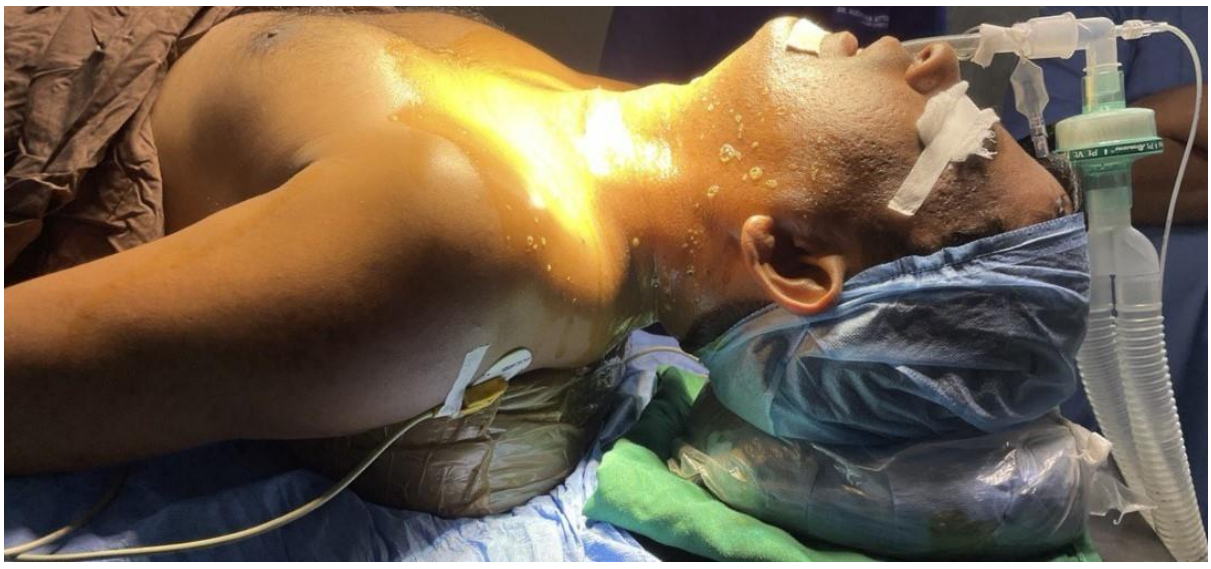
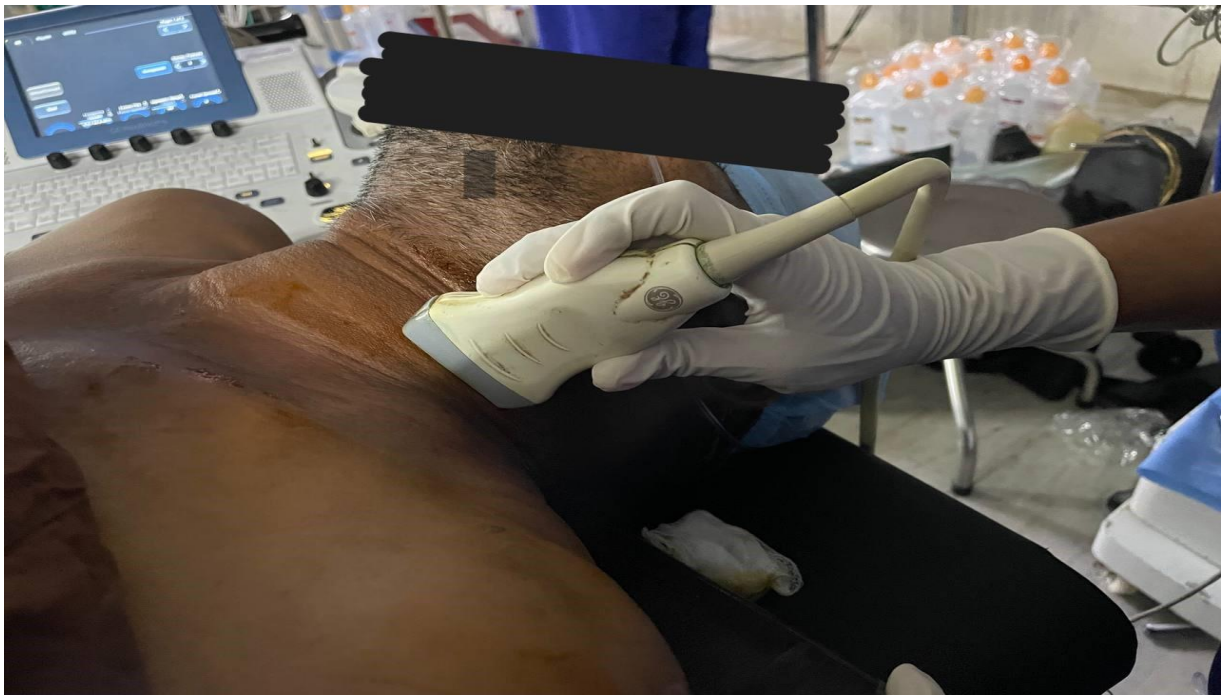


FIG.12 – PROBE PLACEMENT FOR SCPB



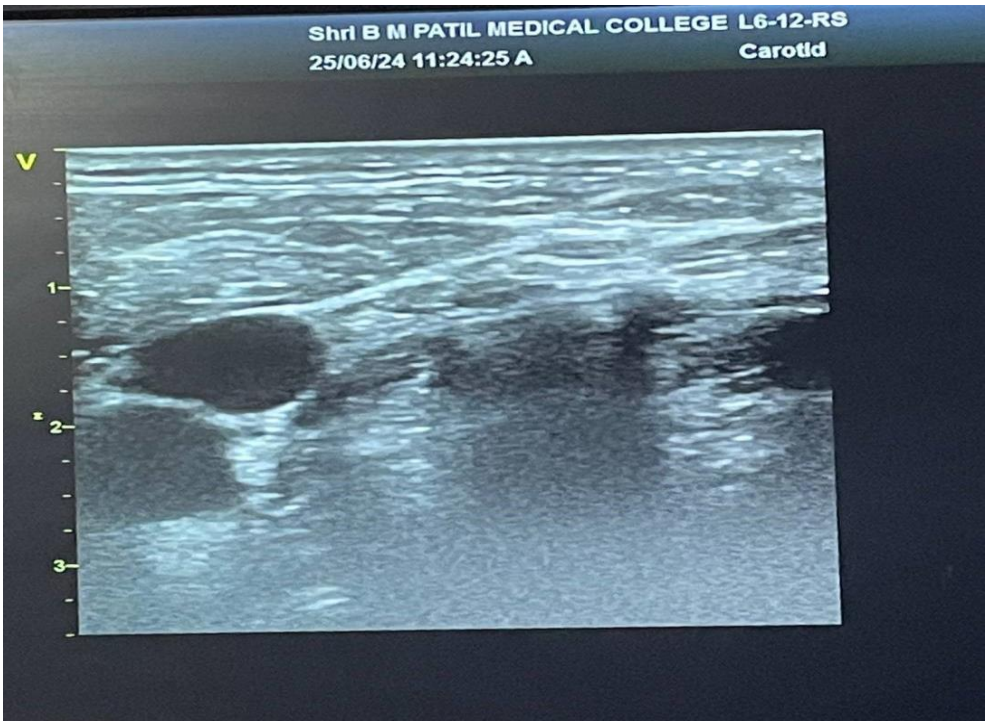


FIG.13 – USG OF SUPERFICIAL CERVICAL PLEXUS

BUPIVACAINE [37, 38, 39, 40]

The first ever used local anaesthetic was cocaine and it was introduced in the year 1884 by Karl Coller. Local anaesthetics have a wide range of applications in day -to-day anaesthesia practice. They are used in spinal anaesthesia, epidural anaesthesia, regional anaesthesia and analgesia, local infiltration all of which aim at achieving perioperative analgesia and postoperative comfort.

Structure:

A local anaesthetic contains a tertiary amine attached to aromatic ring linked together by an intermediate chain which can either be an ester or an amide. Based on the intermediate chain local anaesthetics can be divided into 2 groups namely, esters (eg: Procaine) and amides (eg: bupivacaine).

Bupivacaine is a local anaesthetic which belongs to the amide group. It was first developed in 1957 by Ekenstam and clinically used for the first time in 1963 by L.J. Telivuo. It is a water-soluble hydrochloride salt of lipid soluble bases.

It is tertiary amine which is a relatively hydrophilic basic end while the aromatic ring attached to it by an amide linkage imparts a lipophilic property.

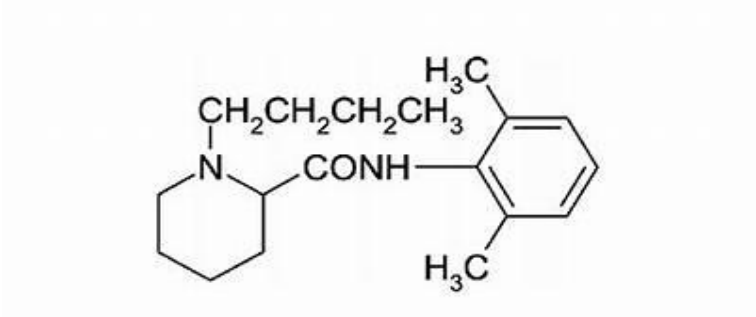


FIG.14- CHEMICAL STRUTURE OF BUPIVACAINE

Chemical formula of Bupivacaine:

1-n-butyl-DL-piperidine-2-carboxy acid -2-b-dimethyl anilide hydrochloride.

Chemical properties:

Molecular weights of the base: 288

Molecular weight of chloride: 324

Protein binding capacity: 96%.

pKa = 8.2 at 25 degrees

specific gravity – 1.0.35 – 1.040

MECHANISM OF ACTION:

The resting membrane potential of all living cells is -60 to -70mV and is usually due to potassium efflux since the membrane is generally leaky to potassium. Neurons and cardiac muscle cells have a unique ability to generate action potentials. Local anaesthetics act by preventing the generation of action potentials in these cells.

Voltage gated sodium channels present in the membrane play an important role in initiation and transmission of the of action potential in neurons and muscle cells. These voltage gated sodium channels have one large α subunit with four domains and 6 loops and one or two smaller β subunit. They exist in one of the three conformational states: 1. Resting state 2. Active state 3. Inactive state

Resting and inactive states are non-conducting while active state is conducting. When the membrane depolarises, sodium channels change their conformation and allow the sodium influx hence generating an action potential.

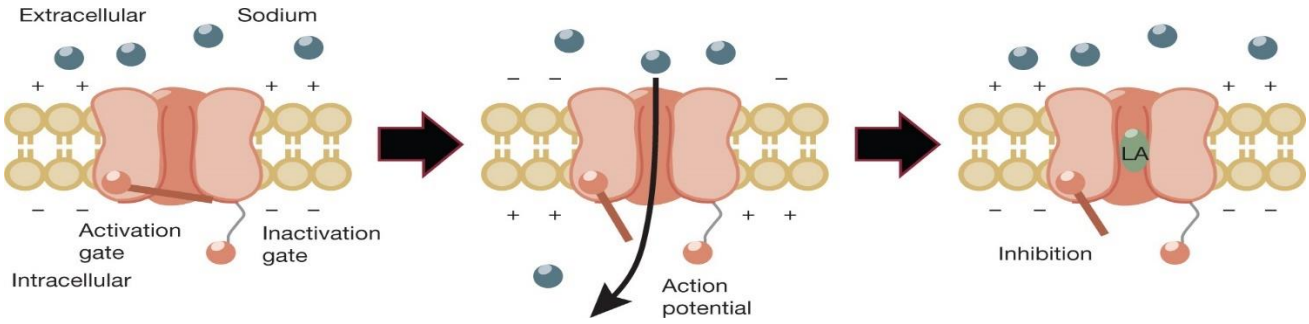


FIG.1615- SODIUM CHANNEL

Local anaesthetics in their ionised bind to the larger α subunit of the sodium channel. They selectively inhibit the sodium channels in active state and blocks the sodium influx which results in prevention of generation and propagation of action potential by increasing the firing threshold, essentially ceasing the nerve transmission. This results in reversible nerve conduction inhibition ensuing sensory loss in the affected area. Higher the concentration of the local anaesthetic, higher fraction of the sodium channels are inhibited.

Factors that influence the nerve fibre sensitivity to local anaesthetics are diameter of axons, myelination of nerve fibres and conduction velocity.

- Slow conducting and small nerve fibres are more sensitive. Eg: C fibres
- Fast conducting and large fibres are less sensitive. Eg: A-δ fibre
- Myelinated fibres are more sensitive compared to unmyelinated fibres.

The sequence of blockade of nerve function by local anaesthetic administration is as follows:

Autonomic → sensory (pain→temperature→touch→proprioception) → motor.

DOSAGE:

Bupivacaine is available in the concentrations of 0.25%, 0.5% and 0.75% preparations. The hyperbaric nature is due to addition of 80mg dextrose which is used in subarachnoid block. Intrathecally, maximum adult dose is 20mg. The highest recommended dose of bupivacaine in peripheral nerve blockade is 3mg/kg.

ADJUVANTS:

Adding adjuvants to bupivacaine prolongs the duration of action. The drugs used as adjuvants with proven benefit are α-2 agonists (Clonidine, Dexmedetomidine), Dexamethasone, Ketamine, Fentanyl, Magnesium, sodium bicarbonate.

USES:

1. SPINAL ANESTHESIA: Hyperbaric bupivacaine 0.5% is used.
2. EPIDURAL BLOCK: Anaesthesia and analgesia are produced with bupivacaine. It can be used for labour analgesia. 0.0625 to 0.5% concentrations are used according to the requirement.
3. PERIPHERAL NERVE BLOCK: concentrations of 0.125% to 0.5% are used.

PHARMACOKINETICS:

Absorption: Local anaesthetic deposited at a site eventually gets absorbed in to the systemic circulation. Bupivacaine absorption depends on site of injection and dosage. Addition of epinephrine doesn't not affect the duration of action of bupivacaine.

Distribution: bupivacaine is highly protein bound (96%). It binds to α_1- acid glycoprotein and very less extent to albumin. It crosses placenta to a limited extent.

High lipid solubility of bupivacaine makes it a highly potent local anaesthetic. High lipid solubility and high protein binding capacity makes it a long-acting local anaesthetic with somewhat delayed onset of action. It takes 5-7 minutes for onset of action. Duration of action of bupivacaine is 3-4 hours.

Alkalinisation of bupivacaine by adding 1ml of 8.4% sodium bicarbonate to 10ml of bupivacaine makes it more potent, increases the duration of action and to an extent reduces the pain on injection.

