
**“THE EFFECTIVENESS OF 0.5% ISOBARIC
BUPIVACAINE+DEXMEDETOMIDINE Vs 0.75%
ROPIVACAINE + DEXMEDETOMIDINE FOR
POSTOPERATIVE ANALGESIA USING ULTRASOUND GUIDED
TRANSVERSUS ABDOMINIS PLANE BLOCK FOR
ABDOMINAL SURGERIES UNDER GENERAL ANAESTHESIA :
A RANDOMIZED CONTROL STUDY”.**

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LIST OF ABBREVIATIONS USED:

TAPB- Transverse Abdominus Plane Blocks

TAP- Transverse Abdominus Plane

VAS- Visual Analogue Scale

HR- Heart Rate

NIBP- Non-Invasive Blood Pressure

SpO₂- Saturation percentage of Oxygen

PACU- Post Anaesthetic Care Unit

R-group- Ropivacaine Group

B-group- Bupivacaine Group

NSAID- Non Steroidal Anti-inflammatory Drug

cm- centimeter

LA- Local Anaesthetic

USG- Ultrasonography

II-Ilio-inguinal

IH-Ilio-Hypogastric

BPB-Brachial Plexus Block

GA- General Anaesthesia

Mcg-microgram

Kg-kilogram

CNS- Central Nervous System

WDR- Wide Dynamic Range

NS- Nociceptive Specific

PAG- Periaqueductal grey matter

RVM-Rostral Ventromedial Medulla

V/Q-Ventilation/Perfusion
CO₂-Carbon-di-Oxide
VRS-Verbal Rating Scale
Hz-Hertz
MHz-Mega Hertz
M/s-Metre per Second
L-Litre
ml-Millilitre
mg-Milligram
SD-Standard Deviation
IV-Intravenous
Min-Minute
ASA- American Society of Anaesthesiologists
PAE- Preanaesthetic Evaluation
SBP-Systolic Blood Pressure
DBP-Diastolic Blood Pressure
ECG-Electrocardiogram
N₂O-Nitrous Oxide
MAC-Minimum Alveolar Concentration
Bpm-Beats per minute
H-hour
MW-Mann Whitney test
DEX-Dexmedetomidine

TABLE OF CONTENTS

SL.NO	CONTENTS	PAGE NO
1	INTRODUCTION	1-3
2	AIMS AND OBJECTIVES	4
3	REVIEW OF LITERATURE	5-13
4	BASIC SCIENCES	14-65
5	MATERIALS & METHODS	66-70
6	RESULTS	71-80
7	DISCUSSION	81-86
8	CONCLUSION	87
9	SUMMARY	88-89
10	BIBLIOGRAPHY	90-98
11	ANNEXURES	
	ANNEXURE I - INFORMED CONSENT FORM	99-102
	ANNEXURE II - PROFORMA	103-105
	ANNEXURE III - PHOTOGRAPHS	104-106
	ANNEXURES IV - KEY TO MASTER CHART	107
	ANNEXURES V - MASTER CHART	108-113

LIST OF FIGURES

SL. NO	FIGURES	PAGE NO.
1	Pain Pathway	17
2	Ascending tracts in the spinal cord.	20
3	Organization of the sensory pathway	21
4	Gate control theory of pain.	22
5	Visual Analog Scale	29
6	Muscles of the Abdomen.	36
7	Thoracolumbar fascia	37
8	Dermatomes of the Abdomen	38
9	Various types of the TAP block	42
10	Principles of Ultrasonography	43
11	Out of plane technique	47
12	In-plane Technique	48
13	Chemical structure of Bupivacaine.	51
14	Chemical structure of ropivacaine	55
15	Chemical structure of Dexmedetomidine	60

LIST OF TABLES

Sl. No	TABLES	PAGE NO.
1	Comparison of demographical variables over groups.	71
2	ASA comparison.	72
3	Comparison of surgery	73
4	Comparison of VAS score	74
5	Difference in time for rescue analgesia between groups.	75
6	Vitals over time and groups.	77

LIST OF GRAPHS

Sl. No	GRAPHS	PAGE NO.
1	Gender distribution	71
2	ASA-distribution	72
3	VAS score along duration	75
4	Mean plot of time for rescue analgesia.	76
5	Mean plot of heart rate	79
6	Mean plot of SPO2	79
7	Mean plot of SBP	80
8	Mean plot of DBP	80

LIST OF PHOTOGRAPHS

SL. NO	PHOTOGRAPH	PAGE NO.
1	0.5% Bupivacaine	106
2	0.75% Ropivacaine	106
3	Dexmedetomidine Ampoule	107
4	Performing TAP block	107
5	USG image of TAPB	108

ABSTRACT:

BACKGROUND:

Laparoscopic surgeries have an increased popularity when compared to laparotomy procedures as they have many advantages like less postoperative pain and early recovery. However laparoscopic surgeries are not completely devoid of pain. One such method of analgesia following abdominal surgeries are Transverse Abdominus Plane Blocks (TAPB) performed with the guidance of ultrasound for an accurate block.

AIMS AND OBJECTIVES:

To compare the duration of postoperative analgesia of ultrasound guided TAP Block using 0.5% Bupivacaine + Dexmedetomidine vs 0.75% Ropivacaine + Dexmedetomidine based on VAS scores.

To assess the effect of TAPB on perioperative hemodynamic parameters (HR, NIBP, SpO₂)

METHODS:

68 patients posted for abdominal surgeries were randomized into 2 groups. Routine GA induction with drugs was done for patients, baseline vital signs were documented. At the end of the surgery before extubation, TAP (transverse abdominis plane) block was performed. Patients in group R (n=34) received 15 mL 0.75% Ropivacaine + 10mcg Dexmedetomidine and patients in group B (n=34) received 15 mL 0.5% Bupivacaine + 10 mcg Dexmedetomidine. Postoperatively the patients were assessed for postoperative pain at various intervals with VAS scores.

Postoperative vitals were noted after extubating the patient and shifting the patient to the PACU.

If VAS > 3 rescue analgesia of 1 gram paracetamol was given.

RESULTS:

At 2 hours, pain was slightly higher in the Bupivacaine group, but the difference was not significant (p-value = 0.1033). At 4 hours, pain was significantly higher in the Bupivacaine group compared to the Ropivacaine group (p-value < 0.001). This trend continued at 9 hours (p-value < 0.001), 18 hours (p-value = 0.0020), and 24 hours (p-value < 0.001), with the Bupivacaine group consistently reporting higher pain scores.

Overall, the Ropivacaine group experienced lower pain scores at all time points, indicating better pain relief.

The mean time for rescue analgesia was significantly shorter in the Bupivacaine group (6.85 ± 1.69 hours) compared to the Ropivacaine group (12.24 ± 2.7 hours). The median time was 6 hours (range: 4–10) for Bupivacaine and 12 hours (range: 9–18) for Ropivacaine. The difference between the two groups was statistically significant (p-value < 0.001), indicating that patients in the Ropivacaine group required rescue analgesia much later than those in the Bupivacaine group, suggesting better and longer-lasting pain relief with Ropivacaine.

Heart rate showed no significant difference between groups preoperatively (p-value = 0.2058) or postoperatively (p-value = 0.8439), but both groups had significant changes over time (p-value < 0.001 for Bupivacaine, p-value = 0.0259 for Ropivacaine). SPO₂ levels were similar preoperatively (p-value = 0.5071) and postoperatively (p-value = 0.8575), but both groups showed significant improvement over time (p-value = 0.0378 for Bupivacaine, p-value = 0.0013 for Ropivacaine). SBP did not differ significantly between groups at any time point (p-value = 0.2535 pre-op, p-value = 0.3764 post-op), though both groups had a significant reduction over time (p-value = 0.0051 for Bupivacaine, p-value < 0.001 for Ropivacaine). DBP also

showed no significant difference between groups (p-value = 0.5305 pre-op, p-value= 0.0685 post-op), but only the Ropivacaine group showed a significant decrease over time (p-value < 0.001), while the Bupivacaine group did not (p-value = 0.3130). Overall, both groups exhibited changes in vitals over time, but there was no significant difference between them at specific time points.

CONCLUSION:

0.75% Ropivacaine + Dexmedetomidine produces a more effective and longer duration of postoperative analgesia when compared to 0.5% Bupivacaine + Dexmedetomidine in ultrasound guided TAP block in patients undergoing abdominal surgeries.

The mean duration of analgesia in R-group is around 10 to 12 hours whereas in B-group it is 6 to 8 hours.

INTRODUCTION

Regional anesthetic techniques such as nerve blocks are superior to systemic analgesics due to their reduced side effects.

Management of pain following abdominal surgery typically relies on traditional medications, including paracetamol, NSAIDs, and opioids. However, such medications are associated with adverse effects like hypotension, sedation, and nausea, which can hinder recovery and delay discharge. An alternative approach, such as the addition of a transverse abdominal plane block(TAPB) using local anesthetics and adjuncts, can also be used to help manage pain following surgery more effectively. ⁽⁷⁾

Incision to the abdomen leads to the majority of pain experienced by patients. 12% of patients experience a chronic pain state which effects their daily activities even after a comparatively small incision such as that for inguinal herniorrhaphy if care is not taken on pain. ⁽¹⁾

Many adverse events may occur due to inadequate pain control after surgery such as prolonged bedridden state, pulmonary complications and thromboembolic phenomenon. ⁽¹⁶⁾

Laparoscopic surgeries have gained popularity when compared to laparotomy because of its many advantages like increased patient safety, less postoperative pain and decreased cost. ⁽¹⁷⁾

But laparoscopic surgeries are not completely free of pain. The pain is believed to be due to many factors like diaphragmatic irritation following the creation of pneumoperitoneum with carbon dioxide, the irritating nature of the insufflating gas, pockets of gas which maybe residual in the abdominal cavity, and abdominal wall

tissue damage following the introduction of trocar which leads to the stimulation of peripheral nociceptors⁽²⁾

Several methods such as epidural, opioids, NSAIDs can be used to decrease pain postoperatively. Each of these modalities have their own side effects. NSAIDs have side effects such as renal dysfunction, alteration of hemostasis, gastrointestinal hemorrhage etc. Side effects of Opioids include respiratory depression, decreased gut motility, sedation and increased incidence of vomiting.⁽³⁾

Inhibiting the sensory (afferent) nerves that travel through abdominal muscles to regulate somatic and pain following surgery is how TAP block operates.⁽²¹⁾

The anterolateral abdominal wall innervations originating from T6 to T10 are involved in TAP block⁽³³⁾

TAP block can be given after abdominal laparoscopic surgeries and anterior abdominal wall surgeries.

The Lumbar Triangle of Petit, encircled by iliac crest on inferior side, external oblique on anterior side, latissimus dorsi on posterior side, is where TAPB is performed. A 1 cm defect along the midaxillary line, above the iliac crest, indicates this triangle.⁽⁴⁾ The length of the activity of LA utilized, however, limits the TAP block's efficacy.⁽⁵⁾

LA is deposited between internal oblique and transversus abdominis muscles. By applying the LA above transversus abdominis muscle, the sensory blocking of the neural plexus that supplies muscles and skin of the abdominal wall is achieved. This technique has a higher success rate when ultrasound guidance is used.

Bupivacaine has selective cardiotoxic side effects, it leads to hypotension, dysrhythmias, syncope and cardiovascular collapse. Ropivacaine has a lower toxicity than bupivacaine. It is lipophilic and hence has a greater degree of sensory-motor

differentiation ^(6,18)

Dexmedetomidine, an alpha 2 selective agonist has both sedative and analgesic properties. If given in low doses it can also decrease nausea and vomiting after surgery which usually occurs due to opioid use. ⁽²⁶⁾

Numerous studies have showed the productiveness of TAPB for pain relief post abdomen surgeries.

Previous literatures have compared duration of postoperative pain relief using bupivacaine and ropivacaine, but no adjunct such as dexmedetomidine was added to the local anesthetic agent.

This study compared the duration of postoperative analgesia in TAP block for abdominal procedures using 0.5% Bupivacaine + Dexmedetomidine and 0.75% Ropivacaine + Dexmedetomidine.

OBJECTIVES

Primary objective:

Assessment of postoperative analgesia after TAPB in subjects scheduled for abdominal surgeries using 0.75% Ropivacaine + Dexmedetomidine vs 0.5% Bupivacaine + Dexmedetomidine.

Secondary objective:

Assessing the effect of TAP block on perioperative hemodynamic status (HR, NIBP, SpO₂)

REVIEW OF LITERATURE

1. The article "Chronic pain following surgery: the case of inguinal herniorrhaphy" by "E Aasvang and H Kehlet" explores the issue of continual pain following surgery after inguinal hernia repair. It notes that chronic pain affects about 10-12% of patients who undergo this procedure, significantly impacting their quality of life. The authors identify several mechanisms contributing to chronic pain, including nerve injury, scar tissue formation, and inflammation. They stress on the role of inadequate pain management which leads to chronic pain. The article highlights risk factors such as preoperative pain, psychological factors (like anxiety and depression), and the surgical approach used. It also discusses the importance of early pain control strategies, including local anaesthetics, nerve blocks, and non-opioid analgesics, to reduce the chances of chronic pain. In addition, the authors suggest that a comprehensive understanding of pain mechanisms and improved surgical techniques can help minimize the occurrence of chronic pain. The article calls for a shift toward better preoperative assessments, individualized pain management, and following up the patients after surgery to improve outcomes for patients undergoing hernia repair.

2. The article "Pain after laparoscopy" by "J.I. Alexander", provides a thorough review of pain following laparoscopic procedures. Alexander identifies several factors contributing to pain after laparoscopy, including pneumoperitoneum, which refers to the insufflation of gas into the peritoneal cavity, leading to abdominal discomfort due to peritoneal irritation. Additionally, pain can arise from port site incisions, as the insertion of trocars and instruments through abdominal wall incisions can cause localized pain at the entry points. Visceral stretching also contributes, as the manipulation of abdominal organs during surgery may result in stretching and subsequent pain. The review discusses several methods to reduce pain after

laparoscopy. It has been demonstrated that intraperitoneal local anaesthetics lessen postoperative discomfort. Port site infiltration, where local anaesthetics are injected around port sites, can alleviate localized pain. Using NSAIDs and opioids, is another approach to managing pain. Furthermore, they advised to use multimodal analgesia, which blends several analgesic methods, to minimize adverse effects and provide efficient pain management. The review advocates for a multifaceted strategy for pain relief to enhance recovery following laparoscopic procedures.

3. The study titled "An evaluation of (0.25%) Bupivacaine Vs (0.5%) Ropivacaine for postoperative analgesia using ultrasound-guided transversus abdominis plane (TAP) block for abdominal surgeries" by "Sharma et al. "(2016) compares the benefits of two LA—0.25% Bupivacaine and 0.5% Ropivacaine—administered via TAPB (USG guided) for pain relief in subjects scheduled for abdominal surgeries .The research aimed to assess and compare the onset time, duration of analgesia, and any complications related with the drugs. Results showed that both anaesthetics provided effective analgesia, but Ropivacaine demonstrated a quicker onset and prolonged analgesia in comparison to Bupivacaine. Additionally, Ropivacaine was safer and had little complications, such as reduced risk of motor block. They concluded - Because of its superior efficacy and safety profile over bupivacaine (0.25%), ropivacaine (0.5%) may be a better option for postoperative analgesia following abdominal procedures.

4. The 2001 study by Rafi AN introduces a novel abdominal field block technique using the lumbar triangle, an area between the iliac crest, lower ribs, and posterior axillary line. This method targets the sensory nerves of the anterior abdominal wall, offering a safer and simpler alternative to traditional nerve blocks like the TAP and ilioinguinal/iliohypogastric blocks. The technique is less technically challenging, with

decreased side effects like vascular puncture, nerve injury. Clinical results showed effective relief of pain following surgery, decreasing the need for additional medication and enhancing recovery. This approach has become a valuable addition to regional anesthesia for abdominal surgeries.

5 “Hebbard et al.” examine requirement of USG for TAPB to provide pain relief following abdominal surgeries. Ultrasound improves needle placement accuracy and safety by offering real-time visualization of the target tissue. The TAPB blocks anterior abdominal wall nerves, offering pain relief without significant motor blockade. The study shows that USG guided TAPB reduces pain following surgery and opioid use, leading to faster recovery. The approach enhances precision, minimizes complications, and ensures effective local anesthetic spread. It is particularly useful in lower abdominal surgeries like cesarean sections and hernia repairs. This research contributed to the broader adoption of ultrasound in regional anesthesia.

6. The 2000 study by “Wildsmith JAW”, titled "Relative potencies of ropivacaine and bupivacaine", compares the effectiveness and safety of two commonly used local anesthetics, ropivacaine and bupivacaine, in regional anesthesia. The study shows that ropivacaine’s potency is lower than bupivacaine in achieving sensory blockade at similar concentrations, requiring higher doses for the same effect. However, ropivacaine is less likely to cause motor impairment at clinically effective doses. The research also highlights that ropivacaine has a lower risk of cardiovascular toxicity, especially in cases of accidental intravascular injection, making it a safer choice compared to bupivacaine, particularly for high-risk patients or extensive regional blocks. Clinically, while bupivacaine remains more potent for sensory blockade, ropivacaine is a safer alternative, especially when motor blockade should be

minimized. The study confirms ropivacaine as a less potent but safer option for regional anesthesia, providing clinicians with an important alternative when selecting the most suitable anesthetic for a patient or procedure.

7. The study by “Sahin AS, Akay MK, Demiraran Y, et al”., titled "Analgesic effects of ultrasound-guided transverse abdominis plane block using different volumes and concentrations of local analgesics after laparoscopic cholecystectomy" explores varying volumes and strengths of LA used in USG guided TAPB for pain control in patients undergoing laparoscopic cholecystectomy. The participants were allocated into different categories by basis of concentration and volume of local anaesthetic (either bupivacaine or ropivacaine) administered through the TAP block. The aim was to study how these variables influenced pain control following surgery. The study revealed that both volume and strength of local anaesthetic had a pronounced impact on the analgesic outcomes. Higher volumes and concentrations provided better analgesia, reducing the need for other analgesics after surgery. The group receiving higher volumes and concentrations of local anaesthetic also experienced prolonged pain relief and a reduced incidence of pain following surgery. In particular, the study found that ultrasound guidance was crucial for the accurate administration of the TAP block, ensuring precise injection of the drug and improving the overall efficacy of the procedure. The study found that for patients undergoing laparoscopic cholecystectomy, the ideal amount and strength of LA for a TAPB relies on achieving good pain control without complications like local anaesthetic toxicity. In summary, the research demonstrated that ultrasound-guided TAP blocks using appropriate volumes and strengths of LA lead to adequate pain control post laparoscopic cholecystectomy , with higher volumes and strength offering better pain control. This

approach contributes to a reduction in opioid use and enhances patient recovery following surgery.