Volume of distribution is 0.9±0.4L/kg.

Half-life is 2.4±1.2 hours.

Metabolism and excretion: metabolism of bupivacaine is slower due to which a sustained plasma concentration is maintained thus systemic toxicity chances are high. It is metabolised in the Liver by Cytochrome P450. It undergoes

hydroxylation, hydrolysis and conjugation. The end products are eliminated by kidneys. Clearance is approximately 0.58L/min.

PHARMACODYNAMICS:

Locally, at the site of injection it causes nerve blockade. Due to this nerve blockade, the region supplied by the nerves experience loss of sensation to pain, touch, proprioception, motor power and vasomotor tone.

The systemic effects of bupivacaine are due to the systemic absorption of the drug.

1. Cardiovascular system: Bupivacaine causes dose dependent myocardial depression recovery from which is slower due to its slow elimination from the cardiac muscle. Bupivacaine also affects cardiac contractility and pacemaker capacity of SA node causing bradycardia and extreme cases sinus arrest. Bupivacaine is 4 times more cardio-depressant than lidocaine. Low concentration produces vasoconstriction and at high concentrations it produces vasodilatation.

   Accidental intravenous bupivacaine injection causes ventricular tachyarrhythmias, fibrillations or bradycardia and cardiac arrest. It is a life-threatening condition.

2. Central nervous system: Bupivacaine, when used in therapeutic doses is safe with no significant adverse effects. The symptoms of bupivacaine CNS toxicity ranges from circumoral numbness, metallic taste, tinnitus, restlessness, dizziness to generalised convulsions and generalised CNS depression. Plasma level of 1.6 to 2 mcg/kg/ml causes toxicity and convulsions occur at level of 2.3 to 5 mcg/kg/ml.

High plasma levels of bupivacaine results in adverse systemic reactions and toxicity. Hypoxia, hypercarbia and pregnancy increases the chances of toxicity. Following are the toxic effects of Bupivacaine:

1. Primary cardiac failure as bupivacaine causes myocardial depression. It can lead to hypotension, bradycardia and in severe cases arrest. Local anaesthetics tend to bind to adrenoreceptor hance preventing action of epinephrine making the cardiotoxicity refractory to standard resuscitation measures.
2. Above effected CNS manifestation most severe being agitation, convulsions, coma.
3. Bupivacaine causes medullary respiratory centre depression resulting in respiratory depression and apnoea. This is observed to be more common in obstetric patients.

MANAGEMENT OF TOXICITY:

Prevention: Multiple strategies are to be considered in order to prevent toxicity. Ultrasound guided techniques, restricting drug dosage, slow injection and aspiration technique before injection can be a few considerations.

Treatment:

Local injection to be stopped to begin with. The immediate management includes maintaining oxygenation, provide 100% oxygen, secure airway if required to prevent hypoxia, hypercapnia and acidosis.

In management of convulsions, benzodiazepines are the first line of treatment. If the convulsions of persists then low dose muscle relaxant can be used.

Intravenous lipid emulsion therapy: mechanism of lipid emulsion is that it transports the bupivacaine from blood rich organs such as heart to storage or site of metabolism such as muscle and liver. Fast bolus of 100ml of 20% lipid emulsion for 70 kg adult followed by 200-250ml over next 15-20mins is the recommended dose. In a patient less than 70 kg 1.5ml/kg bolus followed by 0.25ml/kg/min is recommended.

In case of a case as severe as cardiac arrest, ACLS algorithms to be followed for resuscitation.

Dexmedetomidine [37, 41, 42, 43]

Dexmedetomidine is a highly selective (α_2: α_1 = 1600:1) and potent α_2 adrenoreceptor agonist.

It is being used for its properties such as sedation, anxiolysis and analgesia.

Dexmedetomidine has greater selectivity for α_2 receptors when compared to clonidine.

CHEMICAL STRUCTURE:

Structurally it is dextro-enantiomer of medetomidine.

(+) 4-[(5)-1-(2,3-DIMETHYLPHENYL) ETHYL]-1 H-IMIDAZOLE

Molecular formula: $C_{13}H_{16}N_2$

It is available as water soluble hydrochloride salt.

Molecular mass: 236.7

pH- 4.5 – 7.0

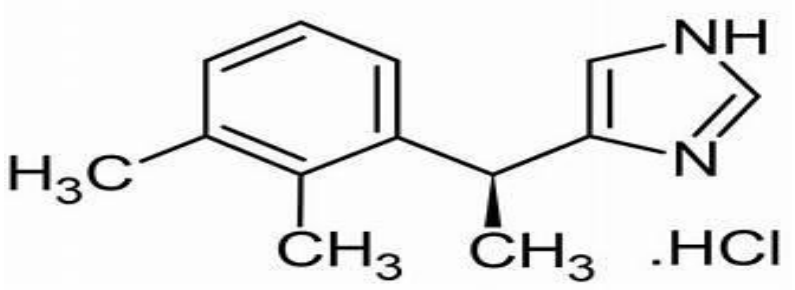


FIG.16 CHEMICAL STRUCTURE OF DEXMEDETOMIDINE

HISTORY:

Dexmedetomidine was first used in 1999.

It was initially approved for intravenous administration as sedative for mechanically ventilated patients in ICU settings.

In 2008, it was additionally used for sedation of non-intubated patients early to or during surgical and non-surgical procedures.

PHYSIOLOGY:

There are two types of adrenergic receptors alpha and beta receptors classified by Ahliquist.

Alpha adrenoreceptors are presynaptic alpha-2 and post synaptic adrenoreceptors.

Stimulation of alpha receptors causes vasoconstriction, intestinal and bladder sphincter contraction, pilomotor contraction and iris dilatation. Alpha -1 is excitatory and alpha-2 is inhibitory and excitatory.

Alpha-2 has three isoreceptors: α_{2a} (presynaptic), α_{2b} (postsynaptic), α_{2c} (extrasynaptic).

Central distribution of alpha-2 receptors

the alpha-2 receptors are present in high density in following sites:

1. Medullary dorsal root complex in brain stem
2. Locu coeruleus
3. Vagus nerve
4. Intermediolateral cell column and substantia gelatinosa
5. Dorsal horn of spinal cord

DRUG FORMULATIONS AND DOSING REGIMEN.

Dexmedetomidine is available in two concentrations:

a. 50mcg/0.5ml ampoule
b. 100mcg/ml- as 1 ml and 2 ml ampoules.

Dosage:

	Loading Dose	Maintenance Dose
For ICU sedation	1mcg/kg over 10 minutes	0.2-0.7mcg/kg/H
For procedural sedation	1mcg/kg over 10 minutes	0.6 mcg/kg/H

PHARMACOKINETICS:

Dexmedetomidine displays linear pharmacokinetics. Onset of action is approximately 15 minutes following IV administration with peak concentrations achieved within 1 hour after continuous IV infusion.

Routes of administration:

a. Absorption and distribution.

Dexmedetomidine is approved for IV use currently. Nonetheless, a number of studies have looked into the usefulness of utilizing it in different ways. Several established methods of administering dexmedetomidine that have gained popularity include intrathecal, intranasal, oral, intramuscular, transdermal and inhalational. Bioavailability through oral, intramuscular and transdermal is 16%, 73% and 88% respectively.

It is a heavily protein bound drug, as high as 94%. It is bound to albumin and α_1-glycoprotein. The protein displacement interaction with other drugs such as fentanyl, lignocaine, ketorolac is negligible. It readily crosses the blood brain barrier

The distribution half-life of dexmedetomidine is 6 mins in adults at 0.2-0.7mcg/kg/H dose range, with volume of distribution of 1.31-2.46L/kg.

b. Metabolism and Excretion:

Dexmedetomidine is metabolized to a large extent by Liver via N-glucuronidation and cytochrome P450 enzyme mediated (i.e., CYP2A6) biotransformation. All the resultant metabolites are inactive and non-toxic. Its elimination half-life is 2.1 - 3.1 hours with clearance of 0.6-0.7L/min.

Patient with altered albumin levels have prolonged or shortened elimination half-life but this has no effect on clearance whereas, patients with low cardiac output have decreased clearance due to decreased blood flow to liver.

PHARMACODYNAMICS:

A. SEDATIVE EFFECTS: Sedation caused by dexmedetomidine is similar to natural sleep. The sedative and hypnotic effects are seen with activation of presynaptic and post synaptic alpha-2 receptors in Locus Coeruleus. Dexmedetomidine also has an impact on the endogenous sleep promoting pathways. Arousable sedation is seen at the plasma concentration of 0.2-0.3ng/ml. non-arousable deep sedation is seen with plasma concentration ≥ 1.9ng/ml.

B. ANALGESIC EFFECTS: these effects are seen via action of dexmedetomidine at alpha-2 receptors present in intermediolateral cell column and substantia gelatinosa in the spinal cord. Analgesic effects are due to two mechanisms, one is hyperpolarization of interneurons and other

one is reduction in release of nociceptive neurotransmitters such as substance P.

C. Cardiovascular effects: a biphasic hemodynamic response is seen that is typical for dexmedetomidine i.e. hypertension at high plasma concentration and hypotension at low plasma concentration. When IV bolus of the drug is given which results in peak plasma concentration this increases blood pressure along significant decrease in heart rate which is followed by decrease in plasma concentration causes vasodilatation hence the hypotension. Cardiac output decreases due to decrease in heart rate. Stroke volume is reduced if the plasma concentration is beyond 5.1ng/ml. high plasma concentration also results in systemic and pulmonary vascular resistance causing systemic and pulmonary hypertension. Bradycardia and sinus arrest can occur which respond to anticholinergics.

D. Respiratory effects: At the plasma levels up to 2.4ng/ml minimal respiratory depression is seen along with ventilatory response to CO_2 (hypercapnic ventilatory response) intact. Even in deep unarousable sedation respiratory drive is unaffected. Hypercapnic ventilatory response

fades with age and hence geriatric population is more prone to respiratory depression.

E. CENTRAL NERVOUS SYSTEM EFFECTS: CBF and CMR are both reduced by dexmedetomidine. It is absorbed by the cerebrospinal fluid as it a lipophilic drug and bond to alpha-2a receptors in the dorsal horn of spinal cord and prolongs the action of local anaesthetics. It also said to be neuroprotective as it modulated the pro and antiapoptotic proteins, reduced cerebral catecholamine and glutamate release.

F. ENDOCRINE EFFECTS: The peripheral alpha-2 presynaptic receptors are activated by dexmedetomidine reducing the release of catecholamines and thus alleviate sympathetic response during surgery.

G. RENAL EFFECTS: Dexmedetomidine acts as a diuretic by inhibition of vasopressin action at collecting duct. It also increases glomerular filtration rate.

H. OTHERS: reduced salivation, decreased bowel motility in the gastrointestinal tract, contraction vascular and other smooth muscle, decreased intraocular pressure are miscellaneous actions seen with dexmedetomidine.

ADVERSE EFFECTS:

The most common adverse effect seen are bradycardia and hypotension.

Cases of cardiac arrest following severe bradycardia have been reported with dexmedetomidine.

Other adverse effects seen with drug are nausea, vomiting, dry mouth, atrial fibrillation.

When administered IV rapidly as an infusion in less than 10 mins it causes transient hypertension.

CLINICAL USES OF DEXMEDETOMIDINE:

The effects of dexmedetomidine are desired throughout the perioperative period.

1. Premedication: it is suitable drug for premedication due to its effects such as sedation, anxiolysis, sympatholysis, analgesia and its anti-sialagogue property. Dosage: 0.33-1 mcg/kg given IV 15mins before procedure can provides cardiovascular stability. Intra-nasal route is used in paediatric population which is effective with the dose of 1mcg/ml.
2. As an adjuvant to general anaesthetics: dexmedetomidine reduces intubation stress response and sympathetic response during emergence. It decreases the requirement of intravenous, inhalational anaesthetics intraoperatively. It has perioperative opioid sparing effect. Along with a

deep sedation the upper airway patency is maintained and hence makes appropriate drug for difficult airway management.

3. In regional anaesthesia: addition of dexmedetomidine to local anaesthetics i.e. 1mcg/kg in epidurally and 3mcg/ml intrathecally is observed to remarkably prolong the sensory and motor block. 1-2mcg/kg dexmedetomidine added to local anaesthetics for peripheral nerve blocks shortens the onset time, prolongs the duration of action and reduced analgesia requirement post operatively.
4. In monitored anaesthesia care: it can be used as baseline sedative for patients undergoing MAC. Dexmedetomidine produces all the desired effects with higher patient satisfaction similar to drugs like midazolam and fentanyl but avoids respiratory depression.
5. Sedation in ICU: dexmedetomidine was originally approved for ICU sedation only. It is famous due its arousable sedation. Patients remain awake, calm hence can communicate their needs. It shortens the ICU. It is recommended to be used for 24 hours only.
6. Procedural sedation: it is an ideal agent for short procedures due its unique type of sedation where the patient will be sedated and becomes responsive if and when aroused and has been used safely in colonoscopy, shockwave lithotripsy, awake fibreoptic intubation in difficult airway situations, paediatric MRI.

CONTRAINDICATIONS:

- Patients with heart block
- Patients with pre-existing bradyarrhythmia or severe bradycardia
- Dexmedetomidine is not extensively studied in obstetric patients hence contraindicated.
- Patients with reduced ventricular functions.
- Hypovolemic or hypotensive patients
- Dose reductions are needed in patients with liver failure or renal impairment.
- Patients on beta blockers and calcium channel blockers require lesser dose than normal.

DEXAMETHASONE [44, 45]

Dexamethasone is a synthetic adreno-cortical steroid with anti-inflammatory effects. It was first described in 1958.

It is more potent than hydrocortisone i.e. 30-40 times and prednisone i.e. 16 times.

CHEMICAL STRUCTURE:

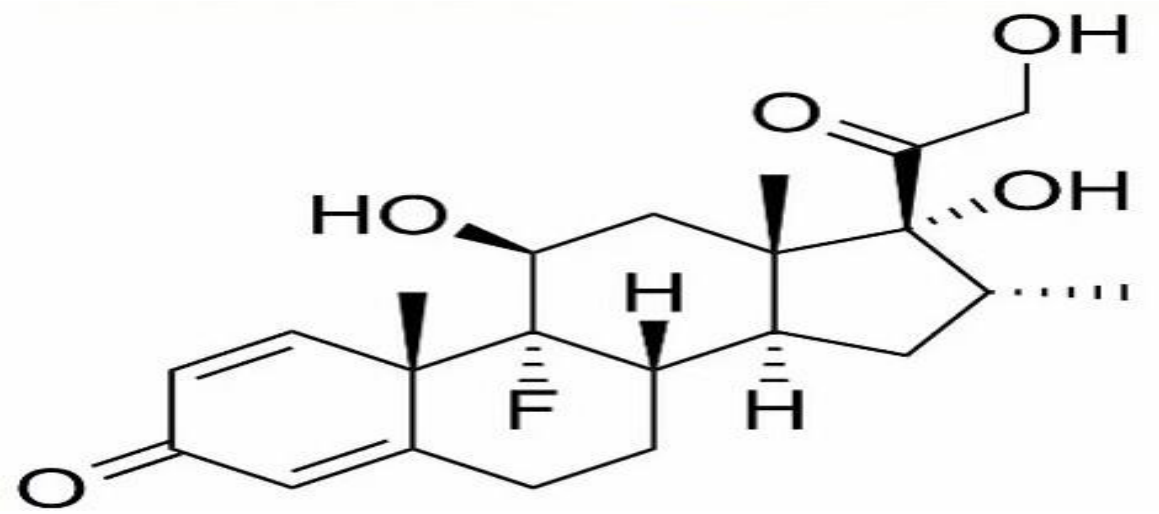


FIG. 17- CHEMICAL STRUCTURE OF DEXAMETHASONE

9-FLURO-11β, 17, 21-TRIHYDROXY-16α-METHYLPREGNA-1,4-DIENE-3,20-DIONE, 21- (DIHYDROGEN PHOSPHATE)

MECHANISM OF ACTION:

Glucocorticoid receptors (GR): Receptors for dexamethasone are located intracellularly on nucleus. They belong to the nuclear receptor subfamily 3, group C, member 1 of transcription factor (NR3C1). GRs are present in majority in cytoplasm in inactive state bound to other proteins. When a steroid ring bind to the GR this leads to activation of the receptor and translocation to nucleus.

Dexamethasone has glucocorticoid effect with minimal mineralocorticoid activity with a complex mechanism of action.

The steroid ring binds to the receptors in the effector site or the target tissue. The binding GRs to the steroid ring dissociated them from the protein binding and are translocated to nucleus where they interact with specific DNA sequences (called GRE). The GREs are responsible for induction of specific genes transcriptions by glucocorticoids. This causes gene transcription of the corticosteroid responsive genes. This transcription leads to variation in the protein synthesis at the effector site an as a result there will be altered cell function.

Genes for COX-2, inducible NOS and inflammatory cytokines are negatively regulated by glucocorticoids.

The effect of gene transcription is reduced release of pro-inflammatory mediators such as bradykinin, IL-1, IL-2, IL-6 along with reduced production of

prostaglandins, collagenase. This leads to immunosuppressive and anti-inflammatory effect.

Dexamethasone also suppresses the migration of neutrophils and decreases lymphocyte colonies. It decreases capillary membrane permeability.

It increases the levels of surfactant and improves pulmonary circulation.

PHARMACOKINETICS:

Absorption: Glucocorticoids can be effectively used by oral route, IM and IV. When administered IV high concentration of the drug are rapidly achieved. IM injection of the drug gives more prolonged effects. Glucocorticoids are systemically absorbed from the site of injection, for example skin, synovial spaces in the joint, respiratory tract.

Distribution, Metabolism and Excretion: Glucocorticoids highly reversibly protein bound (up to 90%). The two protein which are known to involve with steroids are transcortin which is a α-globulin and albumin.

Metabolism of cortisol and its derivatives takes place in liver by converting them into dihydro- and tetrahydro- compounds. For example, cortisol is converted to tetrahydrocortisol. These compounds are then conjugated with sulfate or glucuronic acid via an enzymatic reaction in the liver. The resultant conjugates are water soluble and are excreted via kidneys.

CLINICAL USES:

1. As replacement therapy in adrenal insufficiency.
2. Used in diagnosis of hypercortisolism.
3. Widely used in treatment of rheumatic diseases like SLE, polyarteritis nodosa, Wegener's granulomatosis, rheumatic arthritis.
4. used in treatment of non-inflammatory conditions like osteoarthritis, tendinitis, bursitis as local injection.
5. Used for the treatment of renal diseases like nephrotic syndrome, membranous glomerulonephritis, renal diseases secondary to SLE.
6. As a supplement to primary therapy in treatment of allergic diseases.
7. For the treatment of pulmonary diseases such as bronchial asthma.
8. To prevent RDS and IVH in premature neonates.
9. Used in treatment of certain specific infectious pathogen. eg: pneumocystis carinii pneumonia in AIDS patients, hemophilius influenza B meningitis in infants and children, COVID infections.
10. Topical steroids are used in ocular and skin diseases.
11. As a combination therapy in treatment of leukaemia and lymphomas. It is also used in treatment of chemotherapy induced nausea and vomiting.
12. At high doses it is used to cerebral edema.

ADVERSE EFFECTS:

Adrenal suppression on withdrawal, suppression of somatic growth, osteoporosis, osteopenia, osteonecrosis, impaired glucose tolerance, increased susceptibility to infections due to immunosuppression, myopathy which is proximal limb muscle weakness, cataracts.

ROLE OF DEXAMETHASONE IN ANAESTHESIA:

1. Dexamethasone is used in treatment of post-operative nausea vomiting.

2. Post-operative pain relief: the mechanism of dexamethasone in pain relief is by inhibiting cyclooxygenase and lipoxygenase which suppresses inflammatory, metabolic and immune responses to surgical stimulus. The dose and time of administration before surgical incision is of importance here. Several studies suggests that an intermediate dose of dexamethasone that is 0.1-0.2mg/kg given 60mins or prior to that of surgical incision will provide significant pain relief and has opioid sparing effect.

   Dexamethasone can be used as intravenous injection, as adjuvants in epidural anaesthesia and as epidural injections and as perineural injection.

   IV dexamethasone: It significantly decreases post-operative pain when given along with general anaesthesia. When given with spinal anaesthesia it increases duration of sensory block without affection the duration of

motor block. It also increases duration of peripheral nerve blockade when given iv.

Epidural dexamethasone: The membrane stabilizing effects on nerves or the direct action on spinal cord by means of transcription factors like NFKB (nuclear factor kappa B) renders it pain relieving property. When given epidurally it prolongs duration of post-operative anaesthesia and analgesia and also has opioid sparing effect.

Perineural dexamethasone: It causes duration of block by vasoconstriction which decreases absorption of LA from the site. It is also observed to decrease incidence of rebound pain.

3. Effect of dexamethasone on neuromuscular blockade: it is found to decrease the rocuronium induced and cis-atracurium induced block by 15-20% if given 2-3hrs before surgery.

4. Dexamethasone for shivering: Shivering causes sympathetic stimulation, increased oxygen consumption and affects post operative recovery. Dexamethasone reduces shivering by decreasing the gradient between skin and core body temperature. It is found to be more effective than pethidine in preventing postoperative shivering.

5. Dexamethasone at the dose of 0.1mg/kg when given increases the quality of recovery in the postoperative period.

6. Postoperative sore throat: it is a common and distressing problem encountered after general anaesthesia. Dexamethasone when administered either IV, topical or nebulization decreases the incidence of postoperative sore throat, nebulization being the most effective technique.

MATERIALS AND METHODS

1 SOURCE OF DATA:

This study was carried out in the Department of Anaesthesiology, B.L.D.E. (Deemed to be) University, Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapur.

2 METHOD OF COLLECTION OF DATA:

Study Design: This is a comparative prospective study.

Study Period: one and half year from to November 2022 to April 2024

Sample Size:

Using G*Power ver. 3.1.9.4 software for sample size calculation, The post operative visual analogue scale at 2 hour for Group D (Mean=2.4, SD=0.21) and Group S (Mean=5.4, SD=4.14), this study required a sample size of 74 (for each group 37,assuming equal group size).So to achieve a power of 99% for detecting a difference in Means (t tests - Means: Difference between two independent means (two groups)) with 5% level of significance.

STATISTICAL ANALYSIS

- The data obtained was entered in a Microsoft Excel sheet, and statistical analysis was performed using a statistical package for the social sciences (SPSS) (Version 20).

- Results are presented as Mean ± SD, and percentages and bar graphs.
- For normally distributed continuous variables between the two groups independent sample t-test was applied. For not normally distributed variables, the Mann-Whitney U test was used. For Categorical variables between the two groups were compared using the Chi-square test/Fisher's exact test.
- $P<0.05$ was considered statistically significant.
- All statistical were performed two-tailed.

Inclusion criteria:

- Patients aged between 18-60 years, of either sex.
- Patients admitted for thyroid surgeries under General anaesthesia with ASA Grade I and ASA Grade II

Exclusion criteria:

- Patient refusal
- Inability to consent for the procedure
- Local site infection
- Allergies
- Coagulopathies
- A history of cardiac, respiratory, hepatic, or renal failure, and those who refused local anaesthesia or had an aversion to local anaesthesia were excluded.

- Patients with heart block
- Patients on adrenoreceptor agonist or antagonist treatment were also excluded

METHODOLOGY

PRE-ANESTHETIC EVALUATION:

Patients were included in the study by a thorough pre-operative evaluation done on the previous day of surgery, which included the following:

- **HISTORY:**

  History of underlying medical illness, previous history of surgery, previous anesthetic exposure, and hospitalization was elicited.

- **PHYSICAL EXAMINATION:**

  The general condition of the patient, Vital signs (heart rate, blood pressure, respiratory rate), Height and weight, examination of the cardiovascular system, respiratory system central nervous system and the vertebral system, Airway assessment by Mallampati grading was carried out

- **CONSENT**

  Written informed consent was obtained from the patient during the pre-anaesthetic evaluation.

INVESTIGATIONS:

- Routine investigations for the surgery such as Complete blood count, PT INR, blood sugars, blood urea and serum creatinine, serology, ECG and chest radiography were performed
- Specific investigations included thyroid profile and neck X-ray with AP and lateral view.

PROCEDURE:

A study was conducted in our institute on 74 patients who underwent thyroid surgeries.

- The patients were randomly allotted into two equal groups of 37 each.

  Group A received ultrasound guided BSCPB with dexmedetomidine as adjuvant to 10ml bupivacaine after induction with GA.

  Group B received ultrasound guided BSCPB with dexamethasone as adjuvant with 10ml bupivacaine after induction with GA.
- Patients were educated about the visual analogue score during the pre-anaesthetic evaluation on the previous day.
- Once the patients were shifted to the operation theatre after confirming nil per oral status, a 20G IV Cannula was secured and monitors were attached, which included E.C.G. leads, NIBP cuff for non-invasive blood pressure measurement, and pulse oximeter for SPO2 measurement, and baseline readings were noted.

- The patients were premedicated with inj. Ondansetron 0.15mg/kg, Midazolam 1mg, Glycopyrrolate 0.015mg/kg. Induction of anaesthesia was done with Injection Propofol 2mg/kg with Injection Fentanyl 2mcg/kg as analgesic and the muscle relaxant inj. Atracurium 0.5mg/kg.
- Patients were intubated with the appropriately sized ETT and tube was fixed after confirming bilateral equal air entry.
- Volatile anaesthetics like isoflurane was used according to the requirement.
- Patients were randomized into 2 groups as mentioned above.
- BSCPB was performed by the anaesthesiologist before surgical incision was taken.
- With patients' head in extension and turned towards the opposite side with a linear transducer [A linear 7-13 MHz ultrasound-guided probe (Sonosite M-Turbo, U.S.A.) was used] was placed at the level of cricoid cartilage, the superficial cervical plexus was visualized with ultrasound as hypoechoic structures (a honeycomb appearance) lateral to the posterior border of sternocleidomastoid muscle. 10ml bupivacaine with an adjuvant will be injected on each side. Aspiration technique was used to rule out the presence of blood in the hub of the needle before injection.

- Adjuvant dosage: Dexmedetomidine: 25mcg on each side

  Dexamethasone: 4mg on each side

- Intraoperatively patient's heart rate (HR), blood pressure (BP) and mean arterial pressure (MAP) were monitored at every 10 mins after 30 minutes from the time of block up to 120mins.
- Neuromuscular blockade was reversed with neostigmine and glycopyrrolate and patient was smoothly extubated.
- Post-operatively visual analogue score was assessed for both groups at 2, 6, 8, 10 and 12 and 24 hours after surgery.
- Postoperatively the time for rescue analgesia was noted.
- If the VAS score is more than 4, rescue analgesia will be given with intravenous infusion of inj. diclofenac 75mg in 100ml normal saline.

VAS Score Intensity of pain

- 0 – 2- No pain to slight pain
- 1 – 3 - Mild pain.
- 4 – 6 - Moderate pain.
- 7 – 9 - Severe pain.
- 10 - Worst possible pain.

OBSERVATION AND RESULTS

The collected data from our study conducted was represented in the master chart.

Total sample size is 74 (group A and group B containing 37 patients each who are undergoing thyroid surgeries).

Group A received dexmedetomidine with bupivacaine in the block.

Group B received dexamethasone with bupivacaine in the block.

P value lea than 0.05 is considered statistically significant.