8. The study by “Aveline C, Hetet HL, Roux P, Vautier F, Cognet E, and Vinet,” titled "Comparison between ultrasound-guided transversus abdominis plane and conventional ilioinguinal/iliohypogastric nerve blocks for day-case open inguinal hernia repair”, sought to determine if USG guided TAPB or traditional ilioinguinal/iliohypogastric (II/IH) nerve blocks were more effective to provide comfort after surgery.

. The participants were divided to receive either an USG guided TAPB or the conventional II/IH block for pain management. The goal was to assess and compare both techniques in terms of analgesic intake, and complications.

The analysis showed that both methods provided effective analgesia, but the ultrasound-guided TAP block had some distinct advantages. Patients who received the TAPB had lower pain scores in the immediate postoperative period. Furthermore, these patients required less supplemental analgesia, leading to a decrease in the use of opioids compared to those receiving the conventional nerve block.

In addition, the TAP block was found to be technically easier and safer to perform using ultrasound guidance, as there was better visualization of the needle and the surrounding anatomy, decreasing the risk of complications. The II/IH nerve block, while effective, had a higher variability in the quality of the block, potentially leading to less consistent pain relief.

In terms of pain alleviation, opioid-sparing effects, and convenience of use, the study found that USG-guided TAP block outperformed traditional ilioinguinal/iliohypogastric blocks. Given its capacity to deliver efficient, long-lasting

analgesia with fewer side effects, the authors suggested TAPB as a useful substitute for pain control.

9. The study by “Alsharari AF, Abuadas FH, Alnassrallah YS, and Salihu D,” titled “Transversus abdominis plane block as a strategy for effective pain management in patients with pain during laparoscopic cholecystectomy: a systematic review” assesses the success of TAPB as a method of pain relief in patients undergoing laparoscopic cholecystectomy. Key outcomes, including pain scores, opioid use, and overall post-operative patient satisfaction, were the main focus of the review. The TAPB, was analyzed for its ability to reduce both intraoperative and postoperative pain. According to the review, TAPB minimized opioid use and post-procedural discomfort in patients undergoing laparoscopic cholecystectomy. Furthermore, the TAP block was found to offer good analgesia, especially in managing the somatic pain associated with the abdominal wall, which is common after laparoscopic procedures. Additionally, the systematic review highlighted that the TAP block had a favorable safety profile, with minimal complications when performed correctly, particularly under ultrasound guidance. However, the authors noted that there was variability in the quality of the blocks and the effectiveness of pain relief across different studies. The review concludes that TAP block is an excellent way to control pain in those scheduled for laparoscopic cholecystectomy. It suggests that TAP block could play a significant role in enhancing recovery, reducing opioid use, and improving patient outcomes. However, the authors also recommend further research to standardize techniques and explore long-term outcomes to optimize their use in clinical practice.

10. The study by “McDonnell JG, Donnell G, Curley A, Heffernan C, Power JG, and Laffey”, titled "The analgesic efficacy of transversus abdominis plane block after abdominal surgery: a prospective randomized controlled trial", was published in 2007. They aimed to study effectivity of TAPB for analgesia post-abdominal surgery. The participants received either a TAPB or a control treatment. Comparing the TAPB to the control group, the results demonstrated a considerable reduction in pain levels following surgery. The TAPB was effective in lowering the requirement for systemic analgesics since patients who had it used less opioid drugs. Furthermore, the TAP block significantly decreased postoperative somatic and visceral pain, enhancing patients' overall quality of recovery. Additionally, the TAPB was linked to a good safety profile with little adverse effects, according to the study.

This demonstrated the block's potential as a secure and efficient substitute for more conventional analgesic methods, like parenteral opioids or epidural anaesthesia, which have a higher risk of adverse effects and complications. The TAPB is an efficient way for postprocedural pain after abdominal surgery. According to the study, the TAP block is a useful regional anaesthetic option for patients having abdominal procedures in order to improve pain management, decrease narcotic use, and speed up recovery. For improved pain management following such surgeries, the authors suggested a wider application of TAPB in clinical practice.

11. "Marhofer et al." (2013) investigated the effects of dexmedetomidine on the quality and duration of peripheral nerve blocks performed in combination with ropivacaine. There were three groups: Group R received simply ropivacaine, Group RsD received systemic dexmedetomidine and ropivacaine, and Group RpD received perineural dexmedetomidine and ropivacaine. The duration of sensory and motor blocks and onset times for the motor and sensory blocks were studied.

The results showed that participants treated with perineural dexmedetomidine experienced a faster motor onset time and a considerably longer duration for both motor and sensory blocks compared to those treated with Ropivacaine alone. A longer block duration was possible with systemic dexmedetomidine administration, but the impact was less pronounced than with perineural injection. The study came to the conclusion that, in comparison to single administration, the adjunct administration of dexmedetomidine during a peripheral nerve block with ropivacaine offers a better onset and duration of anaesthesia. Compared to systemic treatment, perineural administration produced superior results. Dexmedetomidine's usage as a helpful adjuvant in regional anaesthesia is thus supported.

12. The 2017 study by “Vorobeichik et al” was conducted to evaluate the effectiveness of perineural dexmedetomidine as a supplement to LA in BPB, they carried out a systematic review. Results such as the length of sensory and motor block, amount of analgesic used, intensity of pain, and adverse effects were assessed. Although there wasn't a statistical significance, the study demonstrated that dexmedetomidine considerably extended the time of the sensory blockade by roughly 284 minutes. The lengths of the sensory and motor blockade, & the time before the initial request for analgesia, were prolonged. No occurrences of respiratory depression were observed, blood pressure was unaffected, and bradycardia occurred in 7% of individuals. Bradycardia should be taken into consideration as a possible side effect, although the study found that dexmedetomidine can improve brachial plexus nerve blocks by extending block durations and lowering analgesic usage.

13. The study by Paudel et al.” (2021) aimed at comparing effectiveness of postoperative analgesia in TAPB using USG versus local infiltration at port sites in participants listed laparoscopic surgery under GA. There were two divisions. While

the other group got local infiltration, the first group received TAPB. The VAS was utilised to assess degree of discomfort. Additionally noted were total amount of opioids consumed and the period since the initial request for rescue analgesia. According to the findings, the TAPB group used less analgesics and opioids and had considerably lower resting pain levels than the local infiltration group. The study found that, in comparison to local infiltration at port locations, ultrasound-guided subcostal TAPB is a useful, dependable, & efficient method for delivering analgesia in patients having laparoscopic surgeries.

Combining dexmedetomidine with ropivacaine in TAPB for postoperative analgesia in participants enlisted for laparoscopic surgery was studied by Sarvesh et al.⁽³²⁾ 0.375% ropivacaine with 1 mcg/kg dexmedetomidine was administered to one division of patients, and another division received 0.375% ropivacaine alone. VAS was used to analyse pain levels, and records were kept of opioid use and period of request for rescue analgesia. According to the findings, the ropivacaine + dexmedetomidine group needed less opioid medication and had considerably lower VAS pain levels than the ropivacaine group. The research concluded that, dexmedetomidine +ropivacaine in TAPB improves postoperative analgesia and lowers opioid intake without having much appreciable negative effects.

BASIC SCIENCES

Pain is a complex sensory, emotional, cognitive and behavioral phenomenon. According to the International association for the study of pain as “an unpleasant sensory and emotional experience associated with actual tissue damage, or described in terms of such damage”.^[35]

The term “nociception” [Latin - noci means harm or injury] is used only to describe the neural response to traumatic or noxious stimuli. Nociceptive pain aims to detect, localize and limit the tissue damage.

Pain can be acute, chronic or even transient. Acute pain is primarily due to nociception and chronic pain may be due to nociception, also it has psychological and behavioural factors.

Acute pain:

Acute pain is caused by noxious stimuli due to injury, disease process, or the abnormal function of muscle or viscera. It is self-limited, usually heals within few days to weeks. Failure of resolving acute pain progresses to chronic pain.³⁶ Postoperative pain, obstetric pain, post traumatic pain and medical illness like acute pancreatitis are examples of acute pain.

There are two types of acute pain: somatic and visceral pain.

Somatic pain:

Somatic pain is due to nociceptive input arising from skin, subcutaneous tissues, and mucous membranes. It is characterized by being localized and described as sharp, pricking, throbbing or burning sensation.³⁷

It is further classified into superficial and deep.

Superficial somatic pain is due to nociceptive input arising from skin, subcutaneous tissues, and mucous membranes. It is well localized and described as a sharp, pricking, throbbing, or burning sensation.

Deep somatic pain arises from muscles, tendons, joints, or bones. It is dull, aching pain and less well localized.³⁶

Visceral pain:

Visceral pain is due to nociceptive input arising from internal organ or from the structures covered by the organs. It is dull diffuse pain, which is frequently associated with abnormal sympathetic or parasympathetic activity causing nausea, vomiting, sweating and /or changes in blood pressure or heart rate.

Four subtypes are described: (1) true localized visceral pain, (2) localized parietal pain, (3) referred visceral pain, and (4) referred parietal pain.

Parietal pain is typically sharp and often described as a stabbing sensation that is either localized to the area around the organ or referred to a distant site. The phenomenon of visceral or parietal pain referred to cutaneous areas results from patterns of embryological development and migration of tissues, and the convergence of visceral and somatic afferent input into the central nervous system. Thus, pain associated with disease processes involving the peritoneum or pleura over the central diaphragm is frequently referred to the neck and shoulder, whereas pain from disease processes affecting the parietal surfaces of the peripheral diaphragm is referred to the chest or upper abdominal wall.³⁶

Chronic pain:

Chronic Pain is the pain that persists beyond the expected time of healing and is usually due to long standing inflammation or neuropathic pain.

Chronic pain can be nociceptive, neuropathic or mixed. This type of pain can last longer even after injury or disease process heals itself, as Pain signals remain active in the nervous system for weeks, months or years. Most common causes of chronic pain include headache, musculoskeletal disorders such as arthritis, back pain, nerve lesions, cancer pain, etc. ³⁶

Neurophysiology of pain:

Pain experience involves a complex neurophysiologic process known as nociception, which includes four distinct components: transduction, transmission, modulation, perception.

Transduction is the process by which noxious stimuli is converted to an electrical impulse in sensory nerve endings. **Transmission** is the conduction of these electrical impulses to the CNS with various nerve connections in the spinal cord and brain. **Modulation** is the process of altering pain transmission it can either inhibitory or excitatory mechanism. **Pain perception** is mediated by thalamus which acts as a central relay station for incoming pain signals. Pain can also be experienced in the absence of these steps. ³⁸

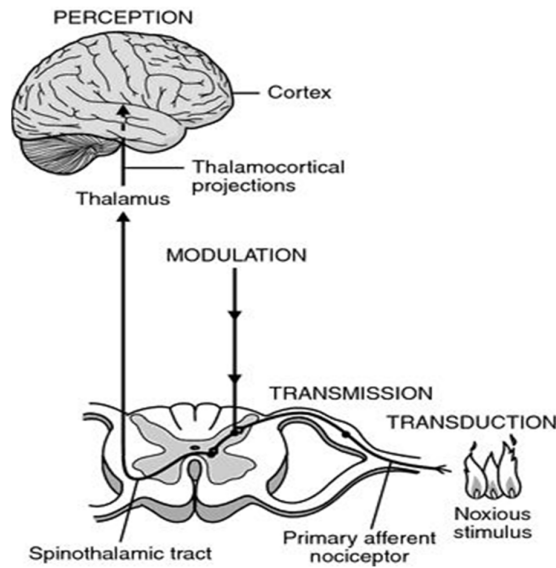


Figure 1: Pain Pathway

Nociceptors (Pain Receptors)

Nociceptors are specialized primary afferents that react to intense, noxious stimuli in skin, muscles, joints, viscera, and vasculature. They generally provide information to the CNS about the site and strength of noxious stimuli. Nociceptors are inactive in normal tissues until they are being stimulated above their resting threshold.⁽³⁹⁻⁴²⁾

Types:

- 1) Mechano nociceptors- pinch and pinprick
- 2) Silent nociceptors – Inflammation
- 3) Polymodal mechanoheat nociceptors – most prevalent and responds to excessive pressure, extremes of temperature, mechanical and chemical stimuli

Transmission to the spinal cord

Pain is transmitted from peripheral nociceptors to the spinal cord and higher structures in the CNS involving several pathways, numerous receptors, neurotransmitters, and secondary messengers.

First order neuron:

Cell bodies of these neurons are present in the dorsal root ganglion, they are pseudo unipolar neurons and have one axon which divides into a peripheral and central branch. Impulses are transmitted through the axons to the spinal cord. ⁽³⁵⁻

^{37,43)} **Second order neurons**

They carry signals from the spinal cord to the thalamus. Cell bodies of these neurons are present in the Rexed laminae (dorsal horn is anatomically organized in the form of layers or laminae) of the spinal cord, or in the nuclei of the cranial nerves within brainstem.

The unmyelinated C fibers terminate in the most superficial lamina I and II, the thinly myelinated A delta fibers in the lamina I, III to V, whereas collaterals of the large myelinated fibers A beta terminate in lamina III to V. Lamina I and II are known as marginal nucleus and substantia gelatinosa of Rolando respectively. ^{43,37}

Two predominant types of second order neurons:

- Wide dynamic range (WDR)
- Nociceptive specific (NS)

WDR cells are found in the deeper laminae III to V. They receive inputs from both low threshold A beta and nociceptive A delta and C fibers. These are activated by both inoffensive and noxious stimuli. NS cells are found in the

superficial laminae I and II. They respond only to noxious stimuli under physiologic conditions.^{43,37}

The axons of both the WDR and NS second order neurons, they cross the midline near the cell bodies form bundles of ascending fibers in the contralateral, anterolateral spinal region and ascend cranially in the spinothalamic to the ventral posterolateral nucleus of the thalamus.^{43,37}

Third order neurons:

They carry signals from the thalamus to primary sensory cortex. Cell bodies of these are present in ventro-posterolateral area of thalamus.^{36,37,43}

Ascending tracts in the spinal cord, there are two main pathways that carry nociceptive signals to higher centres in the brain.^{38,44}

- The spinothalamic tract secondary afferent neurons decussate within a few segments of the level of entry into the spinal cord and ascend in the contralateral spinothalamic tract to nuclei within the thalamus. Third order neurons then ascend to terminate in the somatosensory cortex. There are also projections to the periaqueductal grey matter (PAG). The spinothalamic tract transmits signals that are important for pain localization.
- The spinoreticular tract fibres also decussate and ascend the contralateral cord to reach the brainstem reticular formation, before projecting to the thalamus and hypothalamus. There are many further projections to the cortex. This pathway is involved in the emotional aspects of pain.
- In addition to the A δ and C fibres that carry noxious sensory information, there are primary afferent A β fibres that carry non-noxious stimuli. Each of these fibre types possesses different characteristics that allow the transmission of

particular types of sensory information. A β fibres are highly myelinated and of large diameter, therefore allowing rapid signal conduction. They have a low activation threshold and usually respond to light touch and transmit non-noxious stimuli.

- A δ fibres are lightly myelinated and smaller diameter, and hence conduct more slowly than A β fibres. They respond to mechanical and thermal stimuli. They carry rapid, sharp pain and are responsible for the initial reflex response to acute pain.
- C fibres are unmyelinated and are also the smallest type of primary afferent fibre.

Hence, they demonstrate the slowest conduction. C fibres are polymodal, responding to chemical, mechanical and thermal stimuli. C fibre activation leads to slow, burning pain.

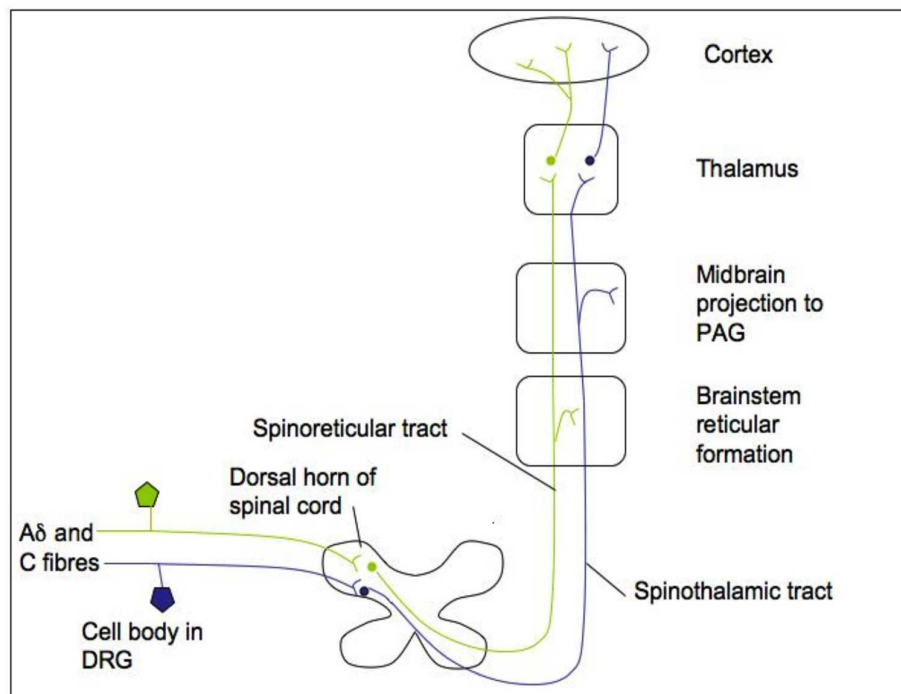


Figure 2: Ascending tracts in the spinal cord.