1. DEMOGRAPHIC VARIABLES:

Age (years)	Group A		Group B	
	Number of patients	%	Number of patients	%
18-20	0	0	0	0
21-30	2	5.4	3	8.1
31-40	15	40.5	12	32.4
41-50	13	35.1	10	27
51-60	7	18.9	12	32.4
N=	37	100.0	37	100.0

TABLE 1(A)- AGE DISTRIBUTION

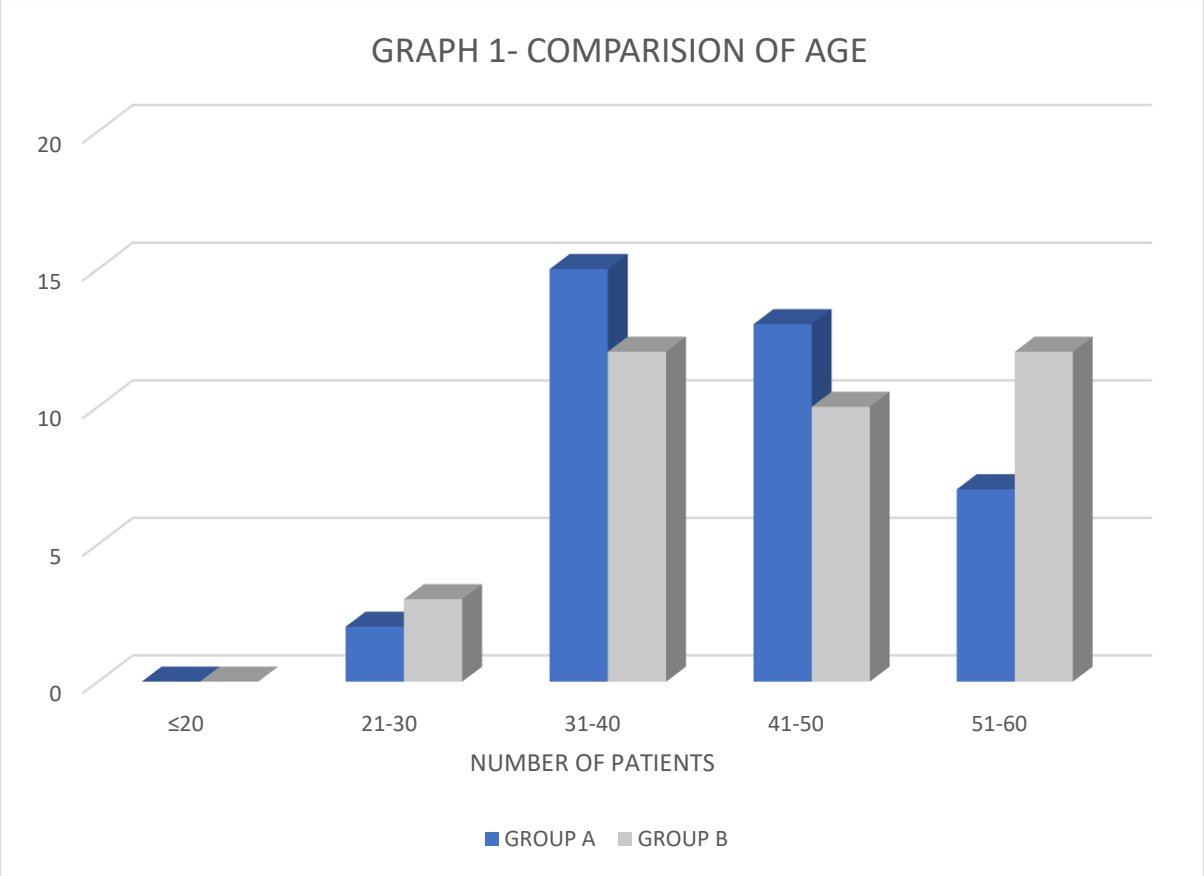


TABLE 1(B) – MEAN AGE OF TWO GROUPS

AGE (IN YEARS)	MEAN	SD	P VALUE
GROUP A	42.59	8.642	**0.219**
GROUP B	45.4	8.96	

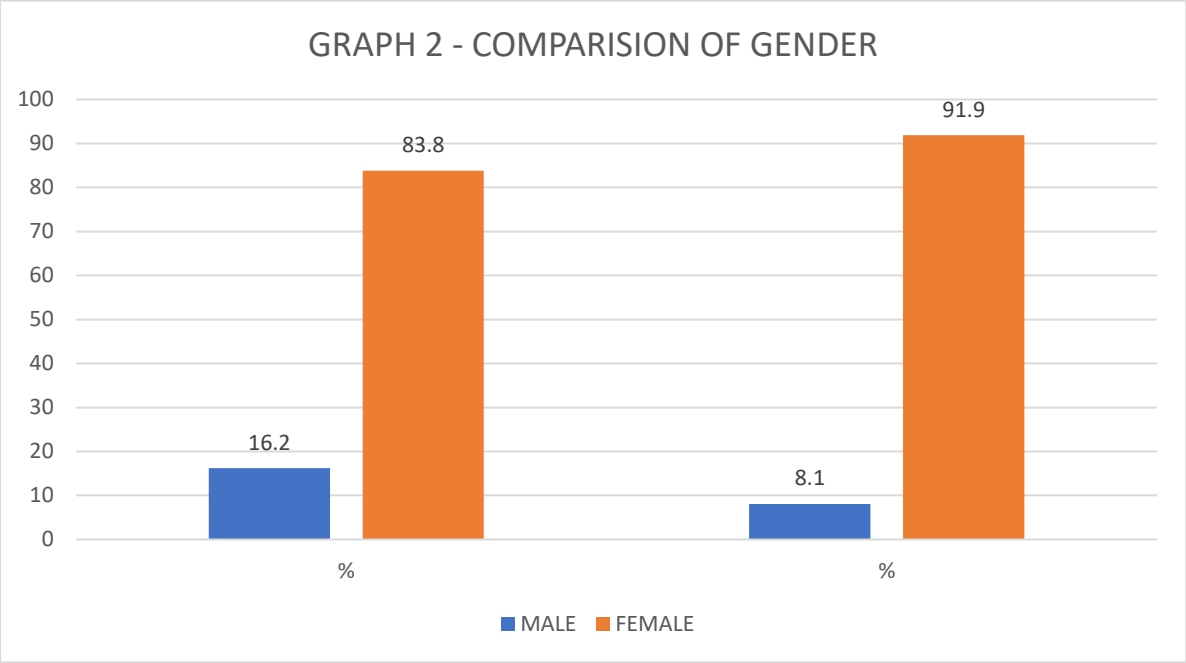
In our study, 5 patients were 21-30 years of age, 27 patients were in the range of 31-40 years, 23 patients were 41-50 years of age, 19 patients were of 51-60 years of age.

Age wise distribution of the sample in both groups are comparable with P-value statistically insignificant.

COMPARISION OF GENDER:

GENDER	GROUP A		GROUP B	
	NUMBER OF PATIENTS	%	NUMBER OF PATIENTS	%
MALE	6	16.2	3	8.1
FEMALE	31	83.8	34	91.9
TOTAL	37	100	37	100

TABLE 3. DISTRIBUTION OF GENDER

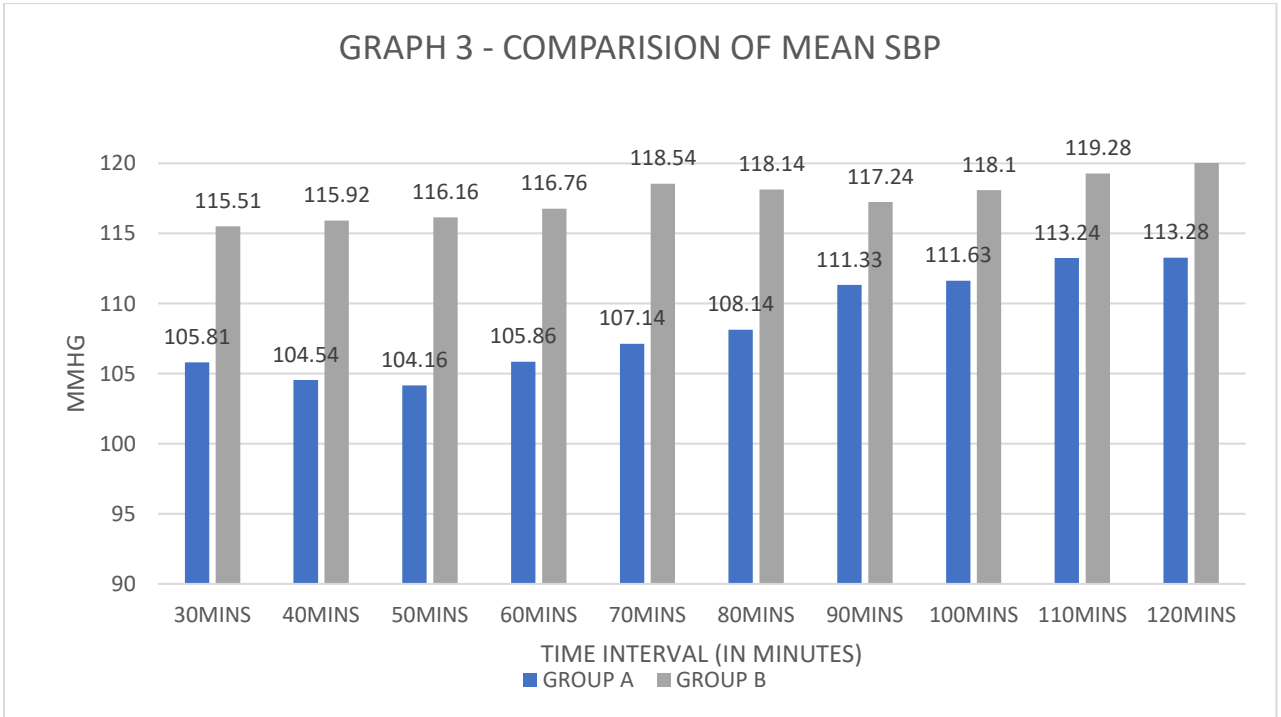


In this trial, female population is predominant when compared to male population.

2. COMPARISON OF SYSTOLIC BLOOD PRESSURE AT SPECIFIC TIME INTERVALS INTRAOPERATIVELY

SBP @INTERVALS	GROUP A		GROUP B		P VALUE
	MEAN	SD	MEAN	SD	
30MINS	105.81	8.498	115.51	11.57	0.000
40MINS	104.54	8.974	115.92	10.286	0.000
50MINS	104.16	8.251	116.16	10.782	0.000
60MINS	105.86	9.361	116.76	10.177	0.000
70MINS	107.14	9.349	118.54	8.924	0.000
80MINS	108.14	10.973	118.14	8.613	0.000
90MINS	111.33	9.049	117.24	9.162	0.009
100MINS	111.63	6.308	118.1	10.293	0.004
110MINS	113.24	8.573	119.28	11.319	0.003
120MINS	113.28	8.556	120.08	11.511	0.002

TABLE 3 – DISTRIBUTION OF SBP BETWEEN 2 GROUPS



At 30 min interval after the administration of block:

In group A, the mean±SD SBP was 105±8.49

In group B the mean±SD SBP was 115±11.57

These values were found to be statistically significant (P value =0.000)

At 40 min interval after the administration of block:

In group A, the mean±SD SBP was 104.54±8.97

In group B the mean±SD SBP was 115.92±10.28

These values were found to be statistically significant (P value = 0.000)

At 50 min interval after the administration of block:

In group A, the mean±SD SBP was 104.16±8.25

In group B the mean±SD SBP was 115.92±10.78

These values were found to be statistically significant (P value = 0.000)

At 60 min interval after the administration of block:

In group A, the mean±SD SBP was 105.86±9.36

In group B the mean±SD SBP was 116.76±10.17

These values were found to be statistically significant (P value = 0.000)

At 70 min interval after the administration of block:

In group A, the mean±SD SBP was 107.14±9.34

In group B the mean±SD SBP was 118.54±8.94

These values were found to be statistically significant (P value = 0.000)

At 80 min interval after the administration of block:

In group A, the mean±SD SBP was 108.14±10.973

In group B the mean±SD SBP was 118.14±8.613

These values were found to be statistically significant (P value =0.000)

At 90 min interval after the administration of block:

In group A, the mean±SD SBP was 111.33±9.04

In group B the mean±SD SBP was 117.24±9.16

These values were found to be statistically significant (P value = 0.009

At 100 min interval after the administration of block:

In group A, the mean±SD SBP was 111.63±6.30

In group B the mean±SD SBP was 118.10±10.293

These values were found to be statistically significant (P value =0.004)

At 110 min interval after the administration of block:

In group A, the mean±SD SBP was 113.24±8.51

In group B the mean±SD SBP was 119.28±11.31

These values were found to be statistically significant (P value = 0.03)

At 120 min interval after the administration of block:

In group A, the mean±SD SBP was 113.28±8.55

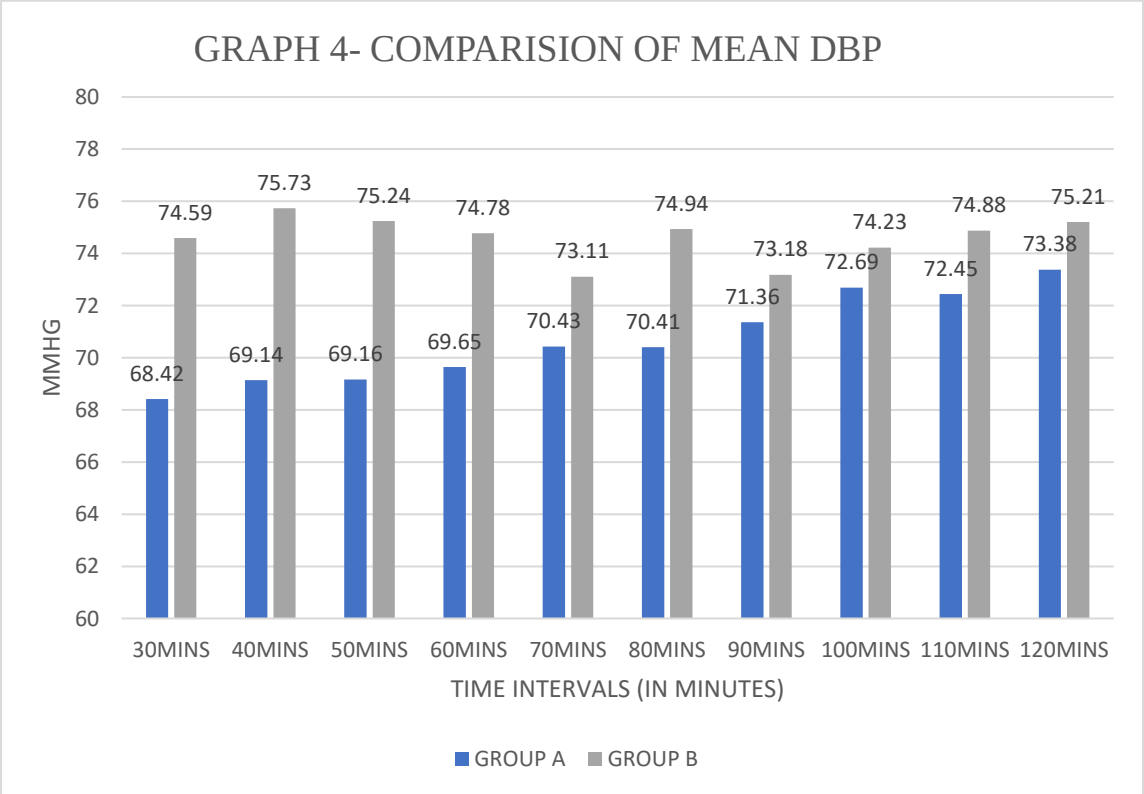
In group B the mean±SD SBP was 120.08±11.51

These values were found to be statistically significant (P value = 0.02)