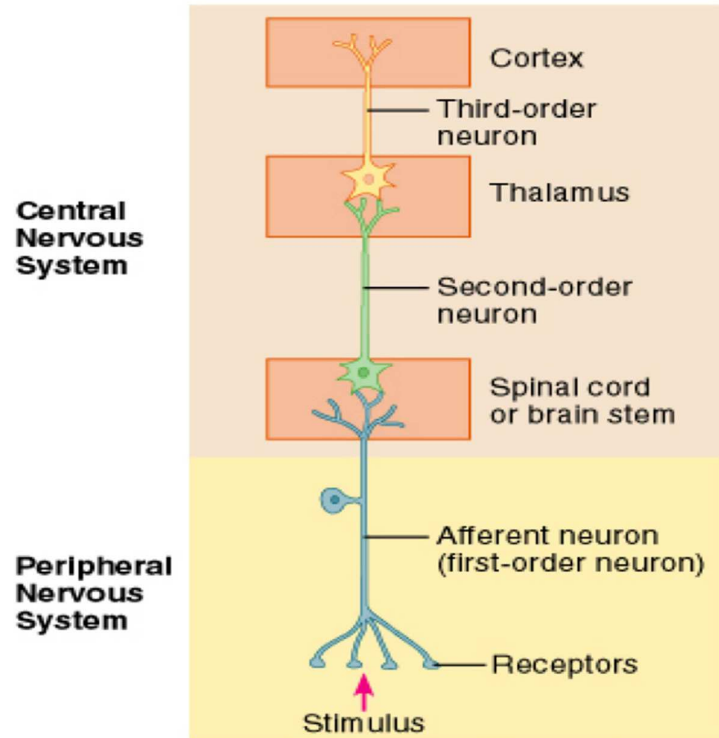


Figure 3: Organization of the sensory pathway

Inhibition of pain transmission ^{35,38,44}

There are mechanisms that act to inhibit pain transmission at the spinal cord level and via descending inhibition from higher centres.

Gate control theory of pain:

The gate control theory of pain was proposed by Melzack and Wall in 1965 to describe a process of inhibitory pain modulation at the spinal cord level. By activating A β fibers with tactile, non-noxious stimuli inhibitory interneurons in the dorsal horn are activated leading to inhibition of pain signals transmitted via C fiber.

Descending inhibition:

The periaqueductal grey (PAG) in the midbrain and the rostral ventromedial medulla (RVM) are two important areas of the brain involved in descending

inhibitory modulation. Both these centres contain high concentrations of opioid receptors and endogenous opioids, which helps explain why opioids are analgesic.

Descending pathways project to the dorsal horn and inhibit pain as well as pain transmission. These pathways are monoaminergic, utilising noradrenaline and serotonin as neurotransmitters.

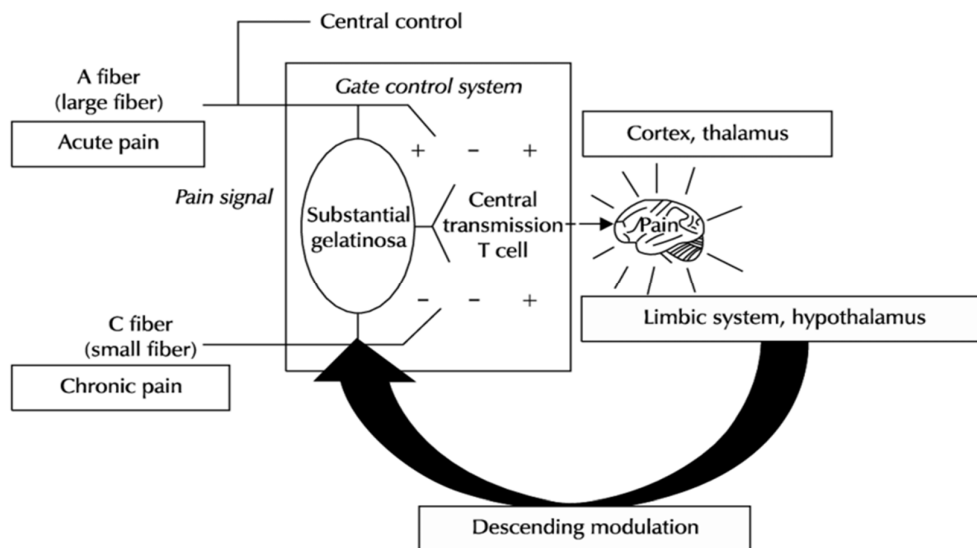


Figure 4: Gate control theory of pain.

POSTOPERATIVE PAIN ⁴⁵⁻⁵²

Postoperative pain is one of the major concerns of patients. Moderate to severe acute pain can affect organ function nearly and can cause postoperative morbidity and mortality. Inadequate pain management could be associated with various respiratory, cardiovascular, gastrointestinal and psychologic complications. Thromboembolic events can happen because of reduced mobility due to postoperative pain.

Cardiovascular system

It increases heart rate, blood pressure, systemic vascular resistance and myocardial irritability, increases oxygen demand, can precipitate infarction and arrhythmias.

Respiratory system

It increases oxygen consumption and carbon dioxide production leading to increased minute ventilation. It reduces Functional Residual Capacity leading to atelectasis, V/Q mismatch and hypoxemia.

Hematological system:

Pain reduces fibrinolysis and hypercoagulability and causes increased incidence of thromboembolic phenomenon.

Gastrointestinal system:

Pain increases sympathetic tone leading to raised sphincter tone and reduces motility leading to paralytic ileus and urinary retention. It also increases acid secretion causing stress ulceration.

Endocrine:

It increases catabolic hormones like catecholamines, cortisol and glucagon and reduces anabolic hormones like insulin, testosterone.

Immune system:

It causes leukocytosis with lymphopenia and depresses reticuloendothelial system and decreases immune function.

Psychological manifestations like anxiety, disturbed sleep, depression and anger.

Pain after open surgeries is usually somatic in origin, whereas pain following laparoscopic surgeries is of both somatic and visceral in origin. Laparoscopic surgeries are minimally invasive, less pain compared to open surgeries but not completely pain free.

Mechanism of pain in laparoscopic surgeries ⁵³⁻⁵⁷

Early postoperative pain is the most troublesome that it requires strong analgesia including opiates after elective laparoscopic surgeries. Many efforts have been made to improve postoperative analgesia, but postoperative pain, however, does not completely disappear and several studies have shown that visceral pain is the major component. Nonetheless, pain may be moderate or even severe for some patients during the first 24 postoperative hours, and has frequently been treated with nonsteroidal anti-inflammatory drugs (NSAIDs) or opioid treatment.

The exact etiology of pain after laparoscopic surgeries is still unclear; however, it appears to be multifactorial. The causes include abdominal wall trauma by trocar entrances, diaphragmatic irritation secondary to CO₂ insufflation and pneumoperitoneum, type and temperature of insufflated gas, residual intraperitoneal gas, intraabdominal trauma, micro ruptures of the parietal peritoneum due to abdominal distension, chemical irritation of the peritoneum, etc.

Therefore, abdominal distention should be slow with adequate muscle relaxation to ensure suitable abdominal compliance. The prolonged presence of shoulder tip pain suggests excitation of the phrenic nerve that is caused by the persistence of gas in the abdomen (pneumoperitoneum). There is statistically

significant correlation between the width of the gas bubble and pain score, and this pain can be reduced by the aspiration of the gas under the diaphragm.

Factors associated with gaseous pneumoperitoneum ^{56,57}

1. Neuropraxia of the phrenic nerve

It has been suggested that distention of the diaphragm during gas insufflations and the resultant phrenic nerve neuropraxia possibly contribute to postoperative pain, which may include the related C4 dermatome.

2. The type of insufflated gas and intraabdominal pH

The phrenic nerves may be damaged by the acid milieu created by the dissolution of CO₂. The intraperitoneal pH when CO₂ gas is insufflated has been measured to be 6.0 immediate postoperatively. On the first postoperative day, the pH rises to 6.4 to 6.7, and on the second postoperative day to 6.8 to 6.9. Thereafter it normalizes to above 7.0. Similar values were found when argon gas was substituted.

3. Residual intraabdominal gas

Several reports have indicated that residual intraabdominal gas after laparoscopy causes pain. Carbon dioxide dissolution, intraabdominal acidosis, and the consequent peritoneal irritation occur for a longer period if the gas is not evacuated at the end of the laparoscopic procedure. Residual gas also may result in a loss of peritoneal surface tension and support to the abdominal viscera, thus contributing to postoperative pain.⁽⁵⁶⁾

4. Temperature of gas

The effect of gas temperature on postoperative pain after gynaecologic laparoscopic procedures has been investigated in a prospective randomized study of standard insufflation gas (20⁰ C) versus gas at body temperature. This study found that pain reduction was significantly greater for those patients in whom warmed gas was used, especially with respect to diaphragmatic and shoulder tip pain, with the lasting effect of three days.⁵⁴

5. Humidity of gas

A prospective randomized controlled trial was conducted at the Queen Elizabeth Hospital, Adelaide, to investigate the outcome when humidified gas was insufflated during laparoscopic cholecystectomy instead of standard dry gas. This study demonstrated significantly reduced postoperative pain in patients who underwent humidified gas insufflation. The humidified insufflations showed a trend of less post-operative analgesic consumption, along with shorter hospital stay and earlier return to work. The exact relation between dry gas and postoperative pain is not yet determined, but other animal studies have observed that dry gas insufflation is implicated in ultrastructural damage to exposed membranes, an effect that was not seen with the use of humidified gas.⁵⁸

Pain assessment^{36,59,60}

- Physical examination
- Questioning on characteristics of pain – onset, duration, location, quality, severity and intensity
- The impact of pain on the patient's functional, behavioral and psychological

status should also be assessed

Pain measurement is done by two methods Type I methods:

Those are objective methods, done by the physician as he assigns numbers about the patient condition. It includes the following:

Physiological indices

- Endocrinal (increase in serum cortisol and catecholamine).
- Cardiovascular (increase in blood pressure and heart rate)
- Respiratory (increase in respiratory rate and decrease in tidal volume) Neuro-pharmacological
- Correlation with beta endorphin (decreased in acute painful conditions)
- Thermography (hypo-emission in chronic pain) Neurological
- Nerve conduction velocity
- Evoked potentials
- Single positron emission tomography (SPET). Behavioral
Sighing, crying, shouting, trembling.

Type II methods:

It includes either

Single-dimension methods

- Category scale (verbal rating scale)
- Numerical rating scale
- Graphic rating scale Multi-dimensional methods

- Mc Gill pain Questionnaire, MPQ
- Dartmouth pain Questionnaire, DPQ
- West Haven-Yale pain Questionnaire, WHYPQ.

Measurement of pain in clinical practice depends largely on verbal dialogue between the patient and the doctor or nurse. A rating scale is mandatory in research projects and ideally when clinical data are being collected.

A number of individual differences between patients make comparisons of pain measurements more difficult. For example, the past experiences of the patients influence their present perception of pain. Also, demographic factors such as gender, age, and ethnic background influence the individual's perception of pain. Again, patients who are clinically depressed and anxious tend to report increased pain intensity.

Although pain is a subjective experience, great attention has been paid to the quantification of this experience. As pain is subjective experience, everyone has different perceptions of that experience. Differences are found in how individuals quantify pain. For example, some individuals would never say that their pain was a (10) on a scale from (0) to (10).

On the other hand, other individuals report their pain as a constant (10) despite looking calm and relaxed. Also, all numeric scales used to measure pain have floor and ceiling effects. If the patients describe their pain to be a (10), there is no way to report an increase in pain intensity.

Of the many methods of pain scoring VAS and VRS are the most commonly used in the single dimension method.

Visual analogue scale (VAS):

The visual analogue scale uses a straight line with extremities of pain intensity on either end. The line is typically 10 cm long with one end defined as “no pain” and the other end being excruciating “unbearable pain”. The line can be either vertical or horizontal. The patients are asked to place a mark on the line to describe the amount of pain that they are currently experiencing. The distance between the end labeled “no pain” and the mark placed by the patient is measured and rounded to the nearest centimeter.

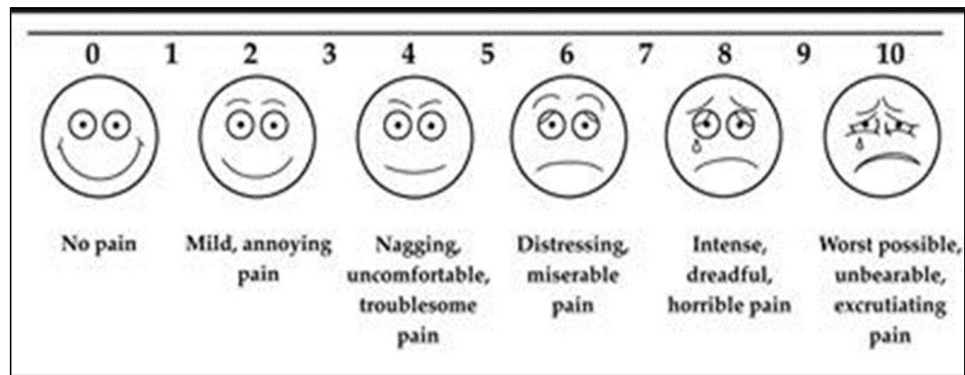


Figure 5: Visual Analog Scale

To assist in describing the intensity of pain, words can be placed along the scale (for example, mild, moderate, or severe). Such descriptors can help to orient the patient for the degree of pain; this particular variation of the VAS has been known as a graphic rating scale. Explanation to the patient is needed by the clinician when using the VAS. Occasionally, the patient may be confused about the line, perceiving it to represent time of degree of relief rather than degree of pain intensity.

MANAGEMENT OF POSTOPERATIVE PAIN ⁴⁵⁻⁵¹

Prophylactic measures:

The incidence, severity, and duration of pain and suffering during the postoperative period can be decreased by proper preoperative and postoperative surgical and psychological care. Although the accepted definition of pain emphasizes the cognitive, emotional response to tissue damage, the role of psychological techniques in the relief of acute pain has been minimized. Psychoeducational care has beneficial effects on recovery, postoperative pain and psychological distress after surgery.

Psycho-educational care was classed as health-care information (information in preparation for surgery, timing of procedures, function and roles of health-care providers, self-care actions, and pain and discomfort information); skills teaching (coughing, breathing and bed exercises, relaxation, hypnosis); and psychosocial support (identifying and alleviating concerns, reassurance, problems solving, and encouraging questions).

Optimal surgical care also helps to decrease the severity of postoperative pain. Skillful and gentle handling of tissues while carrying out the operation and observance of other surgical principles assist to minimize trauma. Proper postoperative care help to decrease the magnitude of postoperative pain which involves continuing psychological support, proper care of wounds, early ambulation, and of course good nursing care.

Active measures

Postoperative pain can be partially or completely relieved by one of the following methods:

Systemic analgesics and adjuvant

- Opioids
- Non-steroidal anti-inflammatory drugs
- NMDA antagonists
- Alpha-2 adrenergic agonists
- Miscellaneous non-opioid compounds

Regional analgesia with local anaesthetics

- Local infiltration and field block
- Continuous segmental epidural block
- Intra-pleural instillation
- Intra-peritoneal instillation
- Infiltration of the incision site

Regional analgesia with neuro-axial opioids and local anaesthetics
Regional analgesia with combined local anaesthetics and opioids

Electrical analgesia achieved with transcutaneous electrical stimulation or electro- acupuncture.

ANATOMY OF THE ABDOMINAL WALL ⁶¹⁻⁶⁴

The abdominal wall protects the internal organs from injury. It is bounded superiorly by the xiphoid process and costal margins, posteriorly by the vertebral column and inferiorly by the pelvic bones and inguinal ligament.

The abdominal wall is divided into anterolateral and posterior abdominal walls, consisting of various layers, from superficial to deep - skin, superficial fascia, muscles and their respective fascia, and peritoneum.

The superficial fascia is a connective tissue which has contents based upon its location

Above the umbilicus – a single sheet of connective tissue. It is continuous with the superficial fascia in other regions of the body.

Below the umbilicus – divided into two layers; the fatty superficial layer (Camper's fascia) and the membranous deep layer (Scarpa's fascia). The superficial vessels and nerves run between these two layers of fascia.

Muscles of the anterior abdominal wall on either side of the midline are four large muscles namely external oblique, internal oblique, the transversus abdominis and rectus abdominis and two small muscles, the cremaster and the pyramidalis.

The external oblique, the internal oblique and the transversus abdominis are large flat muscles in the anterolateral part of the abdominal wall. In the anteromedial aspect of the abdominal wall, each flat muscle forms an aponeurosis (a broad, flat tendon), which covers the vertical rectus abdominis muscle. The aponeuroses of all the flat muscles fuse together in the midline, forming the Linea alba (a

fibrous structure that extends from the xiphoid process of the sternum to the pubic symphysis).

External Oblique

The external oblique is the largest and most superficial muscle in the abdominal wall. Its fibres run infero-medially.

Attachments: Originates from ribs 5-12, and inserts into the iliac crest and pubic tubercle.

Functions: Helps in the maintenance of abdominal tone, also in increasing intra-abdominal pressure, and in the lateral flexion of the trunk against resistance.

Blood supply and Innervation: It is supplied by branches from the lower posterior intercostal, subcostal arteries and deep circumflex iliac artery. Thoraco-abdominal nerves (T7-T11) and subcostal nerve (T12).

Internal Oblique

The internal oblique lies deep to the external oblique. It is smaller and thinner in structure, with its fibres running super medially (perpendicular to the fibres of the external oblique).

Attachments: Originates from the inguinal ligament, iliac crest and thoracolumbar fascia, and inserts into cartilages of lower six ribs and Linea alba.

Functions: Bilateral contraction compresses the abdomen, while along with the external oblique of the other side helps in lateral trunk flexion. It also helps in expiration process.

Blood supply and Innervation: Thoraco-abdominal nerves (T7-T11), subcostal nerve (T12) and branches of the lumbar plexus, Iliohypogastric and Ilioinguinal nerve.

Transversus Abdominis

The transversus abdominis is the deepest of the flat muscles, with transversely running fibres. Deep to this muscle is a well-formed layer of fascia, known as the transversalis fascia.

Attachments: Originates from the inguinal ligament, costal cartilages 7-12, the iliac crest and thoracolumbar fascia. Inserts into the conjoint tendon, xiphoid process, linea alba and the pubic crest.

Functions: Compression of abdominal contents.

Innervation: Thoracoabdominal nerves (T7-T11), subcostal nerve (T12) and branches of the lumbar plexus. Muscles of the posterior abdominal wall are the psoas major, the psoas minor, the iliacus and the quadratus lumborum.

Quadratus Lumborum

It is located laterally in the posterior abdominal wall and quadrilateral in shape. The muscle is positioned superficially to the psoas major.

Attachments: It originates from the iliac crest and iliolumbar ligament. The fibres travel superomedially, inserting onto the transverse processes of L1 – L4 and the inferior border of the 12th rib.

Actions: Extension and lateral flexion of the vertebral column. It fixes the 12th rib during inspiration, so that the contraction of diaphragm is more effective.

Innervation: Anterior rami of spinal nerves T12- L4 nerves.

Psoas Major

The psoas major is located near the midline of the posterior abdominal wall, immediately lateral to the lumbar vertebrae.

Attachments: Originates from the transverse processes and vertebral bodies of T12 – L5. It then moves inferiorly and laterally, running deep to the inguinal ligament, and attaching to the lesser trochanter of the femur.

Actions: Flexion of the thigh at the hip and lateral flexion of the vertebral column.

Innervation: Anterior rami of L1 – L3 nerves.

Psoas Minor

The psoas minor muscle is only present in 60% of the population. It is located anterior to the psoas major.

Attachments: Originates from the vertebral bodies of T12 and L1 and attaches to a ridge on the superior ramus of the pubic bone, known as the pectineal line.

Actions: Flexion of the vertebral column. Innervation: Anterior rami of the L1 spinal nerve. **Iliacus**

The iliacus muscle is a fan-shaped muscle that is situated inferiorly on the posterior abdominal wall. It combines with the psoas major to form the iliopsoas – the major flexor of the thigh.

Attachments: Originates from surface of the iliac fossa and anterior inferior iliac spine. Its fibres combine with the tendon of the psoas major, inserting into the lesser trochanter of the femur.

Actions: Flexion and lateral rotation of the thigh at the hip joint. Innervation: Femoral nerve (L2 – L4).

Diaphragm

The posterior aspect of the diaphragm is considered to be part of the posterior abdomen

Fascia of the Posterior Abdominal Wall is a layer (sheet of connective tissue) that lies between the parietal peritoneum and the muscles of the posterior abdominal wall. This fascia is continuous with the transversalis fascia of the anterolateral abdominal wall.

Psoas Fascia

The psoas fascia covers the psoas major muscle. It is attached to the lumbar vertebrae medially, continuous with the thoracolumbar fascia laterally and continuous with the iliac fascia inferiorly.

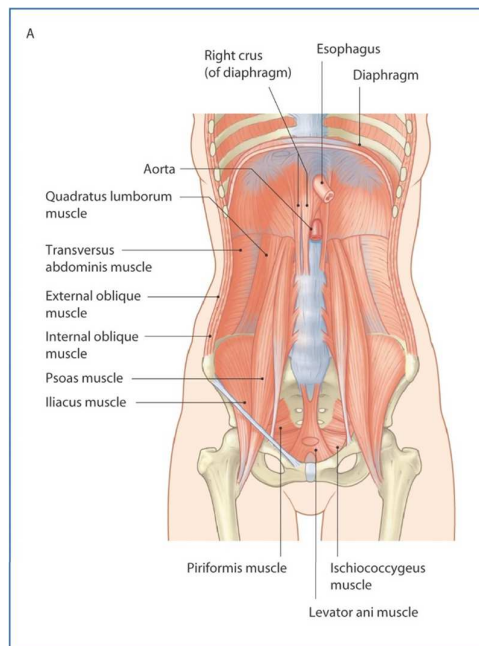


Figure 6: Muscles of the Abdomen.

Thoracolumbar fascia

The thoracolumbar fascia is made up of multiple fascial layers which is most prominent at the caudal end of the lumbar spine. It composes of three layers in the lumbar region. The posterior layer is attached medially to the spines of the lumbar vertebrae and to the supraspinous ligament.

The Aponeurosis of Latissimus Dorsi muscle forms the superficial lamina of posterior layer and its deep lamina encloses the posterior surface of the paraspinal muscles. The middle layer is attached medially to the tips of the lumbar transverse processes and goes laterally behind quadratus lumborum. Inferiorly, it has attachment to the iliac crest, and superior attachment is to the lower border of the twelfth rib. The anterior layer covers the anterior region of quadratus lumborum and is attached medially to the transverse processes of the lumbar vertebrae behind the psoas major muscle. It fuses with the transversalis fascia laterally and to the aponeurosis of transversus abdominis muscle

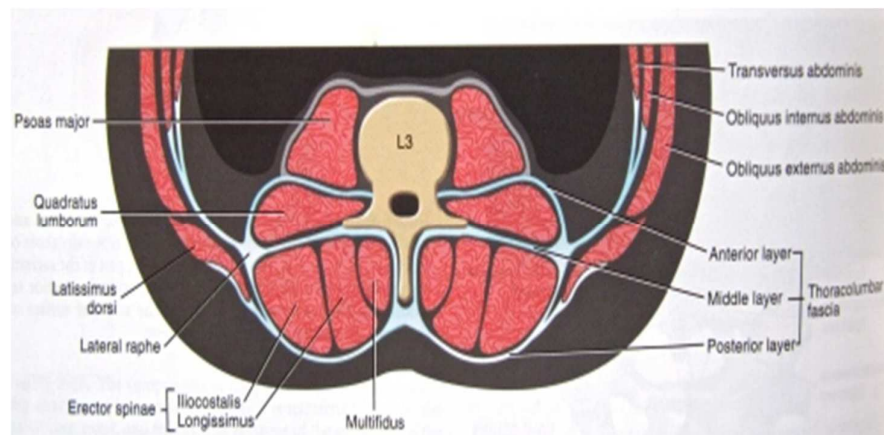


Figure 7: Thoracolumbar fascia

Inferiorly, it is also attached to the iliolumbar ligament and nearby region of iliac crest. The superior attachment of anterior layer of thoracolumbar fascia is to the inferior border of the twelfth rib and it also extends to the transverse process of the L1 vertebra thereby forming the lateral arcuate ligament of the diaphragm. The posterior and middle layers of the thoracolumbar fascia (or the lateral raphe) join at the lateral margin of the paraspinal muscles and encloses them in an osteofascial compartment. The aponeurosis of transversus abdominis muscle joins the anterior layer of thoracolumbar fascia at the lateral border of quadratus lumborum and with the lateral raphe posterior to quadratus lumborum.

NERVES AND ARTERIES OF ABDOMINAL WALL

The abdominal wall has innervations from anterior primary rami of T7- L1. Cutaneous distribution is T7 at xiphoid, T10 at umbilicus and L1 at groin. T7-11 and the subcostal nerve T12 enters the wall of abdomen between the interdigitations of diaphragm and transverses abdominis.

DERMATOMES OF ABDOMEN

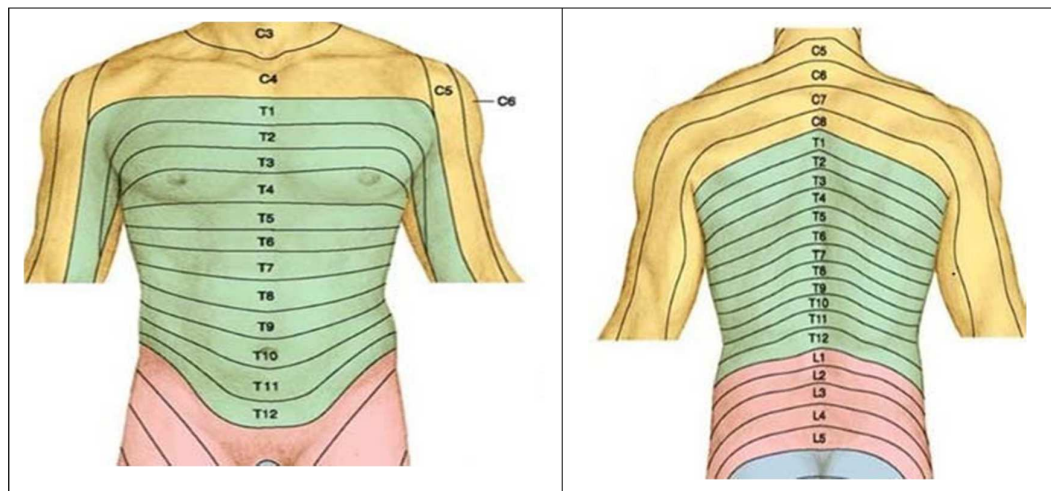


Figure 8: Dermatomes of the Abdomen

In their thoracic course, these nerves lie between the internal and innermost intercostals and in abdomen they lie in Transversus abdominis plane. L1 lumbar nerve divides anterior to quadratus lumborum muscle into iliohypogastric and ilioinguinal nerves which then penetrates transverses abdominis muscle to lie in TAP plane.

The iliohypogastric nerve pierces internal oblique muscle above and in front of Anterior Superior Iliac Spine, goes deep to external oblique and just above the inguinal canal and its ends by giving cutaneous innervations to suprapubic region. The ilioinguinal nerve also penetrates the internal oblique, runs in inguinal canal superior to spermatic cord, emerges via external ring and supplies skin of scrotum/labia majora and upper thigh.

All these nerves except ilioinguinal nerve gives off lateral cutaneous branch in midaxillary line each of which divides into anterior and posterior branches and supplies the area from lateral border of rectus muscle to in front of erector spinae muscle posteriorly.

TAP BLOCK ^{65-67,6,9}

The Transversus abdominis plane block technique was first introduced by Rafi et al in the year 2001 and McDonnell et al in the year 2004 with a landmark technique, via the lumbar triangle of Petit, the plane of injection between the muscle layers the internal oblique muscle and transverse abdominis. The thoracolumbar nerves from T6 to L1 spinal roots supply sensory nerves in the anterolateral abdominal wall, blocking these neural afferents provides analgesia to the anterolateral abdominal wall.

Using conventional technique, it has blind end points two pop offs and their success is unpredictable. With the advancement of ultrasound guided techniques there is better visualization, localization and drug deposition with improved accuracy and higher successful rate.

This block generally is indicated for lower abdominal surgeries including appendectomy, hernia repair, caesarean section, abdominal hysterectomy, prostatectomy and laparoscopic surgeries.

Conventional technique

The point of needle entry is in the triangle of Petit which is bound anteriorly by the External oblique muscle, posteriorly by the Latissimus dorsi muscle and inferiorly by the iliac crest. It is located along the midaxillary line below the lower costal margin and above the iliac crest. This technique depends on feeling of experiencing double pops as the needle pierces the external oblique and internal oblique muscles. The loss of resistance is better felt with a blunt needle. It provides anesthesia and analgesia to the anterior abdominal wall by blocking T6-L1 spinal nerves.

Sonographic anatomy of TAP block

The first step in performing TAP blocks with ultrasound guidance is to identify the muscles of the anterolateral abdominal wall. The external oblique is usually the most echogenic muscle of the anterolateral abdominal wall. The external oblique and internal oblique muscles typically extend farther posteriorly than the transversus abdominis muscle. Retroperitoneal fat (hypoechoic appearance on ultrasound scans) lies under the posterior aspect of the transversus abdominis

muscle. The layers underneath the transversus abdominis muscle are (in order) the transversalis fascia, extraperitoneal fat, and peritoneum.

Lateral TAP block

The three layers of the abdominal wall muscles are visualized with a linear transducer being placed on the midaxillary line between the subcostal margin and the iliac crest.

Anterior TAP block

A linear transducer is positioned medial to the anterior iliac spine pointing towards the umbilicus with a caudal tilt. The target is the fascial plane between the internal oblique and transversus abdominis at the level of deep circumflex artery.

Subcostal TAP block

A linear transducer is placed along the lower margin of the rib, angled towards costal margin. The rectus abdominis muscle and its posterior rectus sheath are visualized along with the transverse abdominis muscle deep to the posterior rectus sheath. The target is the fascial plane between the posterior rectus sheath and transversus abdominis muscle.

Posterior TAP block

A linear transducer is placed in the axial plane in the midaxillary line and moved posteriorly to the posterior most limit of the TAP between the internal oblique and transversus abdominis.

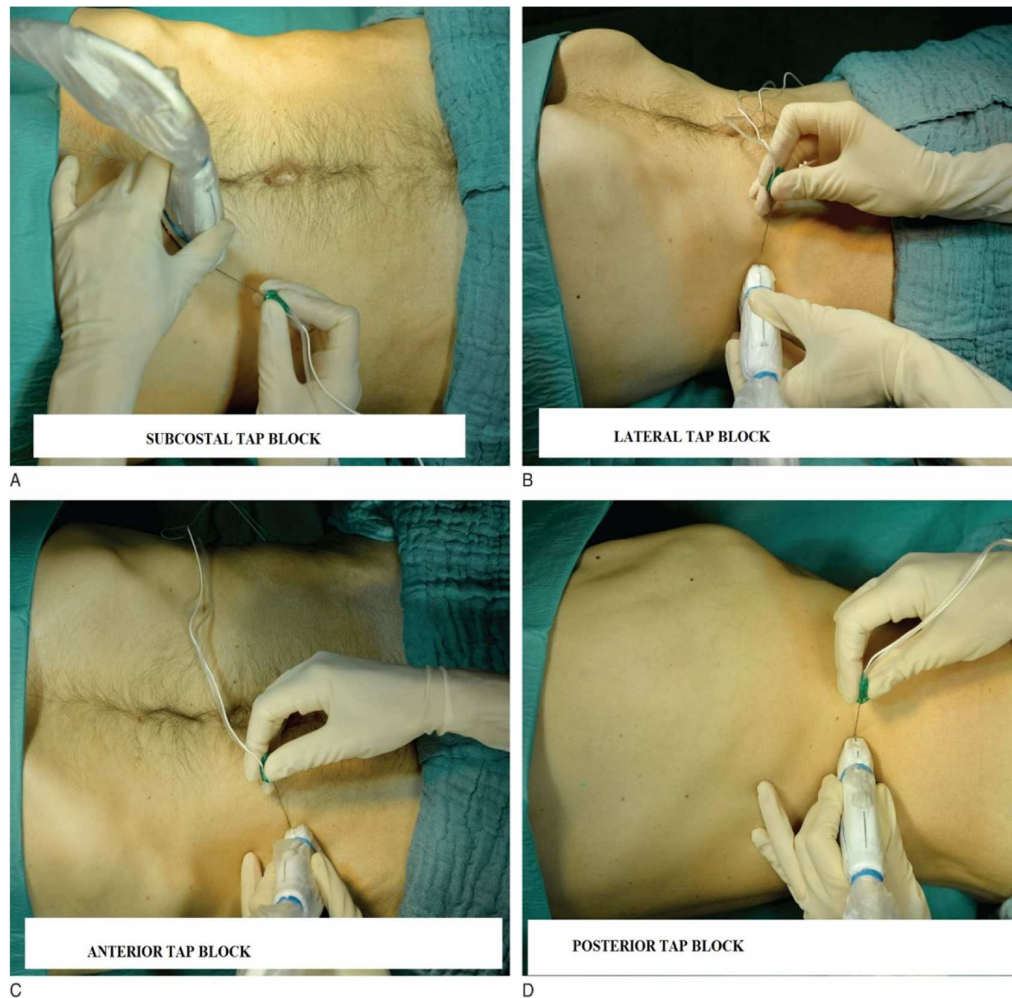


Figure 9: Various types of the TAP block

ULTRASONOGRAPHY ^{68,69}

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

Ultrasound Pulse Generation

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

Ultrasound Wavelength and Frequency

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

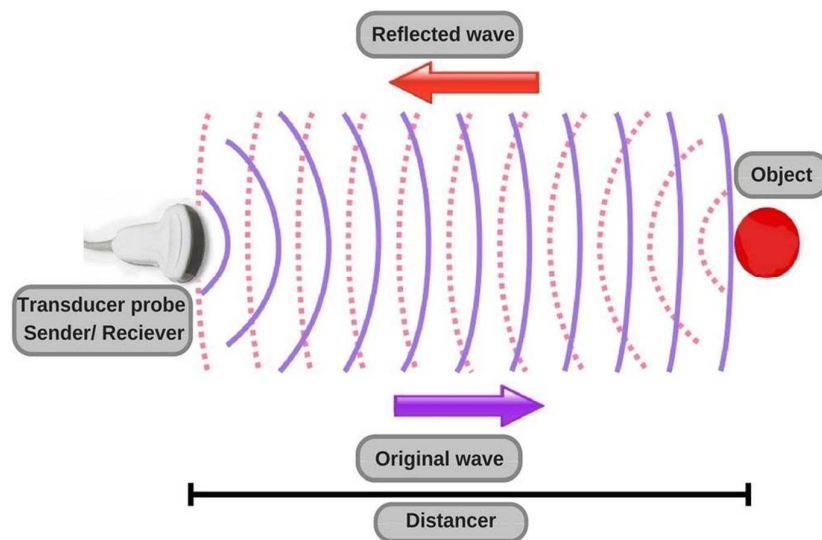


Figure 10: Principles of Ultrasonography

Ultrasound Tissue Interaction:

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

Reflection

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

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

Scattering

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

Absorption

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

Attenuation

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

Diffraction

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

Image Construction

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

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

Scanning Modes

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

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

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

Ultrasound controls

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

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

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

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

Approaches and techniques

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

Out-of-plane:

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

Advantages:

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

Disadvantages:

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

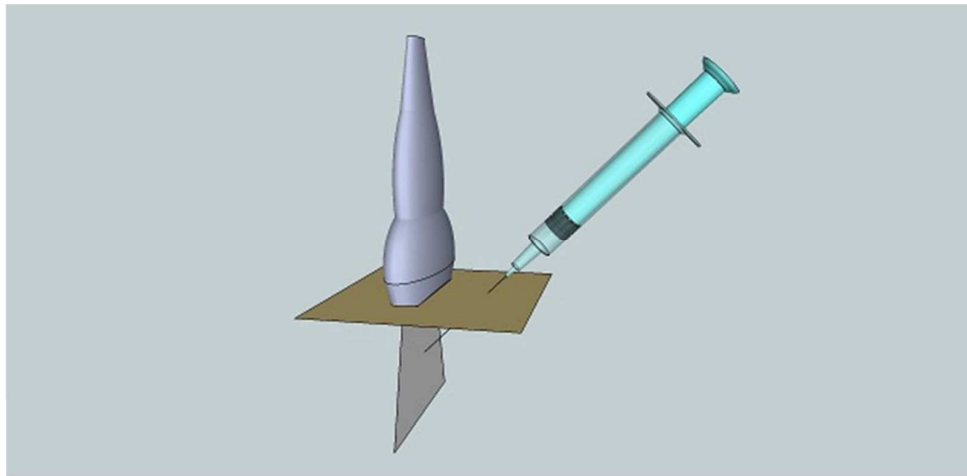


Figure 11: Out of plane technique

In-plane (IP):

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

Advantages:

Most direct visualization. Disadvantages:

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

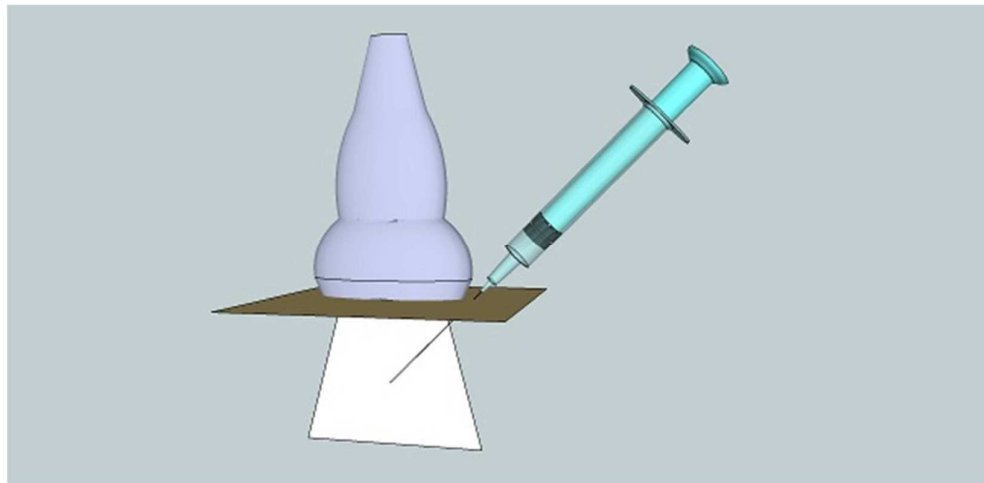


Figure 12: In-plane Technique

Ultrasound probes

Commonly used are three types

- Linear high frequency (6 to 12 MHz) probes which has high resolution and lesser penetration and is ideal for visualizing superficial structures.
- Curvilinear low frequency probes (2-5MHz) which has low resolution, higher penetration and is ideal for deeper structures like intraabdominal organs.