3. COMPARISION OF MEAN DIASTOLIC BLOOD PRESSURE AT SPECIFIC TIME INTERVALS INTRAOPERATIVELY

DBP @INTERVALS	GROUP A		GROUP B		P VALUE
	MEAN	SD	MEAN	SD	
30MINS	68.42	6.851	74.59	8.949	0.001
40MINS	69.14	7.962	75.73	6.748	0.000
50MINS	69.16	7.03	75.24	6.813	0.000
60MINS	69.65	8.015	74.78	7.036	0.005
70MINS	70.43	6.922	73.11	7.07	0.01
80MINS	70.41	7.507	74.94	6.265	0.007
90MINS	71.36	6.114	73.18	5.881	0.21
100MINS	72.69	5.699	74.23	6.74	0.33
110MINS	72.45	6.972	74.88	8.141	0.24
120MINS	73.38	6.196	75.21	8.516	0.37

TABLE 4 – DISTRIBUTION OF MEAN DBP BETWEEN 2 GROUPS



At 30 min interval after the administration of block:

In group A, the mean±SD DBP was 68.42±6.85

In group B the mean±SD DBP was 74.59±8.94

These values were found to be statistically significant (P value = 0.001)

At 40 min interval after the administration of block:

In group A, the mean±SD DBP was 69.14±7.96

In group B the mean±SD DBP was 75.73±6.74

These values were found to be statistically significant (P value < 0.0001)

At 50 min interval after the administration of block:

In group A, the mean±SD DBP was 69.16±7.03

In group B the mean±SD DBP was 75.24±6.81

These values were found to be statistically significant (P value < 0.0001)

At 60 min interval after the administration of block:

In group A, the mean±SD DBP was 69.65±8.01

In group B the mean±SD DBP was 74.78±7.03

These values were found to be statistically significant (P value = 0.005)

At 70 min interval after the administration of block:

In group A, the mean±SD DBP was 70.43±6.92

In group B the mean±SD DBP was 73.11±7.07

These values were found to be statistically significant (P value = 0.01)

At 80 min interval after the administration of block:

In group A, the mean±SD DBP was 70.41±7.50

In group B the mean±SD DBP was 74.94±6.26

These values were found to be statistically significant (P value = 0.007)

At 90 min interval after the administration of block:

In group A, the mean±SD DBP was 71.36±6.11

In group B the mean±SD DBP was 73.18±5.88

These values were comparable (P value = 0.21)

At 100 min interval after the administration of block:

In group A, the mean±SD DBP was 72.69±5.69

In group B the mean±SD DBP was 74.23±6.74

These values were comparable (P value = 0.33)

At 110 min interval after the administration of block:

In group A, the mean±SD DBP was 72.45±6.97

In group B the mean±SD DBP was 74.88±8.14

These values were comparable (P value = 0.24)

At 120 min interval after the administration of block:

In group A, the mean±SD DBP was 73.38±6.19

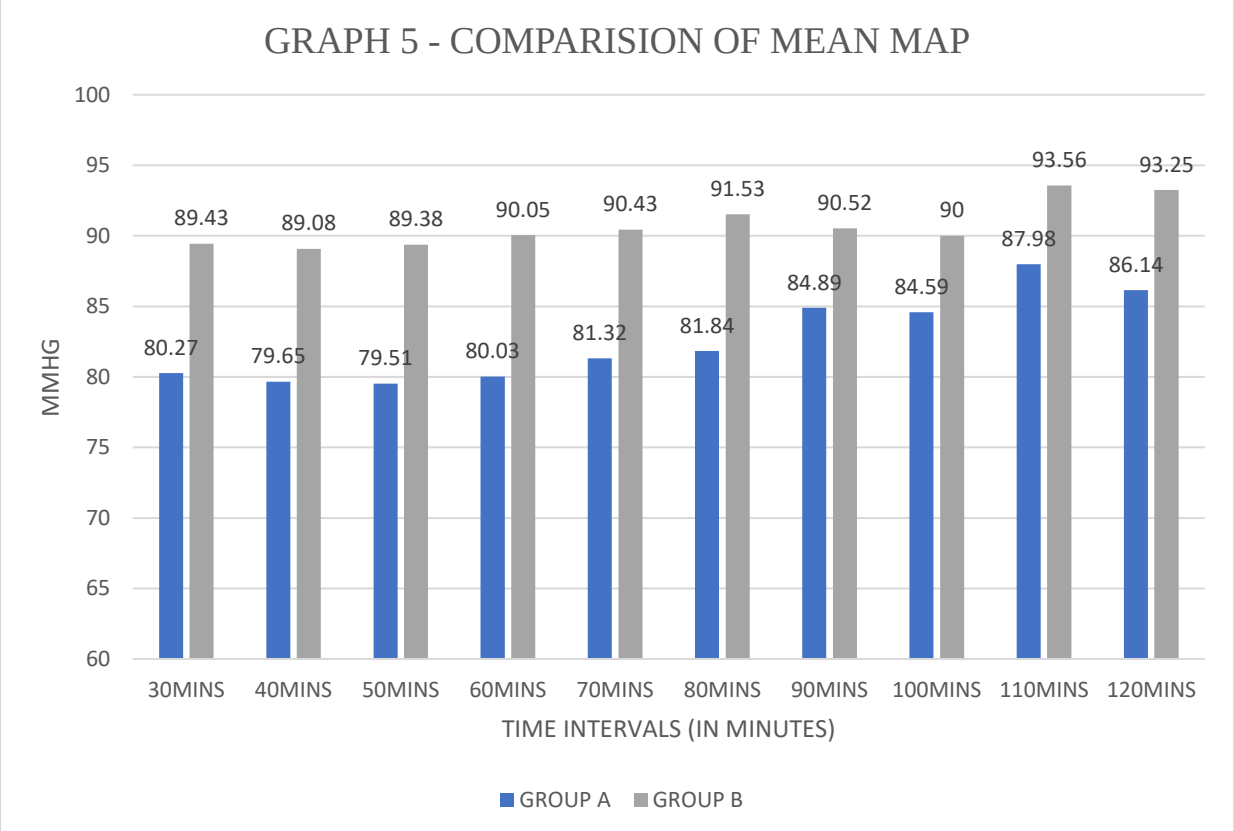
In group B the mean±SD DBP was 75.21±8.51

These values were comparable (P value = 0.37)

4. COMPARISION OF MEAN ARTERIAL PRESSURE INTRAOPERATIVELY AT SPECIFIC TIME INTERVALS

MAP @INTERVALS	GROUP A		GROUP B		P VALUE
	MEAN	SD	MEAN	SD	
30MINS	80.27	7.463	89.43	10.637	<0.0001
40MINS	79.65	8.371	89.08	7.804	<0.0001
50MINS	79.51	7.727	89.38	9.187	<0.0001
60MINS	80.03	8.221	90.05	9.852	<0.0001
70MINS	81.32	9.049	90.43	9.674	<0.0001
80MINS	81.84	10.513	91.53	9.37	<0.0001
90MINS	84.89	9.239	90.52	9.203	0.014
100MINS	84.59	6.026	90	9.663	0.01
110MINS	87.98	9.915	93.56	9.764	0.025
120MINS	86.14	8.505	93.25	9.887	0.007

TABLE 5 – DISTRIBUTION OF MAP BETWEEN 2 GROUPS



At 30 min interval after the administration of block:

In group A, the mean±SD MAP was 80.27±7.46

In group B the mean±SD MAP was 89.43±10.637

These values were found to be statistically significant (P value < 0.0001)

At 40 min interval after the administration of block:

In group A, the mean±SD MAP was 79.65±8.37

In group B the mean±SD MAP was 89.08±7.80

These values were found to be statistically significant (P value < 0.0001)

At 50 min interval after the administration of block:

In group A, the mean±SD MAP was 79.51±7.72

In group B the mean±SD MAP was 89.38±9.187

These values were found to be statistically significant (P value < 0.0001)

At 60 min interval after the administration of block:

In group A, the mean±SD MAP was 80.03±8.22

In group B the mean±SD MAP was 90.05±9.85

These values were found to be statistically significant (P value < 0.0001)

At 70 min interval after the administration of block:

In group A, the mean±SD MAP was 81.32±9.04

In group B the mean±SD MAP was 90.43±9.67

These values were found to be statistically significant (P value < 0.0001)

At 80 min interval after the administration of block:

In group A, the mean±SD MAP was 81.84±10.51

In group B the mean±SD MAP was 91.53±9.37

These values were found to be statistically significant (P value =< 0.0001)

At 90 min interval after the administration of block:

In group A, the mean±SD MAP was 84.89±9.23

In group B the mean±SD MAP was 90.52±9.20

These values were found to be statistically significant (P value = 0.014)

At 100 min interval after the administration of block:

In group A, the mean±SD MAP was 84.59±6.02

In group B the mean±SD MAP was 90.00±9.66

These values were found to be statistically significant (P value = 0.01)

At 110 min interval after the administration of block:

In group A, the mean±SD MAP was 87.38±9.91

In group B the mean±SD MAP was 93.56±9.76

These values were found to be statistically significant (P value = 0.025)

At 120 min interval after the administration of block:

In group A, the mean±SD MAP was 86.14±8.50

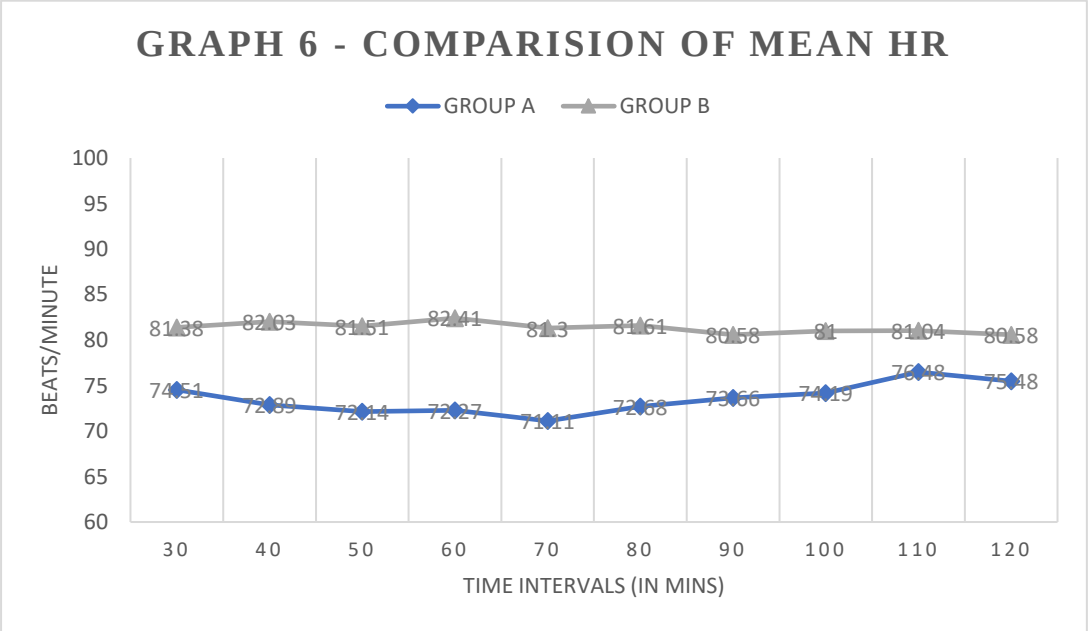
In group B the mean±SD MAP was 93.25±9.88

These values were found to be statistically significant (P value = 0.007)

5. COMPARISION MEAN HEART RATE INTRAOPERATIVE AT SPECIFIC TIME INTERVALS

HEART RATE @INTERVALS	GROUP A		GROUP B		P VALUE
	MEAN	SD	MEAN	SD	
30	74.51	10.767	81.38	10.753	0.008
40	72.89	11.162	82.03	12.44	0.001
50	72.14	10.711	81.51	12.534	0.001
60	72.27	11.047	82.41	12.255	0.0001
70	71.11	10.736	81.3	12.106	0.0001
80	72.68	11.161	81.61	11.455	0.001
90	73.66	9.233	80.58	10.886	0.003
100	74.19	9.58	81	9.296	0.006
110	76.48	9.661	81.04	9.423	0.008
120	75.48	8.605	80.58	9.184	0.04

TABLE 6 – DISTRIBUTION OF HEART RATE



At 30 min interval after the administration of block:

In group A, the mean±SD HR was 74.51±10.76

In group B the mean±SD HR was 81.38±10.75

These values were found to be statistically significant (P value = 0.008)

At 40 min interval after the administration of block:

In group A, the mean±SD HR was 72.89±11.16

In group B the mean±SD HR was 82.03±12.44

These values were found to be statistically significant (P value = 0.001)

At 50 min interval after the administration of block:

In group A, the mean±SD HR was 72.14±10.71

In group B the mean±SD HR was 81.51±12.53

These values were found to be statistically significant (P value = 0.001)

At 60 min interval after the administration of block:

In group A, the mean±SD HR was 72.27±11.04

In group B the mean±SD HR was 82.41±12.25

These values were found to be statistically significant (P value < 0.0001)

At 70 min interval after the administration of block:

In group A, the mean±SD HR was 71.11±10.73

In group B the mean±SD HR was 81.30±12.10

These values were found to be statistically significant (P value < 0.0001)

At 80 min interval after the administration of block:

In group A, the mean±SD HR was 72.68±11.16

In group B the mean±SD HR was 81.61±11.45

These values were found to be statistically significant (P value = 0.001)

At 90 min interval after the administration of block:

In group A, the mean±SD HR was 73.06±9.23

In group B the mean±SD HR was 80.58±10.88

These values were found to be statistically significant (P value = 0.003)

At 100 min interval after the administration of block:

In group A, the mean±SD HR was 74.19±9.58

In group B the mean±SD HR was 81.00±9.29

These values were found to be statistically significant (P value = 0.006)

At 110 min interval after the administration of block:

In group A, the mean±SD HR was 76.48±9.66

In group B the mean±SD HR was 81.04±9.42

These values were found to be statistically significant (P value = 0.008)

At 120 min interval after the administration of block:

In group A, the mean±SD HR was 75.48±8.60

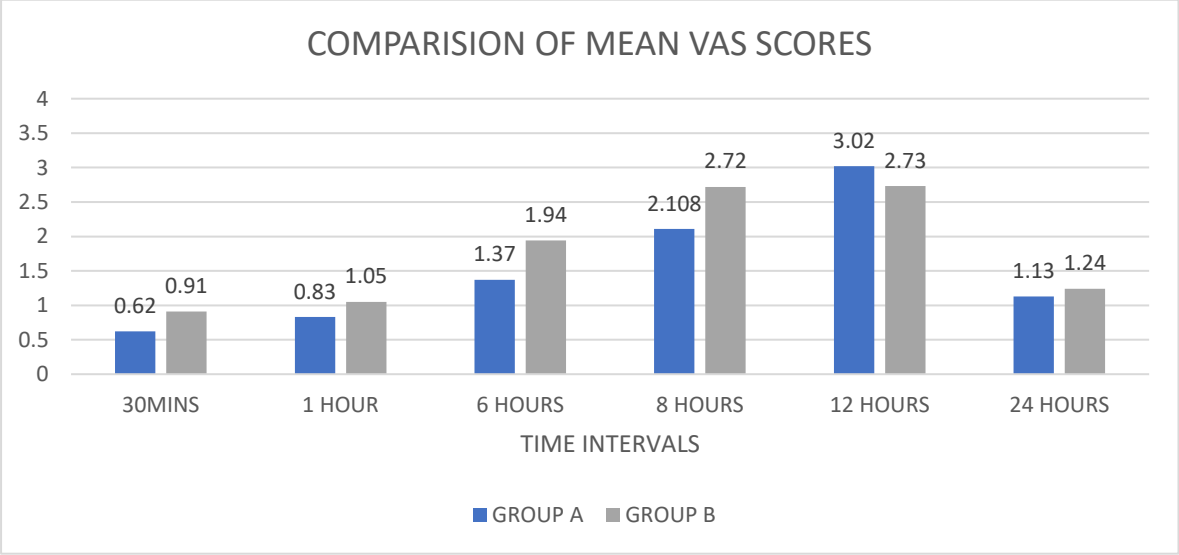
In group B the mean±SD HR was 80.58±9.18

These values were found to be statistically significant (P value = 0.04)

6. COMPARISION OF MEAN VAS SCORE AT SPECIFIED TIME INTERVALS POSTOPERATIVELY

VAS SCORE	GROUP A		GROUP B		P VALUE
	MEAN	SD	MEAN	SD	
30MINS	0.62	0.53	0.91	0.42	0.011
1 HOUR	0.83	0.49	1.05	0.22	0.015
2 HOURS	1.18	0.39	1.45	0.64	0.031
6 HOURS	1.37	0.58	1.94	0.65	0.0002
8 HOURS	2.108	0.68	2.72	0.68	0.0002
12 HOURS	3.02	0.78	2.73	0.64	0.08
24 HOURS	1.13	0.34	1.24	0.48	0.259

TABLE 7 – DISTRIBUTION OF VAS SCORES



Mean VAS scores between Group A and Group B show statistical significance difference.

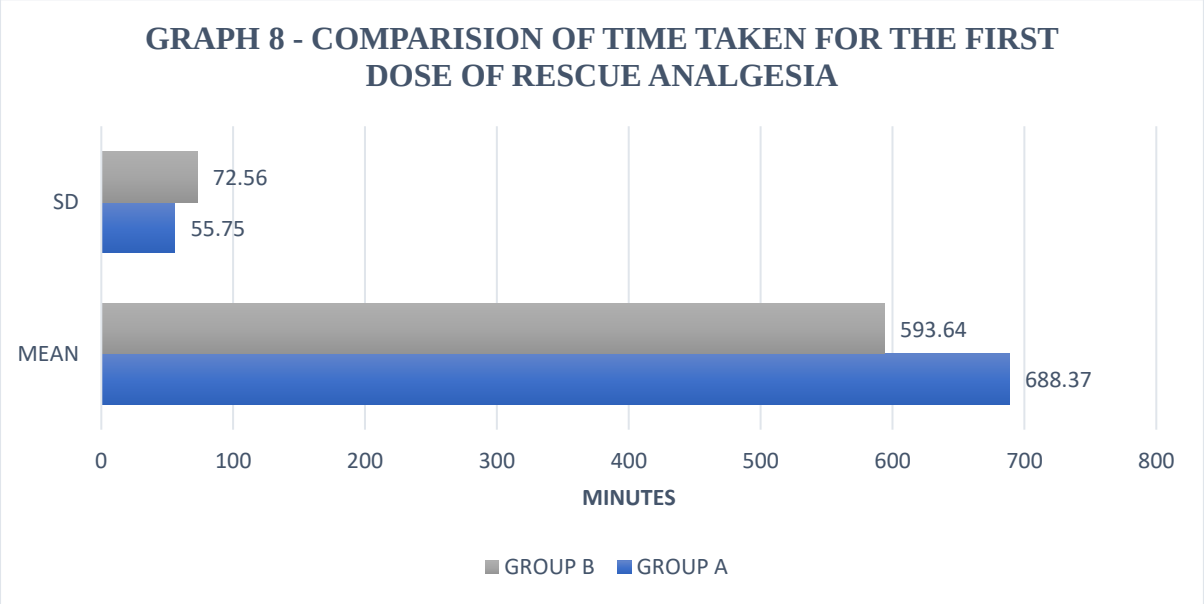
Group A has significantly lower VAS scores at 30mins, I hour, 2 hours, 6 hours, 8 hours with P value <0.05.

VAS scores at 12 hours and 24 hours are comparable with P value > 0.05.

TIME TAKEN FOR THE FIRST DOSE OF RESCUE ANALGESIA:

TIME TAKEN FOR THE FIRST ANALGESIA DOSE REQUEST (IN MINS	MEAN	SD	P VALUE
GROUP A	688.37	55.75	0.001
GROUP B	593.64	72.56	

TABLE.8 – DISTRIBUTION OF TIME TAKEN FOR THE FIRST DOSE OF RESCUE ANALGESIA



The mean time period before the request for the first dose of rescue analgesia (in mins) when compared between group A and group B, it is statistically significant with p value of 0.01.

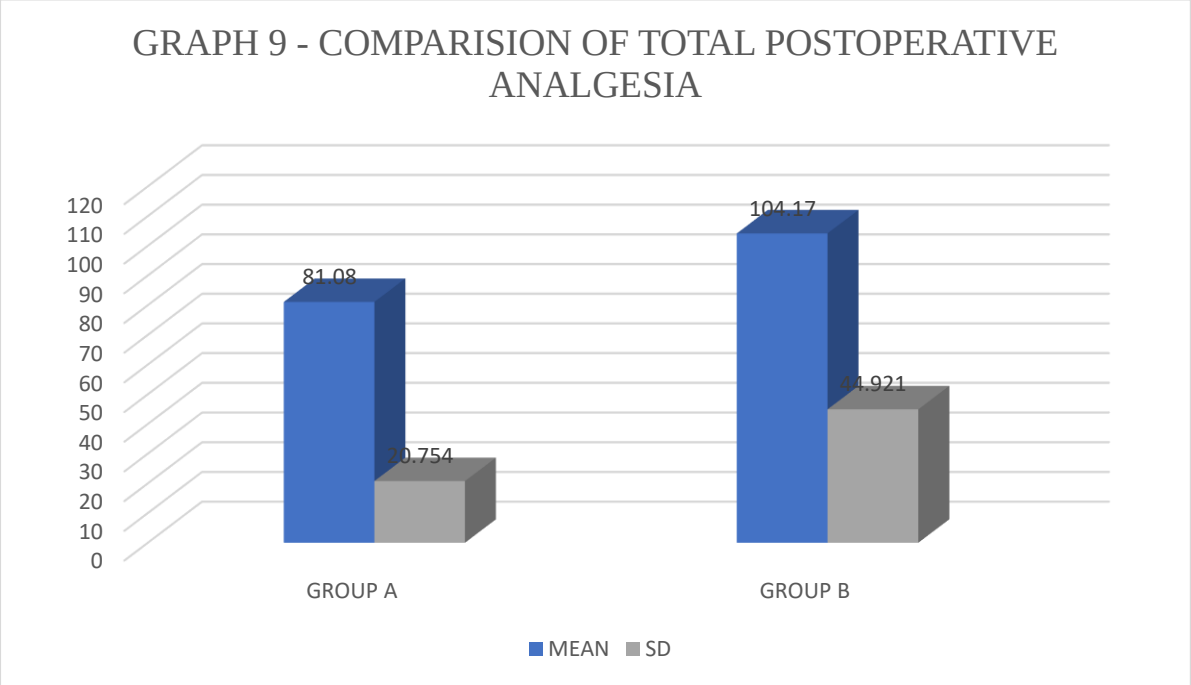
In Group A the mean time-taken in minutes is 688.37±55.75 and Group B it is 593.64±72.56.

Group A has statistically significant longer post operative analgesia when compared to group B.

TOTAL DOSE POST-OPERATIVE ANALGESIA REQUIRED:

TOTAL DOSE OF POSTOPERATIVE ANALGESIA (IN 24H)	MEAN (IN MG)	SD	P VALUE
GROUP A	81.08	20.754	0.006
GROUP B	104.17	44.921	

TABLE 9 – DISTRIBUTION OF TOTAL POSTOPERATIVE ANALGESIA



Total dose of analgesic (diclofenac in mg) consumption in 24 hours in Group A is calculated to be 81.08±20.754 (mean±sd).

Total dose of analgesic (diclofenac in mg) required in 24 hours in Group B is calculated to be 104.17±44.921 (mean±sd).

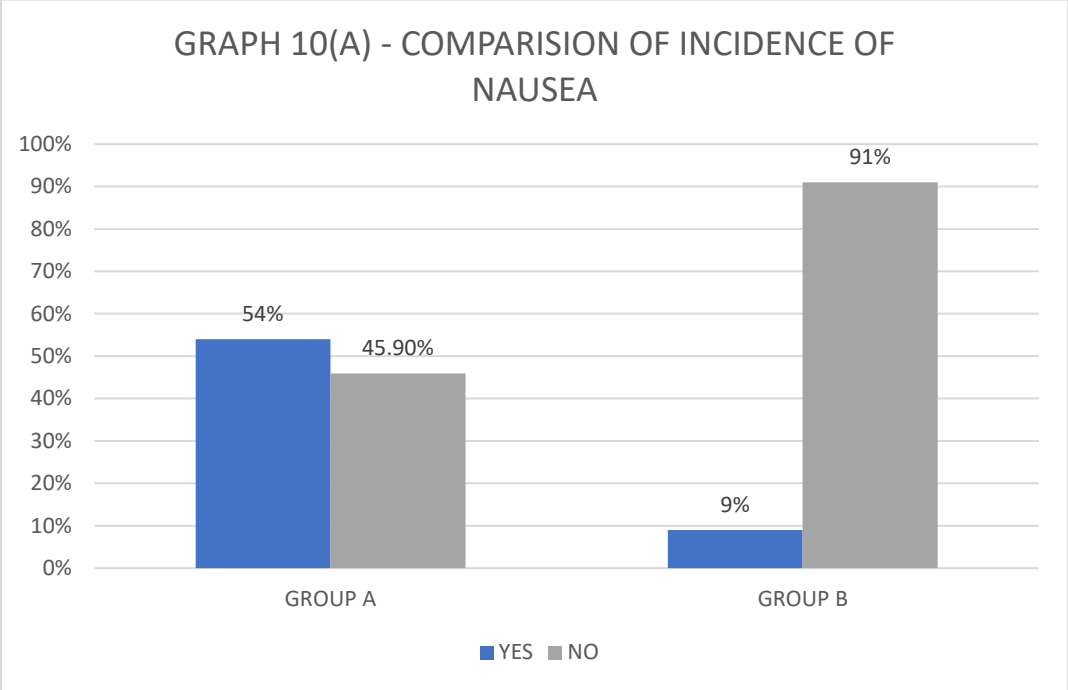
Group A required significantly less dose of analgesic with P value = 0.006.

POST OPERATIVE COMPLICATIONS

1. NAUSEA

NAUSEA	YES	NO	P VALUE
GROUP A	54%	45.90%	0.002
GROUP B	9%	91%	

TABLE 10 (A) – DISTRIBUTION OF PERCENTAGE INCIDENCE OF NAUSEA



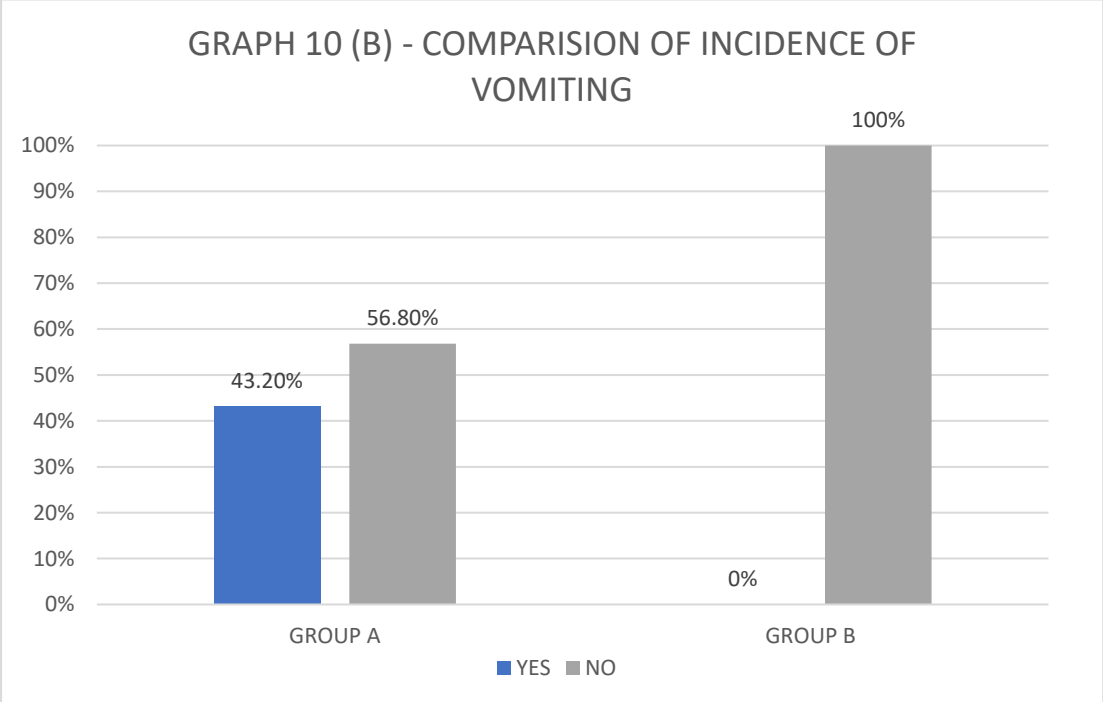
54% of patients in Group A reported nausea while only 9% of patients in Group B complained of nausea postoperatively.

Group B was observed to have significantly reduced incidence of nausea postoperatively with P value = 0.002.