- Phased Array Probe also has low frequency (2MHz – 7.5MHz) gives a large depth with a small acoustic window, ideal for chest ultrasound.

Imaging

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

The nerves appear round or oval in transverse view and are hypo-echoic or they appear as honeycomb structures with septations inside them. Nerves are bordered by a hyper-echoic layer of connective tissue. Blood vessels will appear as circular

hypoechoic to anechoic structures with a well-defined hyper-echoic border which is the vessel wall. Veins are compressible with thinner walls whereas arteries have thicker walls and appear pulsatile in nature. Muscles have fibrous-lamellar texture and appear as heterogeneous or homogeneous hypoechoic structures with hyper-echoic septa in between.

Basic principles of ultrasound guided nerve blocks.

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

BUPIVACAINE ^{35,38,44}

Bupivacaine is an amino amide class of local anaesthetic drug. It was first synthesized by Ekenstam in 1957 and its clinical use was started by LJ Telivuo in 1963. Since then, it has become one of the widely used local anaesthetic agents clinically.

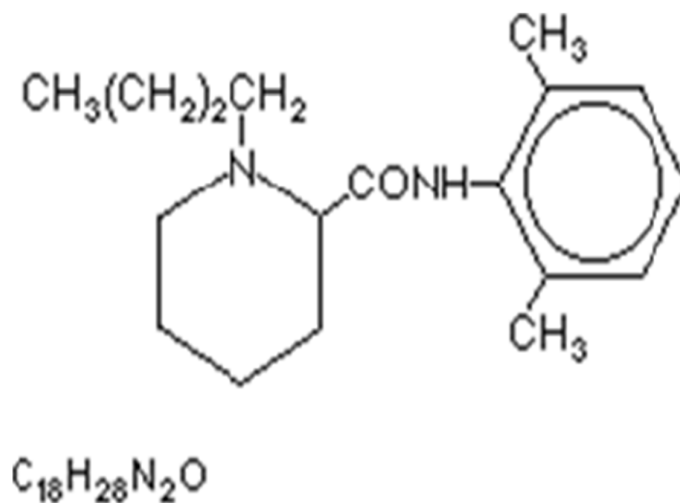


Figure 13: Chemical structure of Bupivacaine.

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

PHARMACODYNAMICS

Bupivacaine permeates the nerve's axon membranes and accumulate within the axoplasm. Binding to sites on voltage-gated Na⁺ channels prevent opening of the channels by inhibiting the conformational changes that underlie channel activation. On comparison with lignocaine, it is four times more potent but the onset of action is slower. The duration of action is considerably longer. The sensory blockade caused by bupivacaine is much more than the motor blockade.

PHARMACOKINETICS

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

The onset and duration of action depend on the dose, concentration, route of administration and vascularity of the site of administration. The volume of distribution is 54 L. The elimination half-life is 210 minutes. The Clearance is 0.32 L/min. Bupivacaine undergoes biotransformation in liver by aromatic hydroxylation, N-dealkylation, amide hydrolysis, and conjugation. The metabolites are excreted via the kidney. Less than 5% of the drug is excreted unchanged.

Dosage and preparations

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

Uses

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

Contraindications

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

Adverse effects

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

Central Nervous System Toxicity

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

Cardiovascular system effects

Part of the cardiac toxicity that occurs with high plasma concentrations of bupivacaine occurs because of the blockade of cardiac sodium channels. Accidental intravenous injection of bupivacaine causes cardiac dysrhythmias, atrioventricular block, ventricular tachycardia and ventricular fibrillation, bradycardia and asystole. Pregnancy increases the sensitivity of cardiotoxic effects of bupivacaine.

ROPIVACAINE (70)

Introduction

Ropivacaine is a newer, longer acting local anaesthetic agent which belongs to the amino amide group. It was first synthesized by Ekenstam in 1957; however it was first introduced for clinical practice only since 1996. Chemically it belongs to the same group as bupivacaine and mepivacaine (epipecoloxylidide local anaesthetic).

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

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

It is becoming increasingly popular among anaesthesiologists and has been used extensively in almost all modes of regional anaesthesia: infiltration,

peripheral nerve blocks, spinal anaesthesia, epidural anaesthesia as well as caudal epidural blocks in paediatric patients.

Chemical Structure

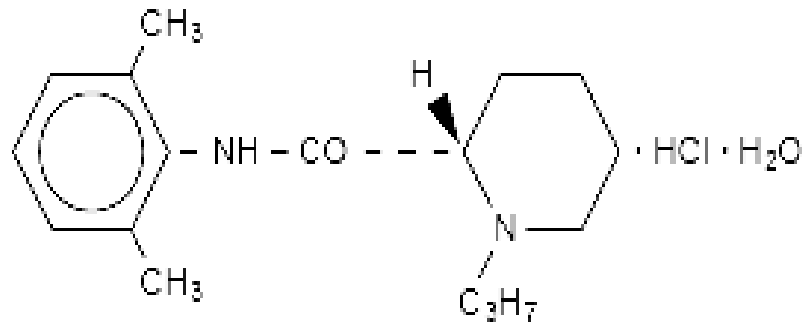


Figure 14: Chemical structure of ropivacaine

Ropivacaine is an amino amide local anaesthetic agent, chemically described as S-(-)-1-propyl -2',6'-pipercoloxylidide hydrochloride monohydrate. *The* International Union of Pure and Applied Chemistry name is (S)-N-(2,6-dimethylphenyl) -1- propylpiperidine-2- carboxamide. It's molecular formula is C₁₇H₂₆N₂O•HCl•H₂O and it has a molecular weight of 328.89.

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

However, ropivacaine has a much lesser lipid solubility as compared to bupivacaine and mepivacaine. This can be explained on the basis of presence of a propyl (3 Carbon) side chain in ropivacaine as compared to a butyl (4 Carbon) side chain in the other two local anaesthetics.

This lower lipid solubility Physical Properties of ropivacaine has a significant effect on the block characteristics of ropivacaine as discussed ahead.

Mechanism Of Action and Corelation with Structure

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

- I. Blocks generation and conductance of nerve impulses.
- II. Slows propagation of nerve impulses
- III. Reduces the rate of rise of action potential

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

The rate of blockade of motor fibres (A α and A β), however depends upon the physio chemical properties like pKa and lipid solubility of the individual drug. As ropivacaine is less lipid soluble than bupivacaine, the A α and A β blockade is slower and hence motor blockade is less potent. Studies of lumbar epidural block in humans have confirmed that equal volumes and concentrations of bupivacaine and ropivacaine produce similar degree of sensory block but the motor block produced by ropivacaine is slower in onset, lesser in intensity and shorter in duration.

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

The nerve impulse transmission is lost in the following order: The order of the loss of nerve function is

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

Pharmacokinetics

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

Distribution:

After intravascular infusion, ropivacaine has a steady state of distribution of 41 w 7 litres. It is 94% protein bound, mainly to α_1 -acid glycoprotein. In case of continuous epidural infusion of ropivacaine the plasma concentration can rise due to increased protein binding and reduced clearance. Ropivacaine can easily cross the placenta.

Metabolism and excretion:

Ropivacaine is extensively metabolized by the liver, predominantly by the cytochrome P₄₅₀1A mediated aromatic hydroxylation to produce 3 – hydroxyl ropivacaine. After a single IV dose, approximately 37% of the total dose is excreted in the urine as both free and conjugated 3- hydroxy ropivacaine. An

additional unquantified amount of 2 – hydroxyl – methyl ropivacaine has also been identified as a metabolite.

Ropivacaine metabolites are mainly excreted via kidney. After i.v. administration 86% of the dose is excreted in urine of which only 1% is in unchanged form. Following IV administration, ropivacaine has a mean w SD total plasma clearance of 387 w 107 mL/min, an unbound plasma clearance of 7.2 w 1.6 L/min and a renal clearance of 1 mL/min. The mean w SD terminal half life is 1.8 w 0.7 h and 4.2 w 1.0 h after i.v. and epidural administration respectively.

Pharmacodynamics *Central Nervous System & Cardiovascular System* :

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

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

Potency:

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

Others:

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

Adverse Effects

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

- a) Injection site pain
- b) Cardiovascular system toxicity: Vasovagal reaction, syncope, postural hypotension, non-specific ECG abnormalities which include wide QRS complexes, increased conduction time and reduced contractility.
- c) Gastrointestinal system toxicity: Faecal incontinence, tenesmus, nausea, vomiting.
- d) Central nervous system toxicity: Tremor, Horner's syndrome, dyskinesia, neuropathy, vertigo, convulsion and coma. Because of depressant effect of ropivacaine on medulla, excitatory stage of CNS might not occur.
- e) Liver and Biliary system toxicity: Jaundice
- f) Metabolic disorders: Hypomagnesemia

Advantages Over Other Local Anaesthetics

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

TOXICITY (71,72)

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

DEXMEDETOMIDINE (73)

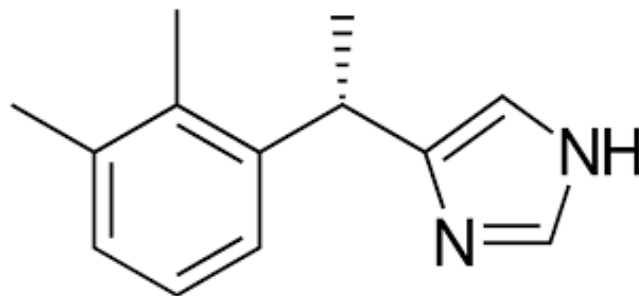


Figure 15: Chemical structure of Dexmedetomidine

Dexmedetomidine is the S-enantiomer of the veterinary sedative medetomidine. It is a highly selective α_2 -adrenoceptor agonist demonstrating an $\alpha_2:\alpha_1$ selectivity ratio of 1620:1. This makes it eight times more selective for the α_2 -adrenoceptor than clonidine.

Drug actions

Sedation and anxiolysis:

These properties are mediated via agonism of α_2 -adrenoceptors primarily in the locus coeruleus of the pons where it results in dose-dependent inhibition of norepinephrine release. It is postulated that this results in disinhibition of the ventrolateral preoptic nucleus which then releases inhibitory neurotransmitters. This pathway is part of the complex circuitry governing natural sleep, resulting in a quality of sedation with dexmedetomidine which more closely resembles normal physiological sleep than the more familiar GABA-ergic sedatives (propofol and the benzodiazepines). This sedation is characterized by preserved muscle tone and ventilation, by spontaneous and evoked movements, and by awakening by external stimuli. Once roused, patients are cooperative and can typically obey simple instructions. Once the external stimulus is discontinued, patients resume the previous level of sedation. Electroencephalogram studies have further confirmed that the sedative effects of dexmedetomidine mimic stage 2 non-rapid eye movement sleep^[74]

Analgesia:

It is likely that dexmedetomidine exerts effects at various sites in the pain pathway, but its main site of action is at the level of the spinal cord where stimulation of α_2 -receptors in the substantia gelatinosa of the dorsal horn reduces the release of nociceptive neurotransmitters such as substance P.

Effects on organ systems:

The cardiovascular effects of the drug are biphasic. At higher rates of infusion, such as during administration of a loading dose, the predominant effect is hypertension due to activation of α_2B receptors on vascular smooth muscle. This is superseded by hypotension and bradycardia as a result of the centrally mediated inhibition of sympathetic outflow. Case reports of bradycardia leading to asystole after loading dose administration of the drug in conjunction with multiple other be found in the literature^[75] Cardiovascular adverse effects associated with dexmedetomidine may be expected to be more pronounced in hypovolaemic patients, in those with diabetes mellitus or chronic hypertension, in the elderly and in those with high vagal tone. A defining feature of the sedative action of dexmedetomidine is its minimal effect on ventilation, even when given in doses 10 times the maximum recommended^[76] In addition, MRI studies have shown that the airway remains patent during dexmedetomidine sedation. Owing to actions on peripheral α_2 -adrenoceptors, dexmedetomidine also has decongestant and antisialagogue effects. It may theoretically reduce bowel motility.

Dexmedetomidine suppresses shivering, possibly due to agonism of α_2B receptors in the hypothalamus. It exerts a diuretic effect by inhibiting the action of ADH at the collecting duct. Despite its imidazole structure, dexmedetomidine has not been found to cause any clinically significant adrenal suppression.

Pharmacokinetics:

Administration is possible via multiple routes, with a bioavailability of 16% when given orally, 65% nasally, and 82% buccally. It is 94% protein bound with the unbound drug freely crossing the blood–brain barrier to exert its central effects, with a distribution half-life of 6 min. It undergoes glucuronidation, hydroxylation, and N-

methylation in the liver to inactive metabolites which are then renally excreted. Hepatic impairment therefore should prompt a dose reduction due to decreased protein binding and metabolism, while renal impairment and renal replacement therapy requires no dose adjustment. It has a terminal elimination half-life of ~2 h with clearance estimated at 39 litre/h. Its steady-state volume of distribution (118 litres) is increased in patients with low plasma albumin concentration, prolonging the terminal half-life and context-sensitive half-time in such patients.^[77]

Drug administration:

The dexmedetomidine infusion is begun at an infusion rate of 0.7 µg kg⁻¹ h⁻¹ and is then adjusted according to response within the dose range 0.2–1.4 µg kg⁻¹ h⁻¹. In contrast to its use in anaesthesia, it is recommended that no loading dose is given when used for sedation in the ICU. After dose adjustment, a new steady-state sedation level may not be reached for up to 1 h.

Perioperative use

Sedative premedication:

Its anxiolytic, sedative, sympatholytic, and antisialagogue properties, along with a lack of respiratory depression make dexmedetomidine suitable for premedication. The drug also acts as an anaesthetic-sparing agent and obtunds the pressor response to intubation. Its versatility in route of administration is an advantage in paediatric premedication where intranasal administration of 1 µg kg⁻¹ dexmedetomidine was shown to be as effective a sedative as midazolam 0.5 mg kg⁻¹ orally, with modest haemodynamic effects.^[78]

Anaesthetic and opioid-sparing agent:

Dexmedetomidine decreases anaesthetic requirements and is opioid sparing. These properties are particularly useful in certain patient populations where the respiratory- depressant properties of opioids may be particularly detrimental, such as in bariatric surgery.

Sympatholysis:

A Cochrane review in 2009 ^[79] examined the theoretical benefits of α -agonists in obtunding the perioperative stress-induced increase in sympathetic activity, and thereby reducing cardiac complications of surgery. The authors found that perioperative α_2 - agonists reduced mortality and myocardial ischaemia, with the greatest benefit seen in patients undergoing vascular surgery. There was, however, an increase in perioperative hypotension and bradycardia with drug administration. Continuous infusion of dexmedetomidine throughout the extubation period has been used for emergence smoothing. The drug also offers effective prevention and treatment of emergence phenomena.

Postoperative analgesia:

Postoperative dexmedetomidine infusions have been used to supplement other forms of analgesia in patients in whom opioid-induced respiratory depression would be potentially deleterious. A small randomized controlled trial of thoracic surgical patients found less supplemental epidural opioid was needed in the group who also received an i.v. dexmedetomidine infusion.

Neuroanaesthesia:

Dexmedetomidine is routinely used in our centre for neurosurgical procedures requiring intraoperative patient cooperation, that is, awake craniotomy for supratentorial tumour resection or deep brain stimulator implantation. It does not

suppress epileptiform activity in patients undergoing electrocorticography and so is useful in epilepsy surgery.

Dexmedetomidine administration has no effect on intracranial pressure. Although there were initial concerns that it may reduce cerebral blood flow leading to ischaemia, multiple studies have demonstrated a matched reduction in cerebral blood flow and cerebral metabolic rate.[80)

It does not affect somatosensory evoked potentials or motor- evoked potentials and so may be a useful anaesthetic-sparing agent and analgesic supplement in scoliosis surgery. Experimental studies show dexmedetomidine has neuroprotective effects in hypoxic–ischaemic and traumatic brain injury models. This neuroprotection appears to be afforded by the action of the drug on α_2A -receptors and at imidazoline receptors.