2. VOMITING

VOMITING	YES	NO	P VALUE
GROUP A	43.20%	56.80%	0.001
GROUP B	0%	100%	

TABLE 10 (B) – DISTRIBUTION OF PERCENTAGE INCIDENCE OF VOMITING



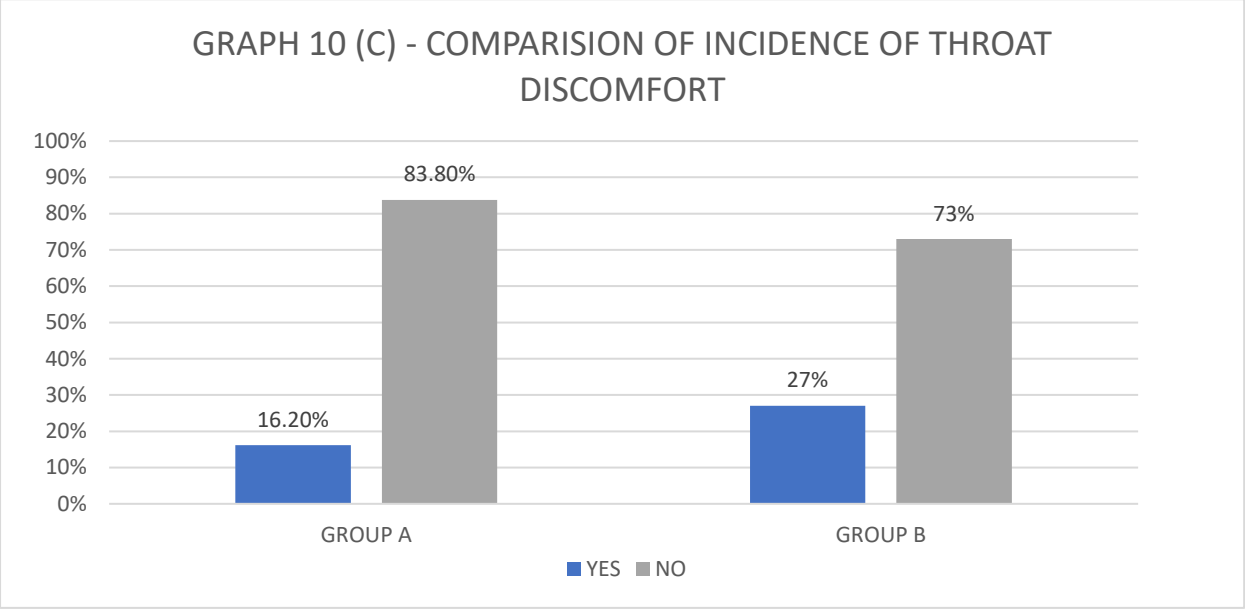
43.20% of patients in Group A had complaints of vomiting while none of the patients in Group B had reported any episodes of vomiting postoperatively.

Group B was observed to have significantly reduced incidence of vomiting postoperatively with P value = 0.001.

3. THROAT DISCOMFORT

THROAT DISCOMFORT	YES	NO	P VALUE
GROUP A	16.20%	83.80%	0.259
GROUP B	27%	73%	

TABLE 10 (C) – DISTRIBUTION OF PERCENTAGE INCIDENCE OF THROAT DISCOMFORT



In Group A, 16.20% of patients reported to have throat discomfort and 83.80% did not experience any throat discomfort.

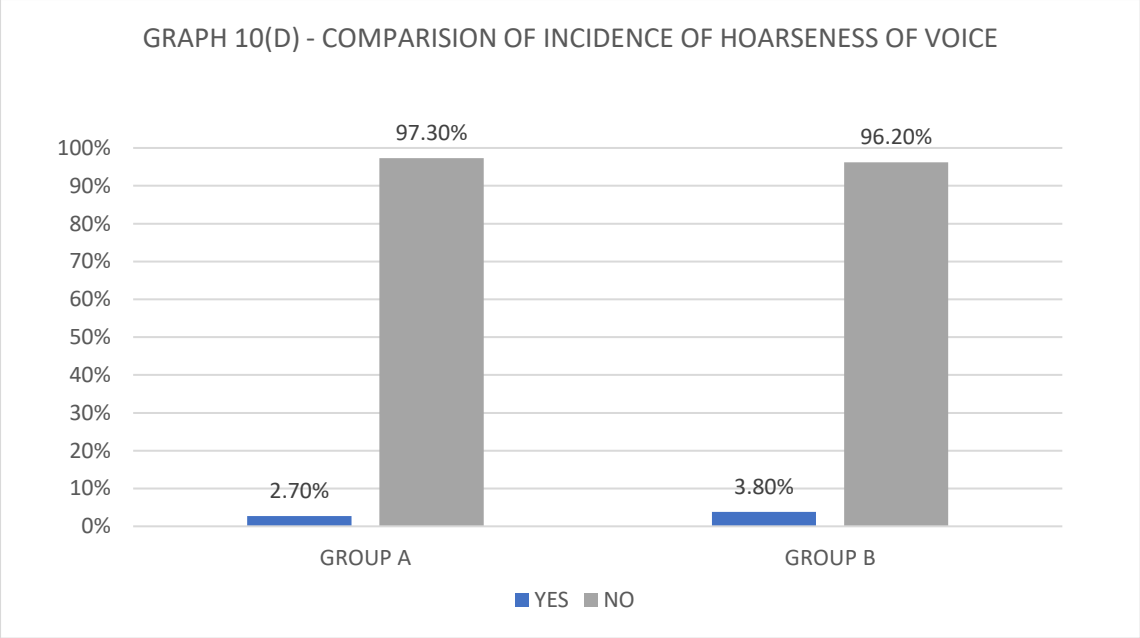
In Group B, 27% of patients experienced sore throat and 73% did not experience any throat discomfort.

The incidence of throat discomfort post-operatively was comparable between two groups with P-value = 0.259.

4. HOARSNESS OF VOICE

HOARSNESS OF VOICE	YES	NO	P VALUE
GROUP A	2.70%	97.30%	0.314
GROUP B	3.80%	96.20%	

TABLE 10 (D) – DISTRIBUTION OF PERCENTAGE INCIDENCE OF HOARSENESS OF VOICE



In Group A, 2.70% complained to have hoarseness of voice while 97.30% had no such complications.

In Group B, 3.80% complained to have hoarseness of voice while 96.20% didn't report any such complications.

The incidence of hoarseness of voice was comparable between Group A and Group B with P-value = 0.314.

DISCUSSION

Thyroid surgeries are being carried out in increasing numbers as they are the main stay modality of treatment for various thyroid diseases. The most common complication in patients undergoing thyroid surgeries is postoperative surgical wound pain particularly in the first 24 hours. Many studies have found that the mean VAS scores following conventional thyroidectomies was 6.9 on a scale of 0 to 10[46].

The post operative pain is usually treated with simple systemic analgesics such opioids or NSAIDS. Treatment with opioids lead to undesirable side effects like nausea, vomiting and respiratory depression eventually increasing the length of duration of PACU and hospital stay.

Using systemic analgesics to treat post thyroid surgery pain at times is inadequate. Thus, acute pain when untreated can lead to development of chronic pain.

Loco-regional anaesthesia is an integral part of multimodal approach which reduces postoperative pain and helps in improving quality of recovery in postoperative patients.

Thus, we proposed a hypothesis that bilateral superficial cervical plexus block might provide postoperative analgesia in the initial postoperative period and can change the amount of systemic analgesia consumption. The advantage of adding adjuvants like dexmedetomidine or dexamethasone avoids the ill effects of using opioids as they reduce their consumption by increasing the duration of analgesia of the block. The advantage of adding adjuvant to local anaesthetics such as dexmedetomidine or dexamethasone is that it negates the ill effects of opioids by increasing the duration of blocks.

The primary purpose of our current study was to compare the analgesic efficacy of dexmedetomidine and dexamethasone as adjuvants to bupivacaine for BSCPB in patients undergoing thyroid surgeries for postoperative pain control.

AGE: In our study, the age of participants in both the groups were comparable. Majority of the patients were between the age of 31-60 years.

GENDER: The female population was higher (87.9%) than male population (12.1%) in our study. This is likely due to the fact that thyroid pathologies being more common in females than males.

A study done by Ökmen K *et al*.[47] which compared efficacy of different concentration and volume of local anaesthetics for SCPB, stressed that the volume of the drug decides the efficacy of the block rather than concentration of the drug. It was determined that higher volume resulted in more effective block. Hence, in our study we chose the volume of drug to be 20ml in total (10ml on each side).

INTRAOPERATIVE HEMODYNAMIC VARIABLES:

After the administration of the block following is the summary of the observations we made:

In our study, intraoperative SBP, DBP, MAP and HR were measured every 10 mins starting from 30 mins from the time of block till the end of surgery or up to 120 minutes whichever is the earliest.

The mean systolic blood pressures had a statistically significant difference between Group A and Group B. Group A had significantly lower mean SBPs at all intervals with P value < 0.05.

When mean diastolic blood pressures were compared, Group A had significantly lower mean DBPs at 30, 40, 50, 60, 70, 80 minutes than Group B with P-value<0.05. The rest of mean DBPs at 90, 100, 110 and 120 minutes were comparable between 2 groups with P-value>0.05.

The means of MAP were significantly lower in Group A when compared with that of Group B at all time intervals with P-value < 0.05.

The means of heart rate at all time intervals were significantly lower in Group A when compared to Group B with P value < 0.05.

Jain. N *et al*.[48], Hassan *et al*.[49] and Achar PB *et al*.[50], have reported the same trends in the hemodynamic parameters being lower in dexmedetomidine group when compared with dexamethasone.

In our study, we also observed that none of our patients, group A and Group B required any analgesia intraoperatively.

The mean VAS scores between 2 groups were observed to be significantly lower in Group A than in Group B at intervals of 30 mins, 1 hour, 2 hours, 6 hours and 8 hours where as they were comparable at 12 and 24 hours. The same is reflected in a study conducted by Thakur *et al*.[51] in 2019, comparing dexmedetomidine and dexamethasone as adjuvants to bupivacaine for TAP block in 120 patients undergoing caesarean section and observed lower VAS scores in patients who received dexmedetomidine as adjuvant. A different observation was made in 2 different studies conducted by Jain. N *et al*.[48] and Gao *et al*.[52] who observed comparable VAS scores when they compared dexmedetomidine and dexamethasone adjuvants with bupivacaine for BSCPB and ESPB respectively.

When we go through the literature, the observations made regarding the analgesic efficacies of the mentioned adjuvants exhibit inconsistency in results.

The mean time period between the administration of BSCPB to first analgesic dose request was observed to be longer in Group A (688±55.75 mins) as compared to that in Group B (593.64±72.56 mins) with a statistical significance (P value = 0.01). Hence, dexmedetomidine prolonged the time to first rescue analgesic dose and provided a longer duration of analgesia than dexamethasone when added as an adjuvant to local anaesthetics for BSCPB.

Similar observations were made by Mohammed Ali DS *et al.*[53] in their study carried out in 84 female patients undergoing TAH. The study concluded that dexmedetomidine as compared to dexamethasone as adjuvant to bupivacaine for ESPB, prolonged duration of analgesia.

Elmaddawy *et al.*[56] carried out a trial where they compared the efficacy of plain bupivacaine-epinephrine with bupivacaine-epinephrine with dexmedetomidine as adjuvant for BSCPB in 2 groups, each group consisted of 21 patients undergoing thyroid surgeries. It was observed that addition dexmedetomidine prolonged duration of pain relief and reduced opioid requirement.

In accordance to our observation, Singla *et al.*[57] observed that addition of dexmedetomidine to bupivacaine for TAP block resulted in prolonged duration of analgesia in patients undergoing caesarean section

Although, research carried out by Adinarayanan S *et al.*[54] in 2019 had a contrasting conclusion. In this study, it was observed that dexamethasone when added to bupivacaine proved to be superior to dexmedetomidine and prolonged the duration of supraclavicular brachial plexus block in patients undergoing upper limb surgeries. Similarly, a study done by Elbahrawy *et al.*[55] (2018) concluded that dexamethasone when added to ropivacaine 0.2% for BSCPB resulted in prolonged duration of block and decreases systemic analgesia requirement.

The mean total dose of postoperative analgesia with injection diclofenac consumed in the first 24 hours was observed to be significantly less in Group A (81.08±20.75) than Group B (104.17±44.92) with P value of 0.006. Thakur *et al.*[51] in their trial made identical observations. The number of requests for rescue analgesic doses were significantly less in patients who received dexmedetomidine with bupivacaine for TAP block than those who received dexamethasone. Mohammed Ali DS *et al*.[53] and Hassan *et al.*[58] also had similar results in their respective trials.

Complementary to the study by Jain. N *et al*.[48] we observed that when dexamethasone was the adjuvant to bupivacaine in patients belonging to Group B, there was significant reduction in the incidence of nausea (P value = 0.002) and vomiting (P value = 0.001) post operatively when compared to Group A who received dexmedetomidine.

Incidence of throat discomfort and hoarseness of voice was comparable between both the groups (P value >0.05)

CONCLUSION:

In conclusion, BSCPB is a simple, easy and effective technique that can be used as one of the postoperative analgesia modalities for patients undergoing thyroid surgeries which when used with adjuvants imparts better and longer analgesia. The analgesia is better and more prolonged with dexmedetomidine. Although, dexamethasone has an advantage over dexmedetomidine in reducing the incidence of postoperative nausea and vomiting.

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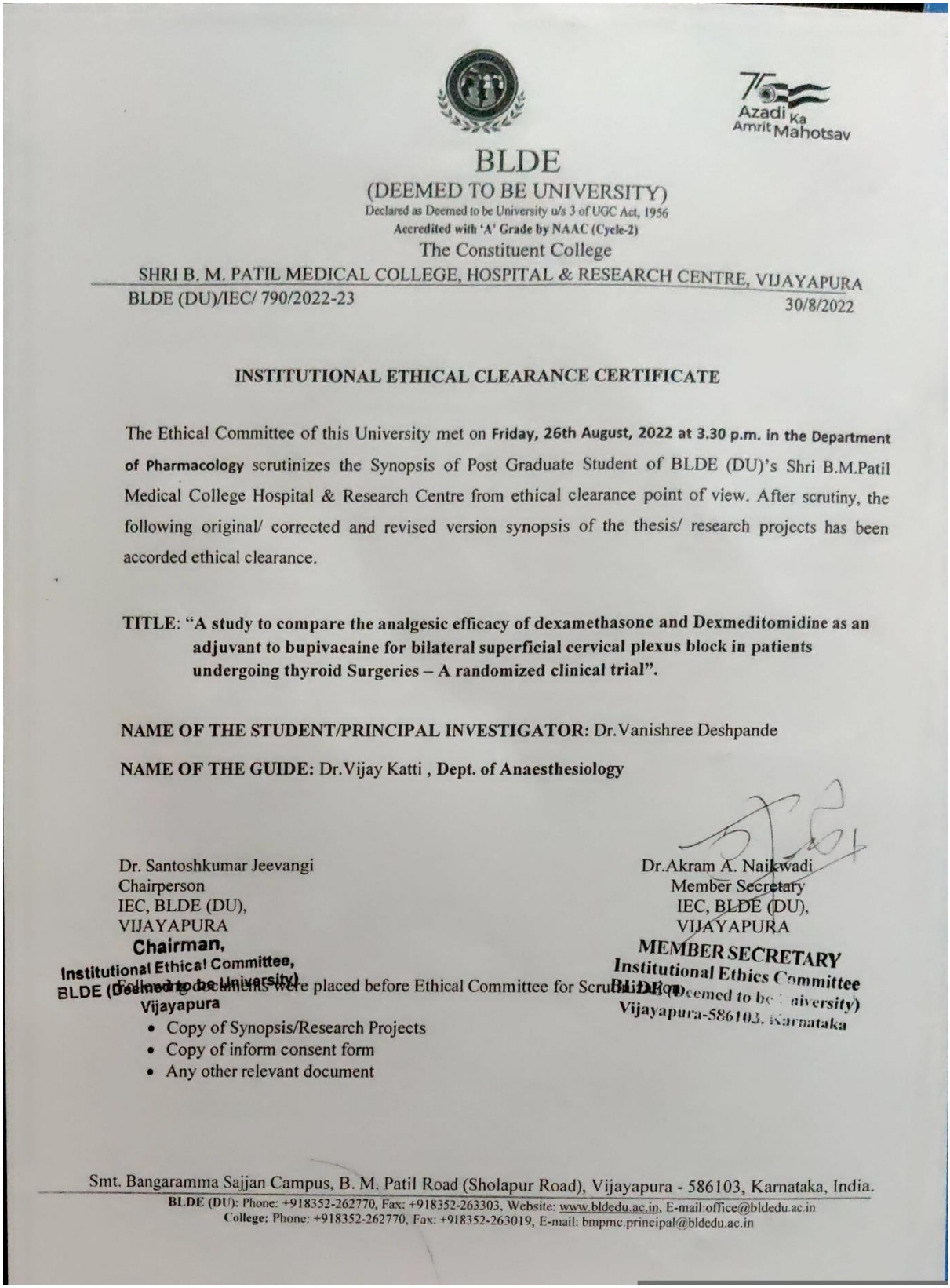
ETHICAL CLEARANCE CERTIFICATE:



BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 790/2022-23 30/8/2022

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Friday, 26th August, 2022 at 3.30 p.m. in the Department of Pharmacology** scrutinizes the Synopsis of Post Graduate Student of BLDE (DU)'s Shri B.M.Patil Medical College Hospital & Research Centre from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "A study to compare the analgesic efficacy of dexamethasone and Dexmeditomidine as an adjuvant to bupivacaine for bilateral superficial cervical plexus block in patients undergoing thyroid Surgeries – A randomized clinical trial".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: Dr.Vanishree Deshpande

NAME OF THE GUIDE: Dr.Vijay Katti , **Dept. of Anaesthesiology**

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutiny:

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE – II

SAMPLE INFORMED CONSENT FORM

B.L.D.E(DEEMED TO BE UNIVERSITY) SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE,

VIJAYAPURA – 586103, KARNATAKA

TITLE OF THE PROJECT: "A STUDY TO COMPARE THE ANALGESIC EFFICACY OF DEXAMETHASONE AND DEXMEDETOMIDINE AS AN ADJUVANT TO BUPIVACAINE FOR BILATERAL SUPERFICIAL CERVICAL PLEXUS BLOCK IN PATIENTS UNDERGOING THYROID SURGERIES – A RANDOMISED CLINICAL TRIAL"

PRINCIPAL INVESTIGATOR: Dr. VANISHREE DESHPANDE

DEPARTMENT OF ANAESTHESIOLOGY,

BLDE'S (DEEMED TO BE UNIVERSITY),

SHRI.B.M. PATIL MEDICAL COLLEGE

HOSPITAL AND RESEARCH CENTRE

VIJAYAPURA–586103.

GUIDE: DR. VIJAY. V. KATTI

PROFESSOR,

DEPARTMENT OF ANAESTHESIOLOGY,

BLDE (DEEMED TO BE UNIVERSITY),

SHRI B. M. PATIL MEDICAL COLLEGE

HOSPITAL AND RESEARCH CENTRE

VIJAYAPURA -586103.

PURPOSE OF RESEARCH:

I have been informed that this study is: "A STUDY TO COMPARE THE ANALGESIC EFFICACY OF DEXAMETHASONE AND DEXMEDETOMIDINE AS AN ADJUVANT TO BUPIVACAINE FOR BILATERAL SUPERFICIAL CERVICAL PLEXUS BLOCK IN PATIENTS UNDERGOING THYROID SURGERIES – A RANDOMISED CLINICAL TRIAL".

I have been explained about the reason for conducting this study and selecting me/my ward as a subject for this study. I have also been given a free choice for either being included or not in the study.

PROCEDURE:

I understand that I will be doing ‘‘A STUDY TO COMPARE THE ANALGESIC EFFICACY OF DEXAMETHASONE AND DEXMEDETOMIDINE AS AN ADJUVANT TO BUPIVACAINE FOR BILATERAL SUPERFICIAL CERVICAL PLEXUS BLOCK IN PATIENTS UNDERGOING THYROID SURGERIES – A RANDOMISED CLINICAL TRIAL."

RISKS AND DISCOMFORTS:

I understand that I/my ward may experience hypotension while doing the procedure, and I understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

I understand that I/my wards participation in this study will help in finding out A STUDY TO COMPARE THE ANALGESIC EFFICACY OF DEXAMETHASONE AND DEXMEDETOMIDINE AS AN ADJUVANT TO BUPIVACAINE FOR BILATERAL SUPERFICIAL CERVICAL PLEXUS BLOCK IN PATIENTS UNDERGOING THYROID SURGERIES – A RANDOMISED CLINICAL TRIAL

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and

audio or video tapes will be used only with my special written permission.
I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time.
Dr. Vanishree Deshpande is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation. If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. And that a copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr. Vanishree Deshpande will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided. I understand that by my agreement to participate in this study, I am not waiving any of my legal rights

I have explained to _____,
the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date:	Dr.VIJAY.V.KATTI	Dr. Vanishree Deshapande
Time:	(Guide)	(Investigator)

STUDY SUBJECT CONSENT STATEMENT

I confirm that Dr. VANISHREE DESHPANDE has explained to me the purpose of this research, The study procedure that I will undergo, and the possible discomforts and benefits that I may experience in my own language. I have been explained all the above in detail in my own language, and I understand the same. Therefore, I agree to give my consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

ANNEXURE – III

SCHEME OF CASE TAKING

PROFORMA

A COMPARITIVE STUDY TO KNOW THE ANALGESIC EFFICACY OF DEXAMETHASONE AND DEXMEDETOMIDINE AS AN ADJUVANT TO BUPIVACAINE FOR BILATERAL SUPERFICIAL CERVICAL PLEXUS BLOCK IN PATIENTS UNDERGOING THYROID SURGERIES – A RANDOMISED CLINICAL TRIAL

Name: Age/ Sex:

I.P No: DATEDate

Group allotted by randomization: Group A / Group B

Type of surgery:

Significant History:

General Physical Examination:

Pallor Y/N Icterus Y/N Cyanosis Y/N Clubbing Y/N

Koilonychia Y/N Lymphadenopathy Y/N Edema Y/N Teeth Y/N

Dentures Y/N

Vital Parameters

Pulse (beats per minute): Blood Pressure:

Respiratory Rate: Temperature:

Systemic Examination

1. CVS 2.RS:

3. C.N.S. 4.Per Abdomen:

Airway Assessment:

Mallampati Grade: Cervical Spine:

Mouth opening: Neck Movement:

A.S.A. Grade:

Investigation

Hemoglobin: TLC:

S. Urea: S. Creatinine:

RBS: Platelet count:

Urine Routine:

Chest Xray: ECG:

Anaesthesia start time:

Block time:

Surgery start time:

Surgery end time:

Time	Heart rate	Blood pressure	Mean arterial pressure
30minutes			
40minutes			
50minutes			
60minutes			
70minutes			
80minutes			
90minutes			
100minutes			
110minutes			
120minutes			

	VAS SCORE
3ominutes	
1hours	
2hours	
6hours	
8hours	
12hours	
24hours	

Time to first Diclofenac Na dose request in minutes	
Total dose of analgesic post operatively	
Duration of analgesia	

Post-operative complications	Yes	No
Nausea		
Vomiting		
Hoarseness of voice		
Throat discomfort		

BIODATA OF THE GUIDE

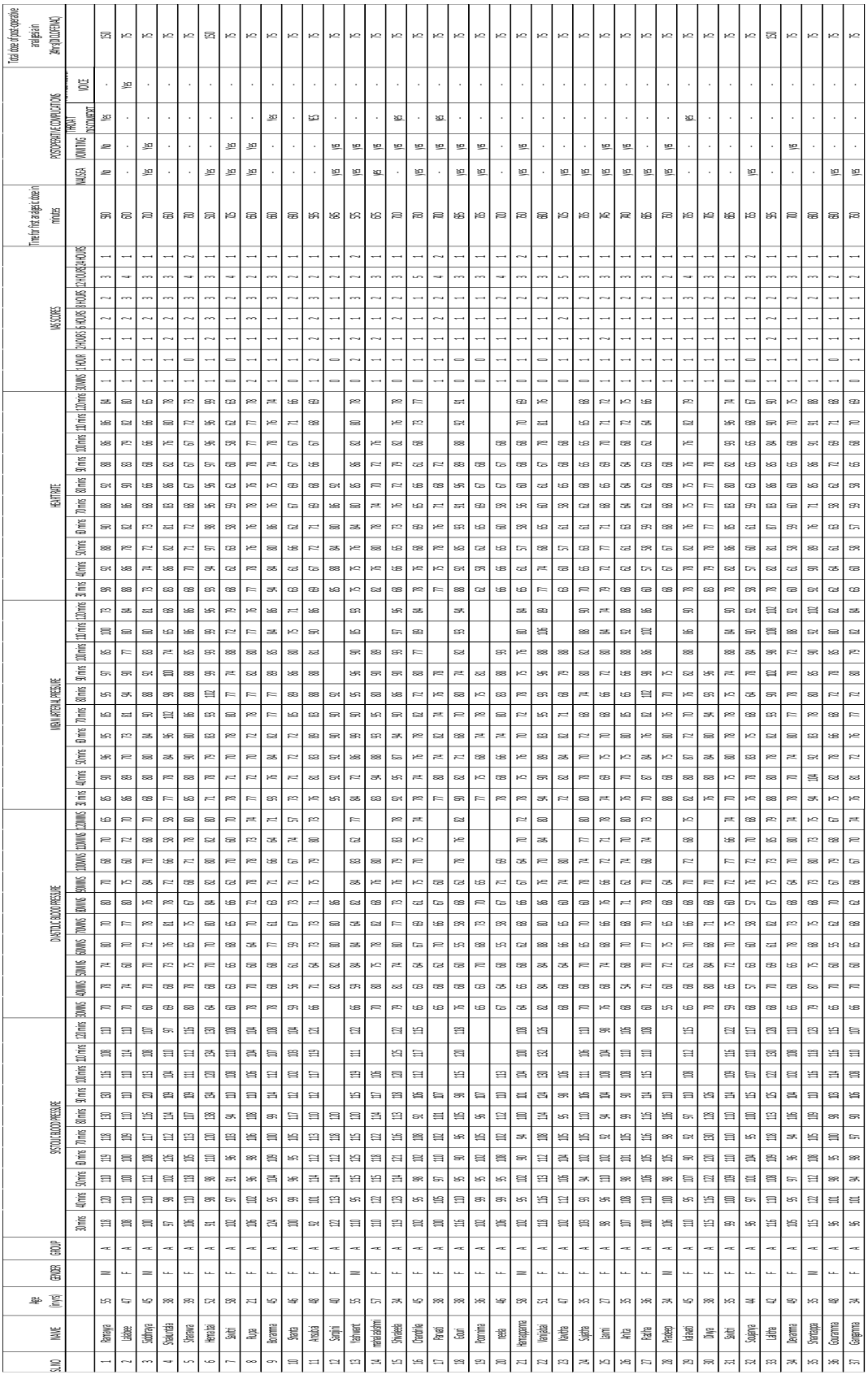
GUIDE NAME:	DR. VIJAY V. KATTI
DATE OF BIRTH:	12/01/1976
EDUCATION:	M.B.B.S. B.L.D.E.A.'s SHRI B.M. PATIL MEDICAL COLLEGE AND RESEARCH CENTRE, VIJAYAPURA – 586103 M.D. ANAESTHESIOLOGY B.L.D.E.A.'s SHRI B.M. PATIL MEDICAL COLLEGE AND RESEARCH CENTRE, VIJAYAPURA – 586103
K.M.C. REG. NO.:	51716
DESIGNATION:	PROFESSOR DEPARTMENT OF ANAESTHESIOLOGY
PUBLICATIONS:	9
TEACHING EXPERIENCE:	20 YEARS
ADDRESS:	PROFESSOR DEPARTMENT OF ANAESTHESIOLOGY B.L.D.E(DEEMED TO BE UNIVERSITY) SHRI B. M. PATIL MEDICAL COLLEGE AND RESEARCH CENTRE, VIJAYAPURA – 586103
PHONE:	(08352)262770 EXT 2052
MOBILE NO:	9844585900
Email:	drvijaykatti@gmail.com

INVESTIGATOR

NAME:	DR. VANISHREE DESHPANDE
QUALIFICATION:	M.B.B.S.
	B.L.D.E.A.'s SHRI B.M. PATIL MEDICAL
	COLLEGE AND RESEARCH CENTRE,
	VIJAYAPURA – 586103
K.M.C. REG. NO.:	138010
ADDRESS:	DEPARTMENT OF ANAESTHESIOLOGY
	B.L.D.E (DEEMED TO BE UNIVERSITY)
	SHRI B. M. PATIL MEDICAL COLLEGE
	AND RESEARCH CENTRE,
	VIJAYAPURA – 586103
Contact Number:	8123278229
Email:	dvanishree14@gmail.com

MASTER CHART

(GROUP A)



MASTER CHART

GROUP B

PLAGARISM CERTIFICATE

iThenticate

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Summary