Regional anaesthesia adjuncts:

A limited number of studies have shown a prolongation of regional nerve block when dexmedetomidine was added to the local anaesthetic.

MATERIALS AND METHODS

Current study was performed on subjects of 18 years to 65 years and both sexes, ASA grade 1 and 2, posted for abdominal surgery under GA at “KLE’s Dr. Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belagavi 590010”.

Study Design: Randomized Controlled Trial.

Study Period: 1 year

Sample Size:

Sample size: Total sample size: 68

Confidence interval: 95%

Sample size formula:

Sample size formula based on mean and standard deviation is

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

where z_{α} = level of significance

z_{β} = power of the test.

For 5% level of significance $z_{\alpha} = 1.96$ and $z_{\beta} = 0.85$ for 80% power of test.

Parameters considered in calculation is the mean of 1st group (12.6) & 2nd group (9.2).

s_1 = SD of 1st group (5.13) and s_2 = SD of 2nd group (4.8).

Sample size = 68

There were 2 groups with 34 subjects in each.

Patients were allocated a group by computer-based randomization.

- **Group B** received 0.5% Bupivacaine + Dexmedetomidine
- **Group R** received 0.75% Ropivacaine + Dexmedetomidine

Inclusion Criteria:

- ASA grades I & II
- 18 years to 65 years
- Subjects enlisted for lower abdomen surgeries under GA

Exclusion Criteria:

- ASA grade III or IV
- Hypersensitivity to local anaesthesia
- Patients with contamination at the injection area
- Subjects who have coagulopathies
- Patients on systemic anticoagulant medication

Study protocol: After ethical committee clearance and written consent, 68 participants enlisted for abdominal surgery under general anaesthesia such as hernia repair, appendectomy, cholecystectomy etc. were enrolled.

- Group R was administered TAPB with 15mL 0.75% or 3mg/kg of ropivacaine + 10mcg Dexmedetomidine on each side
- Group B was administered TAPB with 15mL or 5mg/kg bupivacaine + 10 mcg dexmedetomidine on each side.

A detailed PAE was done for all patients and routine investigations were also reviewed. Peri-operatively routine vital monitors (Spo2, ECG, NIBP) were all applied. Baseline readings of vitals including HR,SBP, DBP and Sp02 were noted. Patients were cannulated.

Pre-medications were administered. Pre-oxygenation was done for 3-5 minutes. GA was induced with IV Inj.fentanyl (1 µg/kg), Propofol (2 mg/kg), and atracurium (0.5 mg/ kg). Endotracheal intubation was carried out and patient was put on mechanical ventilation. Anaesthesia was continued with O₂, N₂O, isoflurane titrated according to the MAC values. Intra- operatively routine analgesics were given.

Before extubating, transversus abdominis block was administered using strict asepsis under USG technique using the drug depending on the group to which the subject was randomly assigned.

TAPB was carried out by USG under asepsis. In between the iliac crest and the costal edge, a high frequency linear array USG probe was positioned in the axial plane across the mid-axillary line. A block needle was put in plane till its tip was situated between internal oblique and transversus abdominis ,after 3layers of the abdominal wall had been identified. Following a meticulous aspiration, study drug was injected and ultrasonography revealed a hypoechoic layer.

Care was taken to not exceed the toxic dose of the study drug.

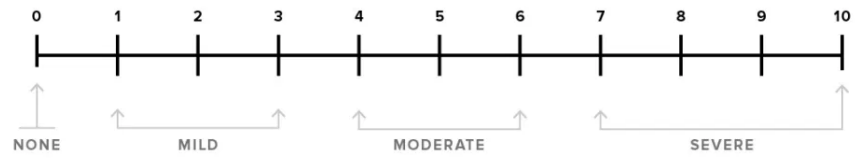
In case adequate volume of the drug could not be given, due to the risk of drug toxicity, the study drug was diluted with normal saline to reach the volume necessary.

Neuromuscular blockade was reversed using Inj.neostigmine(0.05mg/kg)

Patients were extubated after they were responding to commands, fully conscious and oriented they were shifted to the PACU.

VAS(0=no pain; 10=maximum pain) was utilized for post-operative pain.

0-10 NUMERIC PAIN RATING SCALE



When VAS > 3 post-procedurally, rescue analgesic 1gm Paracetamol was administered. Adverse effects such as decreased BP (SBP < 90mmHg), bradycardia (PR < 50bpm), PONV. If VAS > 3 post 3hrs of paracetamol, Inj. Diclofenac 75mg was administered.

VAS for pain will be assessed serially at 2,4,9, 18,24 h after the procedure

2 h	4 h	9 h	18h	24 h

STATISTICAL ANALYSIS:

METHODS:

Data was assessed by 1 “software R version 4.4.2 and Microsoft Excel”. Qualitative data was represented as frequency tables, numerical data expressed as “Mean \pm SD or Median (Min, Max)”. The “Chi-square test” was used to examine the link between qualitative data. The normality of data was assessed using the “Shapiro-Wilk test and QQ plot”. If the data followed a normal distribution, parametric tests were applied; otherwise, non-parametric tests were used. A “two-sample t-test” was employed to analyse mean age across groups. The “Mann-Whitney U test” was used to differentiate the distribution of variables between divisions. The “Wilcoxon test” was applied to differentiate variables between two duration points, and the Friedman test was used to analyze changes in VAS scores over time. A p-value of ≤ 0.05 was considered statistically significant.

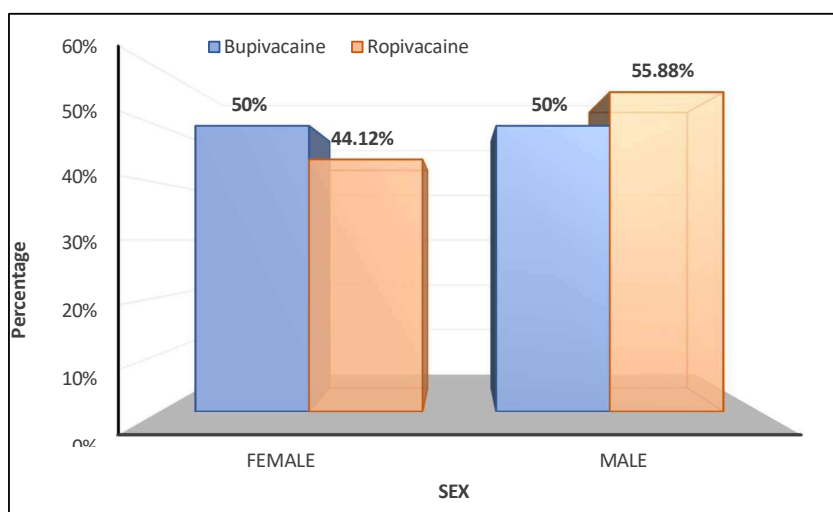
RESULTS

Data includes measurements of 68 subjects, evenly distributed in 2 divisions: 34 in B-group and 34 in R-group.

Table 1: Comparison of demographical variables over groups.

		Bupivacaine	Ropivacaine	Total	p-value
Sex	Female	17 (50%)	15 (44.12%)	32 (47.06%)	0.6270 ^C
	Male	17 (50%)	19 (55.88%)	36 (52.94%)	

Regarding sex distribution, the Bupivacaine group had an equal proportion of males and females (50% each), whereas the Ropivacaine group had 44.12% females and 55.88% males. There was no notable variation in gender. (p-value = 0.6270), indicating that gender proportions were similar between the two groups.



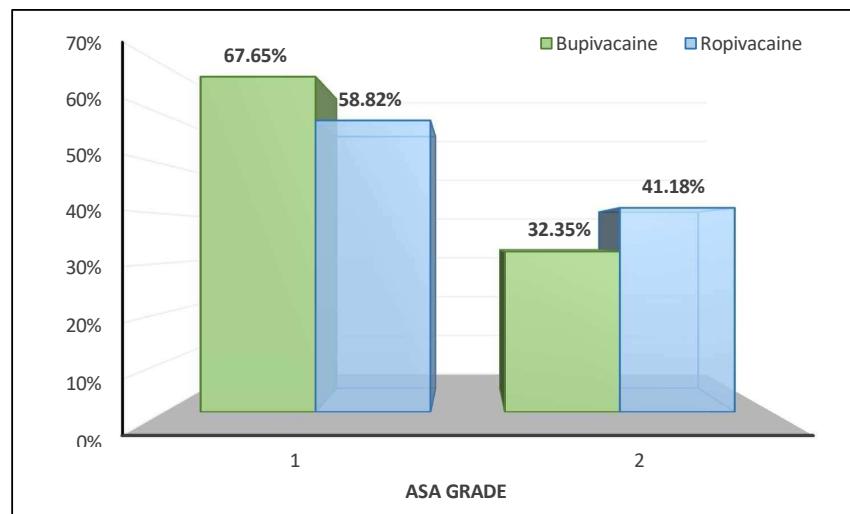
Graph 1: Gender distribution

Table 2: ASA comparison.

ASA grade	Bupivacaine	Ropivacaine	Total	p-value
1	23 (67.65%)	20 (58.82%)	43 (63.24%)	0.4505 ^C
2	11 (32.35%)	14 (41.18%)	25 (36.76%)	

Abbreviation: C – Chi square test.

In the Bupivacaine group, 67.65% of subjects were ASA grade 1, while 32.35% were as ASA grade 2. In the Ropivacaine group, 58.82% of patients belonged to ASA grade 1, and 41.18% to ASA grade 2. The ASA grade between the 2 groups was comparable (p-value = 0.4505)



Graph 2: ASA-distribution

Table 3: Comparison of surgery

Surgery	Bupivacaine	Ropivacaine	Total	p-value
Diagnostic Lap and Proceed	2 (5.88%)	3 (8.82%)	5 (7.35%)	0.8561 ^{MC}
Gastrojejunostomy	0	1 (2.94%)	1 (1.47%)	

Lap Appendicectomy	9 (26.47%)	6 (17.65%)	15 (22.06%)
Lap Cholecystectomy	13 (38.24%)	13 (38.24%)	26 (38.24%)
Lap Cholecystectomy + Hernia Repair	1 (2.94%)	2 (5.88%)	3 (4.41%)
Lap Hernia Repair	6 (17.65%)	8 (23.53%)	14 (20.59%)
Laparoscopy & proceed	1 (2.94%)	0	1 (1.47%)
Open Appendicectomy	1 (2.94%)	0	1 (1.47%)
TAH+BSO+ Inguinal Lymph Node Dissection	0	1 (2.94%)	1 (1.47%)
TLH	1 (2.94%)	0	1 (1.47%)

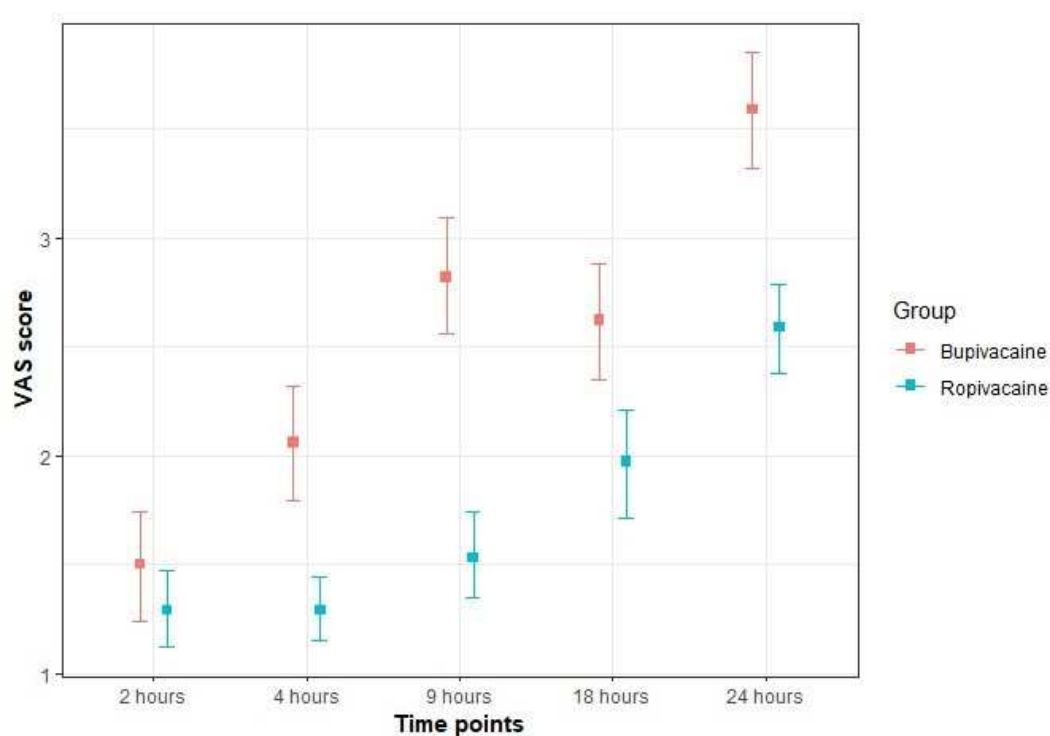
The most common procedure in both groups was laparoscopic cholecystectomy, performed in 38.24% of patients from each group. Laparoscopic appendicectomy was the second most common, accounting for 26.47% in B-group and 17.65% in R-group. From “Chi- square test”, it was noted that there wasn’t a statistically significant difference in the distribution of surgical procedures between the two groups (p-value = 0.8561).

Table 4: Comparison of VAS score

Duration	Bupivacaine	Ropivacaine	Total	p-value
2 hours	1.5 ± 0.75 2 (0, 3)	1.29 ± 0.52 1 (0, 2)	1.4 ± 0.65 1 (0, 3)	0.1033 ^{MW}
4 hours	2.06 ± 0.78 2 (1, 3)	1.29 ± 0.46 1 (1, 2)	1.68 ± 0.74 2 (1, 3)	< 0.001 ^{MW*}
9 hours	2.82 ± 0.83 3 (1, 4)	1.53 ± 0.56 1.5 (1, 3)	2.18 ± 0.96 2 (1, 4)	< 0.001 ^{MW*}
18 hours	2.62 ± 0.82 3 (1, 4)	1.97 ± 0.76 2 (1, 3)	2.29 ± 0.85 2 (1, 4)	0.0020 ^{MW*}
24 hours	3.59 ± 0.78 3 (2, 5)	2.59 ± 0.61 3 (2, 4)	3.09 ± 0.86 3 (2, 5)	< 0.001 ^{MW*}
p-value	< 0.001 ^{F*}	< 0.001 ^{F*}	-	-

2 hours, pain was greater in the B-group, but there wasn't a quantifiable variation (p-value = 0.1033). At 4 hours, pain was quantifiably more in the B-group in comparison to R-group (p-value < 0.001). This trend continued at 9h (p-value < 0.001), 18h (p-value = 0.0020), 24h (p-value < 0.001), the Bupivacaine group consistently reporting higher pain scores.

The "Friedman test" revealed a quantifiable change in pain levels over time within both groups (p-value < 0.001). Overall, the Ropivacaine group experienced lower pain scores at all time points, indicating better pain relief.

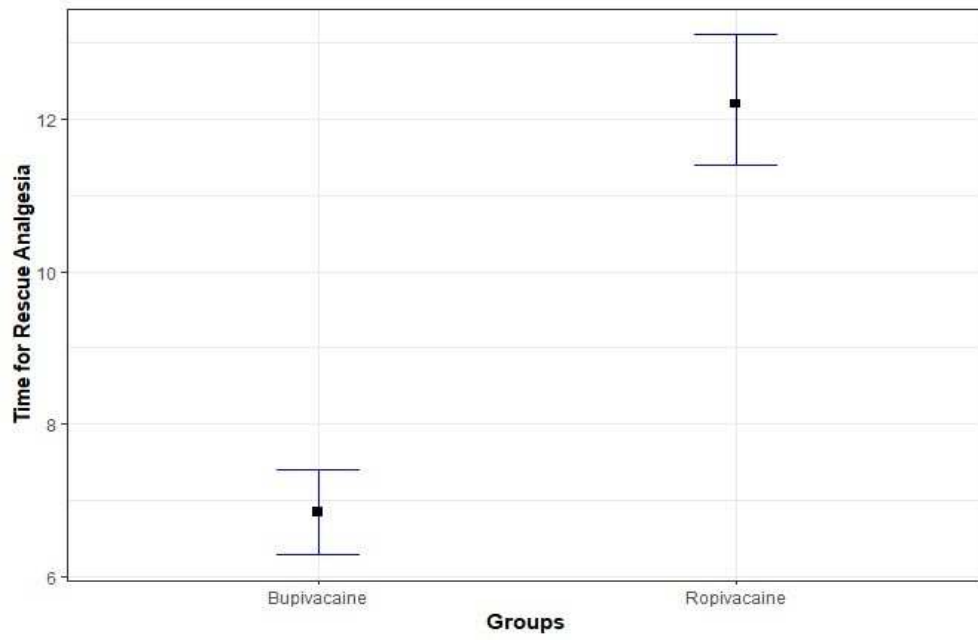


Graph 3: VAS score along duration

Table 5: Difference in time for rescue analgesia between groups.

Variable	Bupivacaine	Ropivacaine	Total	p-value
Time for rescue	6.85 ± 1.69	12.24 ± 2.7	9.54 ± 3.51	< 0.001 ^{MW*}
analgesia	6 (4, 10)	12 (9, 18)	9 (4, 18)	

The mean time for rescue analgesia was notably shorter in Bupivacaine group (6.85 ± 1.69 hours) compared to the Ropivacaine group (12.24 ± 2.7 hours). The median time was 6 hours (range: 4–10) for Bupivacaine and 12 hours (range: 9–18) for Ropivacaine. The statistical significance of the difference between the two groups was established (p-value < 0.001), showing that subjects of R-group needed rescue analgesia much later compared to Bupivacaine group, suggesting better and longer-lasting pain relief with Ropivacaine.

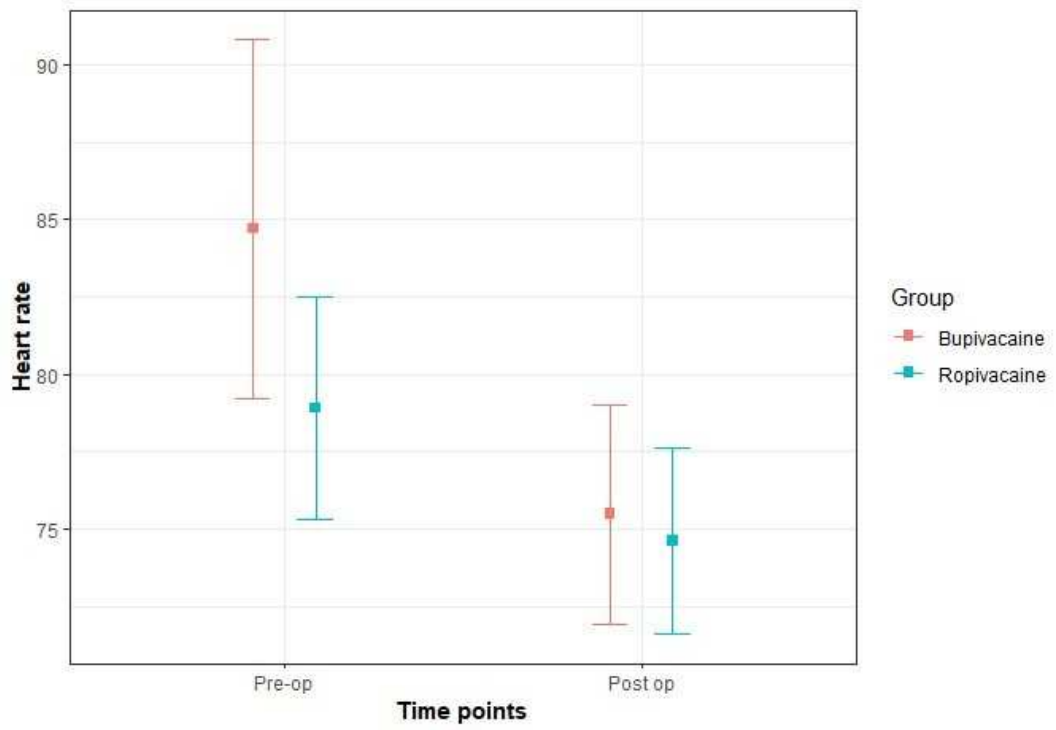


Graph 4: Mean plot of time for rescue analgesia.

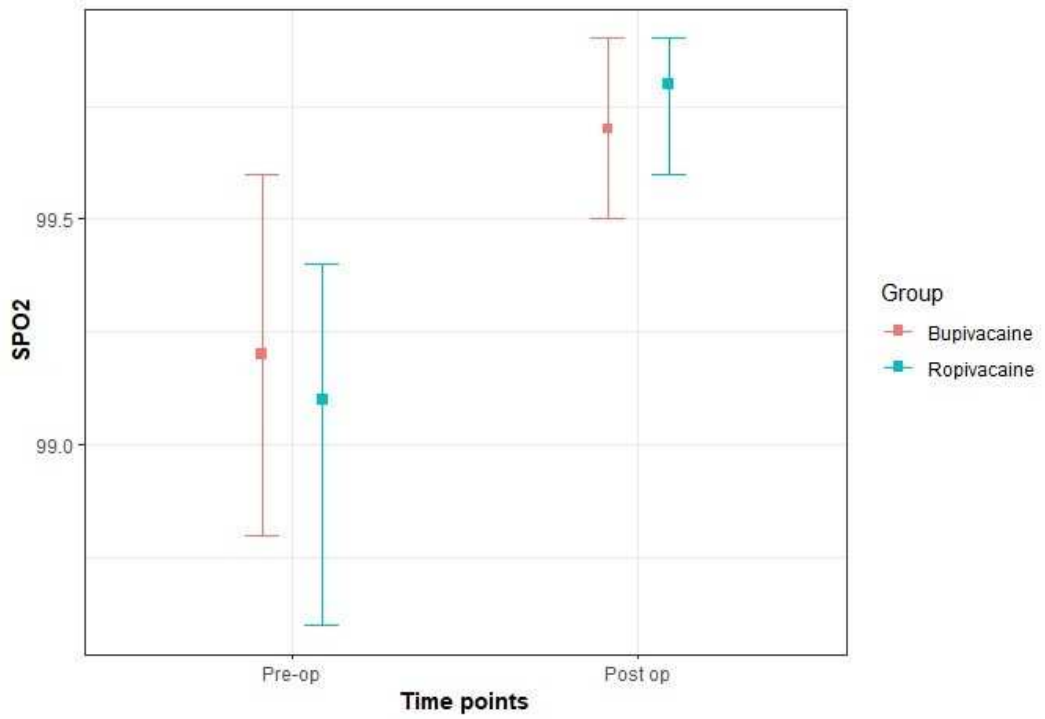
Graph 6: Vitals over time and groups.

Variables	Duration	Bupivacaine	Ropivacaine	Total	p-value
Heart rate	Pre-op	84.74 ± 17.33 82 (54, 126)	78.85 ± 10.9 77.5 (54, 108)	81.79 ± 14.67 78 (54, 126)	0.2058 ^{MW}
	Post op	75.5 ± 10.98 76.5 (58, 100)	74.59 ± 9.17 77 (58, 98)	75.04 ± 10.05 77 (58, 100)	0.8439 ^{MW}
p-value		< 0.001^{W*}	0.0259^{W*}	-	-
SPO2	Pre-op	99.21 ± 1.23 100 (96, 100)	99.06 ± 1.18 100 (96, 100)	99.13 ± 1.2 100 (96, 100)	0.5071 ^{MW}
	Post op	99.74 ± 0.67 100 (97, 100)	99.79 ± 0.41 100 (99, 100)	99.76 ± 0.55 100 (97, 100)	0.8575 ^{MW}
p-value		0.0378^{W*}	0.0013^{W*}	-	-
SBP	Pre-op	126.74 ± 17.28 128 (90, 160)	133 ± 15.99 132.5 (100, 180)	129.87 ± 16.82 130 (90, 180)	0.2535 ^{MW}
	Post op	119.94 ± 9.84 120 (97, 140)	122.65 ± 10.89 122.5 (100, 142)	121.29 ± 10.39 121 (97, 142)	0.3764 ^{MW}
p-value		0.0051^{W*}	< 0.001^{W*}	-	-
DBP	Pre-op	73.62 ± 10.3 73 (54, 100)	75.85 ± 10.41 74 (62, 110)	74.74 ± 10.34 74 (54, 110)	0.5305 ^{MW}
	Post op	71.5 ± 7.15 70 (60, 90)	67.12 ± 8.63 68 (45, 82)	69.31 ± 8.17 70 (45, 90)	0.0685 ^{MW}
p-value		0.3130 ^W	< 0.001^{W*}	-	-

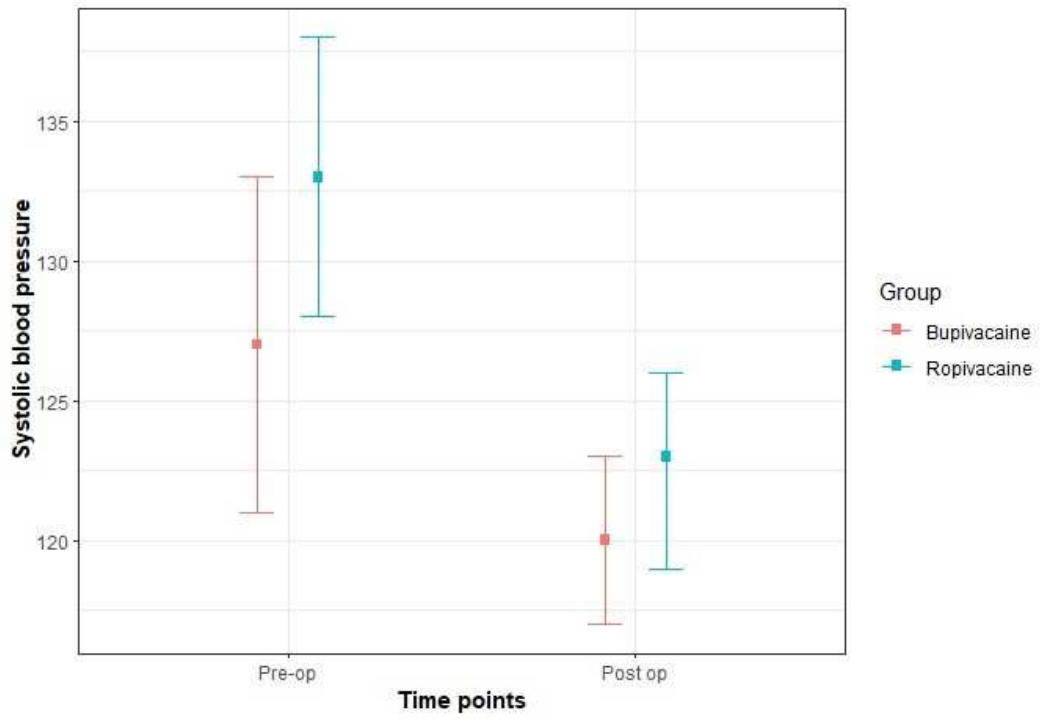
HR wasn't notably different preoperatively (p-value = 0.2058) or postoperatively (p-value = 0.8439), but both groups had significant changes over time (p-value < 0.001 for Bupivacaine, p-value = 0.0259 for Ropivacaine). SPO₂ levels were similar preoperatively (p-value = 0.5071) & postoperatively (p-value = 0.8575), but the 2 groups showed significant improvement over time (p-value = 0.0378 for Bupivacaine, p-value = 0.0013 for Ropivacaine). SBP did not differ significantly between groups at any time point (p-value = 0.2535 pre-op, p-value = 0.3764 post-op), though both groups had a significant reduction over time (p-value = 0.0051 for Bupivacaine, p-value < 0.001 for Ropivacaine). DBP didn't show any notable difference (p-value = 0.5305 pre-op, p-value = 0.0685 post-op), but only the Ropivacaine group showed a significant decrease over time (p-value < 0.001), while the B-group did not (p-value = 0.3130). Overall, both groups exhibited changes in vitals over time, but there was no notable deviation at specific durations.



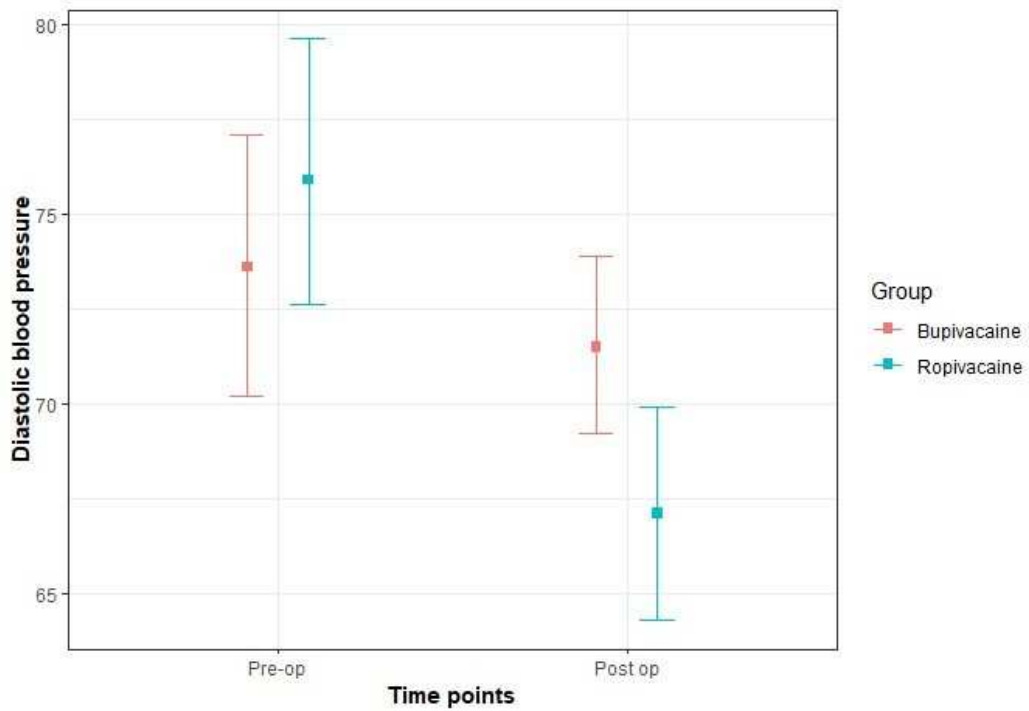
Graph 5: Mean plot of heart rate



Graph 6: Mean plot of SPO2



Graph 7: Mean plot of SBP



Graph 8: Mean plot of DBP

DISCUSSION

Acute discomfort that is thought to begin with surgery and most likely end with tissue healing is known as postoperative pain. The considerable agony brought on by pain throughout the post-operative period persists despite the advanced technologies.⁽¹⁴⁾

Significant advantages of laparoscopic procedures include smaller incisions, less trauma and less discomfort following surgery, quicker recovery times, and a decreased risk of wound infections. All of these elements help to lower perioperative morbidity and shorten inpatient stays. In order to enhance patient outcomes, laparoscopic techniques are increasingly being used for major surgeries that formerly required a lengthy recovery period after surgery, such as radical cystectomy or anterior rectum resection.

However, there are hazards connected with laparoscopic surgery, either because of the physiological changes involved in creating a pneumoperitoneum or because of the dangers associated with various laparoscopic methods. Therefore, anaesthetic techniques for laparoscopic surgery need to be enhanced to take these differences from open surgery into consideration.⁽¹⁵⁾

Chronic pain syndromes, and post-operative myocardial ischemia are among the consequences that might result from acute post-operative events, including pain.⁽⁸⁾

After laparoscopic abdominal surgery, TAPB is a well sought method for postprocedural pain relief. In addition to being safe, it lessens or completely alleviates the need for analgesics. Furthermore, a number of doctors are working to increase the accuracy of LA absorption using ultrasound. Thus, the analgesic effectiveness of laparotomy and laparoscopic surgeries has been established by this innovative approach.⁽⁹⁾

Numerous local anaesthetic medications have been used to provide adequate analgesia following surgery. ⁽¹⁰⁾⁽¹⁹⁾

A novel long-acting local anaesthetic that contains amino acids is ropivacaine. It has fewer side effects, a longer half-life, and a stronger anaesthetic than bupivacaine. It is significantly more drug-clearing, has a smaller volume of distribution, is less lipid soluble, and has a shorter elimination half-life than bupivacaine. ⁽¹¹⁾

TAPB is non dermatomal, therefore a large volume of anaesthetics is required to cover multiple spinal nerves. ⁽²⁸⁾

Systemic dexmedetomidine (DEX) produces sedative, analgesic, sympatholytic, and anaesthetic-sparing effects. ⁽²⁰⁾

As a local anaesthetic adjuvant, DEX has recently drawn more attention due to its potential to extend the duration of blockade. ^(11,22,23)

Perineural DEX can prolong the durations of blockade and pain relief when utilized in brachial plexus block, per some meta-analyses. ^(23-25,12)

Alpha-2 adrenergic receptor affinity is one of the highly specific routes by which DEX can elicit analgesia in the peripheral and spinal regions. ⁽²⁷⁾

DEX effects presynaptic neuronal receptors and decreases the release of norepinephrine at afferent nociceptors in the peripheries. ⁽²⁹⁾ . It also plays an inhibitory role in delayed K⁺ rectifier current and Na⁺ current which leads to decrease in the neuronal activity. ⁽³⁰⁾

This one-year randomized control trial was conducted on 68 ASA 1&2 participants aged between 18-65 years scheduled for abdominal surgeries under GA of both the genders. They were separated into 2 groups based on a computer-generated randomized table. Before extubation, R-group subjects were administered

TAP block with 15mL 0.75% Ropivacaine + 10mcg Dexmedetomidine bilaterally and group B subjects were administered TAP block with 15ml 0.5%Bupivacaine + 10 mcg Dexmedetomidine bilaterally.

Pain following surgery was assessed using VAS scores at 2 h,4 h, 9 h, 18 h and 24 h. If VAS score > 3 rescue analgesia with 1gm of Paracetamol was given.

The sex distribution wasn't notable (p-value = 0.6270), indicating that gender proportions were similar between the two groups.

In the Bupivacaine group, 67.65% of subjects were ASA grade 1, while 32.35% were ASA grade 2. In the Ropivacaine group, 58.82% of patients belonged to ASA grade 1, and 41.18% to ASA grade 2. The ASA grade distribution between the two groups was not notable (p-value = 0.4505), showing ASA grade proportions were comparable across the groups.

The most common procedure in both groups was laparoscopic cholecystectomy, performed in 38.24% of patients from each group. Laparoscopic appendectomy was the second most common, accounting for 26.47% in B-group and 17.65% in R-group. Laparoscopic hernia repair was also frequently performed, with 17.65% in B-group and 23.53% in R-group. Other surgical procedures were less common and distributed between the two groups in varying proportions. "Chi- square test", was used to conclude that there is no notable variation of surgical procedures (p-value = 0.8561).

Heart rate showed no statistical variation preoperatively (p-value = 0.2058) or postoperatively (p-value = 0.8439), but both groups had significant changes over time (p- value < 0.001 for Bupivacaine, p-value = 0.0259 for Ropivacaine). SPO₂ levels were similar preoperatively (p-value = 0.5071) and postoperatively (p-value = 0.8575), but the 2 groups showed significant improvement over time (p-value =

0.0378 for Bupivacaine, p-value = 0.0013 for Ropivacaine). SBP did not differ significantly between groups at any time point (p-value = 0.2535 pre-op, p-value = 0.3764 post-op), though both groups had a significant reduction over time (p-value = 0.0051 for Bupivacaine, p-value < 0.001 for Ropivacaine). DBP also showed no statistical variation (p-value = 0.5305 pre-op, p-value = 0.0685 post-op), but only the Ropivacaine group demonstrated a notable reduction over time (p-value < 0.001), while Bupivacaine group did not (p-value = 0.3130). Overall, both groups exhibited changes in vitals over time, but there was no statistical variation at specific time points.

At 2 hours, pain was slightly more in group-B, but there wasn't a notable variation (p-value = 0.1033). At 4 hours, pain was notably increased in group-B compared to the R-group (p-value < 0.001). This trend continued at 9 h (p-value < 0.001), 18 h (p-value = 0.0020), and 24 h (p-value < 0.001), with B-group consistently reporting higher pain scores.

“Friedman test” showed a notable change in pain levels within both groups (p-value < 0.001). Overall, the R-group experienced lower pain scores at all time points, indicating better pain relief.

Mean duration for analgesic request was notably shorter in B-group (6.85 ± 1.69 hours) compared to the Ropivacaine group (12.24 ± 2.7 hours). The median time was 6 hours (range: 4–10) for Bupivacaine and 12 hours (range: 9–18) for Ropivacaine. The variation between both the divisions was significant (p-value < 0.001), demonstrating that patients in the R-group required rescue analgesia much later in the B-group, suggesting better and longer-lasting pain relief with Ropivacaine.

The above findings co-relate with the study conducted by “Neha S, Mehta N, Sharma S “⁽⁵⁾ titled “Comparison of ropivacaine and bupivacaine in ultrasound-guided transversus abdominis plane block for postoperative analgesia in patients undergoing elective lower abdominal surgeries”

According to the study's findings, ropivacaine offered better postoperative analgesia and time. Compared to bupivacaine, which produced analgesia for an average of 6 hours, ropivacaine produced a noticeably longer period of pain relief (mean of 8.5 hours). Furthermore, ropivacaine-treated patients needed less opioids; on average, they consumed 3.5 mg of morphine compared to 6.5 mg in the bupivacaine-treated group. Additionally, the ropivacaine group experienced superior persistent pain relief, as seen by a longer duration before the first dosage of rescue analgesic. Both medications were well tolerated, and there were not much variations in the two groups' adverse effects. According to the study, ropivacaine might be a better choice for postoperative analgesia following these kinds of procedures.

In another study by Sarvesh et al. ⁽³¹⁾ (2018) titled "Addition of Dexmedetomidine to Ropivacaine in Subcostal Transversus Abdominis Plane Block Potentiates Postoperative Analgesia among Laparoscopic Cholecystectomy Patients" examined the effects of ropivacaine and dexmedetomidine for postprocedural pain relief in TAPB following laparoscopic surgery. In comparison to patients who got ropivacaine alone, the dexmedetomidine and ropivacaine group had appreciably decreased VAS levels at 2, 6, 12,24 h after the procedure. Furthermore, ropivacaine + dexmedetomidine group experienced lengthier pain relief, with a 12-h interval to the initial request for pain relief, compared to the ropivacaine group's 6-hour duration. Dexmedetomidine and ropivacaine together appear to be an excellent combination for

postoperative analgesia, as no notable complications were noted in either division. This also supports the results of our study.

From the above discussion we conclude that the mean duration of postoperative analgesia of patients in group-R was around 10-12 hours (12.24 ± 2.7 hours) whereas in group B it was around 6-8 hours (6.85 ± 1.69 hours).

We did not find any side effects such as block failure, hypotension, bradycardia, LA toxicity, arrhythmias or any other adverse complications.

Limitations of our study were that a fixed amount of drug was injected rather than per kg body weight which could have given a clear idea of the drug required for truncal blocks.

Future scope includes further studies can be done by placing catheters at injection site to deliver continuous infusions for providing a longer time of postoperative analgesia.

CONCLUSION

USG guided TAPB using 0.75% Ropivacaine + Dexmedetomidine leads to a more longer duration of postoperative analgesia compared to 0.5% Bupivacaine + Dexmedetomidine In patients undergoing abdominal surgeries under general anesthesia. The mean duration of postoperative analgesia in Group R was around 10-12 hours whereas in group B it was around 6-8 hours.

SUMMARY

This randomized control study was conducted in KLES Prabhakar Kore Charitable Hospital and MRC, Belagavi in the anaesthesia department for a period of one year following ethical committee clearance. It was done on 68 patients belonging to ASA I and II within age group of 18-65 years of both the genders undergoing elective abdominal surgeries under general anesthesia after the written informed consent being given. A detailed PAE was done prior to the surgery and the patients were allocated to either Group R or Group B according to a computer generated randomization table. Patients were given general anaesthesia. A detailed PAE was done and patients were allocated into group R or B according to a computer generated randomization table. Patients were given general anaesthesia and surgery was performed. At the end of the surgery before extubation, patients in group R received TAPB with 0.75% ropivacaine + Dexmedetomidine and patients in group B received TAPB with 0.5% bupivacaine + dexmedetomidine under ultrasound guidance following which routine extubation was done.

Postoperatively pain was assessed at various intervals (2h,4h,9h,18h,24h) with VAS scores. Paracetamol 1gm was given as a rescue analgesic was >3. Failed blocks, LA toxicity and side effects were also noted. The response of pain postoperatively in HR, SBP, DBP, SpO₂ were also documented.

On analysis the mean VAS scores were comparable between both the groups at various intervals postoperatively. The pain was significantly higher in the bupivacaine group at 4 hours,9 hours, 18 hours and 24 hours. The bupivacaine group consistently reported higher pain scores.

The mean time for rescue analgesia was significantly shorter in the Bupivacaine group (6.85

± 1.69 hours) compared to the Ropivacaine group (12.24 ± 2.7 hours).

In this study, based on our results we conclude that 0.75% Ropivacaine+ Dexmedetomidine provides a more effective and longer duration of postoperative analgesia (around 10-12 hours) compared to 0.5% Bupivacaine + Dexmedetomidine (around 6-8hours) in Ultrasound Guided TAPB in patients undergoing elective abdominal surgeries under general anaesthesia.

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ANNEXURES I
INFORMED CONSENT FORM
KAHERs JNMCBELAGAVI

“THE EFFECTIVENESS OF 0.5% ISOBARIC BUPIVACAINE + DEXMEDETOMIDINE Vs 0.75% ROPIVACAINE + DEXMEDETOMIDINE FOR POSTOPERATIVE ANALGESIA USING ULTRASOUND GUIDED TRANSVERSUS ABDOMINIS PLANE BLOCK FOR ABDOMINAL SURGERIES UNDER GENERAL ANAESTHESIA: A COMPARITIVE STUDY”

Name of Student/Principal Investigator: DR _____

Name of Guide/Co Investigators: DR _____

Objective: Assessment of postoperative analgesia after TAP block in patients undergoing abdominal surgeries using 0.75% Ropivacaine + Dexmedetomidine and 0.5% Bupivacaine + Dexmedetomidine.

Introduction:

I am Dr _____, Resident, JNMC, Belagavi. I am conducting a randomized control study.

The study involves the comparison between 0.75 % ropivacaine + dexmedetomidine and 0.5% bupivacaine + dexmedetomidine for the assessment of postoperative analgesia following administration of TAP block in patients undergoing abdominal surgery under General anaesthesia

Explanation of procedure:

If you agree to enroll in my study, I will ask you present, past and family history. Then you will be clinically examined in detail. You will be allotted into one of the two groups randomly using computer generated software.

Group B will receive TAP block with 0.5% bupivacaine + dexmedetomidine

Group R will receive TAP block with 0.75% ropivacaine + dexmedetomidine

Withdrawal from participation in the study:

Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

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

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

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication. **Financial incentives:** You will not receive any payment for participating in this study.

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

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

Questions: In case of any questions with regard to this study, you are free to contact:

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

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

CONSENT STATEMENT

I am making a voluntary decision to participate in the study **“THE EFFECTIVENES OF 0.5% ISOBARIC BUPIVACAINE + DEXMEDETOMIDINE Vs 0.75% ROPIVACAINE + DEXMEDETOMIDINE FOR POSTOPERATIVE ANALGESIA USING ULTRASOUND GUIDED TRANSVERSUS ABDOMINIS PLANE BLOCK FOR ABDOMINAL SURGERIES UNDER GENERAL ANAESTHESIA: A RANDOMISED CONTROL STUDY”**. My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:Name of the witness:

Signature or left thumb impression of the witness:Name of the investigator:

Signature of the investigator:

General physical examination:

Height(cm):

Weight(kgs):

BMI:

Pallor:

Icterus:

Cyanosis:

Clubbing:

Temperature:

BP:

PR:

RR:

SPO2:

SYSTEMIC EXAMINATION:

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

AIRWAY ASSESSMENT:

Teeth:

Jaw movements:

INVESTIGATIONS:

Hb(gm/dl):

TLC:

Platelet count:

Serum creatinine:

FBS:

Chest x-ray:

ECG:

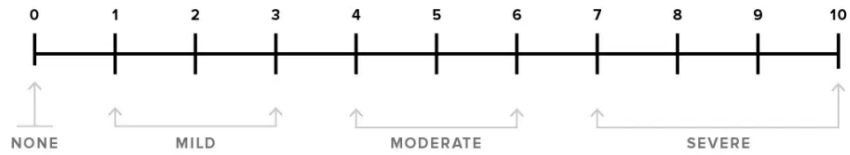
PREOPERATIVE PHYSICAL STATUS:

ASA GRADE I II III IV V

DIAGNOSIS:

PROPOSED SURGERY:

0-10 NUMERIC PAIN RATING SCALE



2 HOURS	4 HOURS	9 HOURS	18 HOURS	24 HOURS

INVESTIGATOR	
WITNESS	
ANAESTHESIOLOGIST	

ANNEXURE III – PHOTOGRAPHS



Photograph 1- 0.5% Bupivacaine



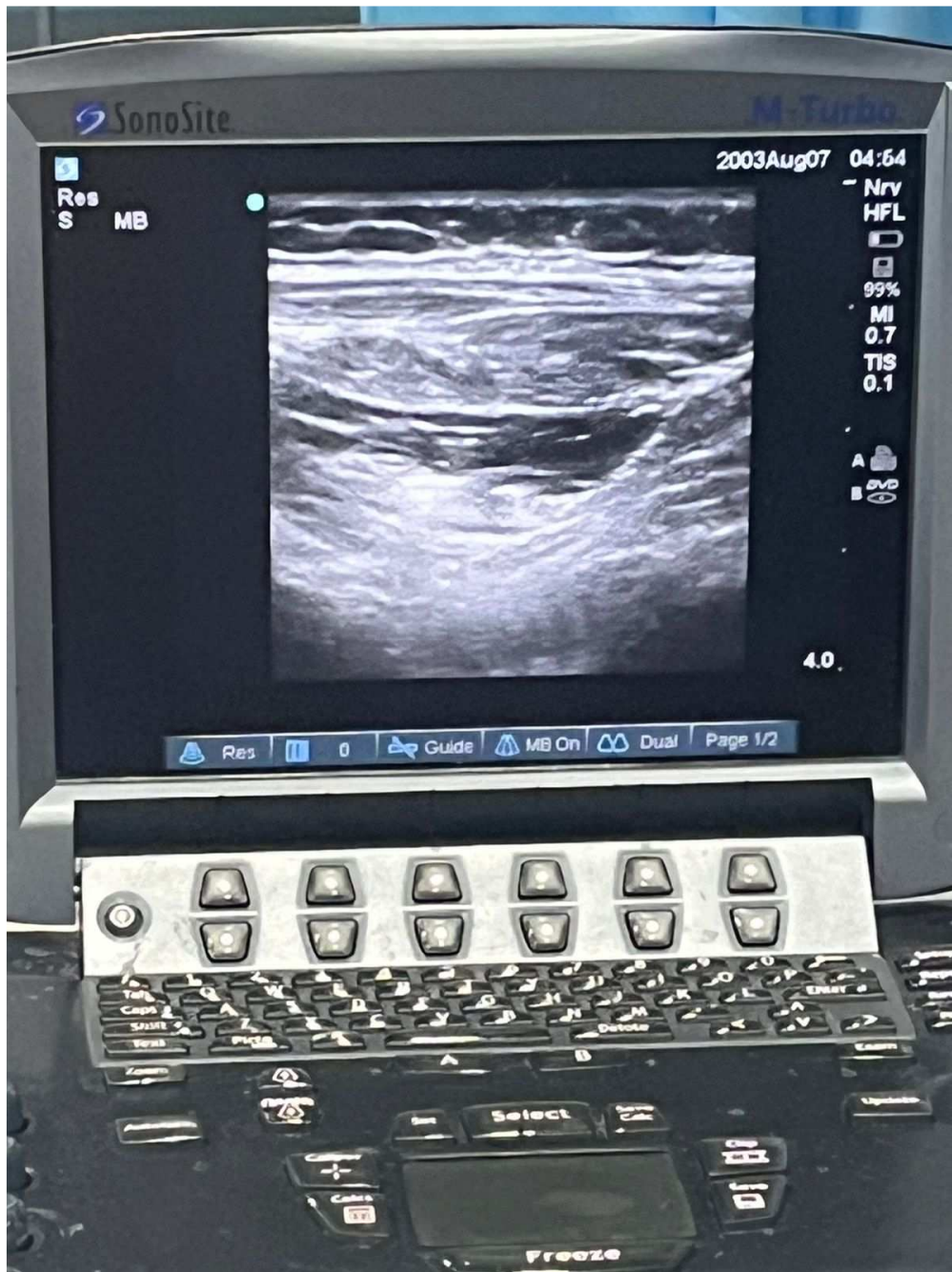
Photograph 2- 0.75% Ropivacaine



Photograph 3: Dexmedetomidine Ampoule



Photograph 4-Performing TAP block



Photograph 5- USG image of TAPB

ANNEXURE IV – KEY TO MASTERCHART

ASA	American Society of Anesthesiologists
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
MAP	Mean Arterial Pressure
HR	Heart Rate
SpO2	Peripheral Oxygen Saturation
M, F	Male, Female
VAS	Visual Analogue Score

ROPIVACAINE GROUP

S.no.	AGE	SEX	ASA GRADE	DIAGNOSIS	SURGERY	VAS SCORE.					TIME FOR RESCUE ANALGESIA				POSTOP VITALS				
						2 HOURS	4 HOURS	9 HOURS	18 HOURS	24 HOURS	HR	SPO2	SBP	DBP	HR	SPO2	SBP	DBP	
1	47	M	2	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	0	1	1	2	3	12	88	97	180	100	76	100	142	76
2	49	F	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	2	1	1	2	3	12	78	99	124	82	82	100	116	56
3	63	M	1	EPIGASTRIC HERNIA + CHOLELITHIASIS	LAP CHOLECYSTECTOMY + HERNIA REPAIR	1	1	2	2	4	9	67	100	136	84	77	100	124	72
4	58	F	1	INTUSSECEPTION	DIAGNOSTIC LAP AND PROCEED	2	2	2	3	2	10	76	98	150	90	78	100	142	66
5	24	F	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	1	1	1	1	3	12	84	97	120	70	78	100	114	62
6	48	F	1	APPENDICITIS	LAP APPENDICECTOMY	1	2	2	3	2	16	92	99	114	64	88	100	132	72
7	61	F	2	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	2	1	1	3	2	9	77	96	130	82	68	99	126	76
8	62	M	1	CA STOMACH	DIAGNOSTIC LAP AND PROCEED	2	1	2	2	3	12	82	99	134	74	88	100	127	64
9	54	M	1	DUODENAL ULCER	GASTROJEJUNOSTOMY	2	2	2	1	2	12	72	98	112	74	68	100	122	72
10	58	M	1	INGUINAL AND UMBILICAL HERNIA	LAP HERNIA REPAIR	1	1	1	1	2	16	98	100	124	68	76	100	138	74
11	59	F	1	CA ENDOMETRIUM	TAH+BSO+ INGUINAL LYMPH NODE DISSECTION	2	2	2	3	2	9	88	99	136	82	82	100	126	74
12	44	M	2	UMBILICAL HERNIA	LAP HERNIA REPAIR	1	1	2	2	4	12	74	100	140	90	77	99	136	82
13	32	F	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	1	1	1	1	3	18	66	98	118	74	62	100	110	64
14	39	F	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	1	1	1	2	3	12	77	97	137	84	64	99	128	78
15	33	F	1	APPENDICITIS	LAP APPENDICECTOMY	2	1	1	3	3	9	89	98	122	72	79	99	112	68
16	52	M	1	UMBILICAL HERNIA	LAP HERNIA REPAIR	2	1	3	3	3	10	77	100	134	62	67	100	129	68

17	33	F	2	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	2	1	1	2	2	12	76	98	128	77	82	99	121	66
18	47	M	1	INGUINAL HERNIA	LAP HERNIA REPAIR	1	1	1	2	2	9	67	100	137	76	79	100	116	58
19	44	F	2	SUPRAUMBILICAL REPAIR	DIAGNOSTIC LAP AND PROCEED	1	1	2	2	2	12	82	100	123	76	66	100	116	67
20	30	F	1	APPENDICITIS	LAP APPENDICECTOMY	1	1	2	2	2	18	62	98	100	70	58	100	102	64
21	46	F	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	1	1	2	2	2	12	88	100	138	77	81	100	100	72
22	43	M	2	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	1	1	2	1	2	10	66	98	124	66	77	100	123	45
23	48	F	2	APPENDICITIS	LAP APPENDICECTOMY	2	2	2	1	3	16	74	100	132	68	68	99	108	77
24	45	M	2	APPENDICITIS	LAP APPENDICECTOMY	2	1	1	3	2	12	67	100	123	64	72	100	110	64
25	62	M	2	INGUINAL HERNIA	LAP HERNIA REPAIR	1	1	1	3	2	12	76	100	142	77	82	100	128	72
26	57	M	2	CHOLELITHIASIS + UMBILICAL HERNIA	LAP CHOLECYSTECTOMY + HERNIA REPAIR	1	1	1	1	3	10	82	100	132	72	77	100	117	64
27	48	M	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	1	2	2	3	3	12	54	99	116	64	62	100	121	68
28	57	M	2	INGUINAL HERNIA	LAP HERNIA REPAIR	1	1	1	2	3	16	76	100	154	72	68	99	126	58
29	63	M	2	UMBILICAL HERNIA	LAP HERNIA REPAIR	1	2	2	2	3	18	82	100	170	110	76	100	142	68
30	21	M	1	APPENDICITIS	LAP APPENDICECTOMY	1	1	1	1	3	12	78	100	121	68	62	100	116	62
31	50	M	2	GALL BLADDER EMPHYSEMA	LAP CHOLECYSTECTOMY	1	2	2	2	3	12	84	100	154	72	78	100	118	69
32	40	F	1	UMBILICAL HERNIA	LAP HERNIA REPAIR	1	1	1	1	2	9	78	100	142	77	82	100	123	48
33	60	M	2	CALCULOUS CHOLECYSTITIS	LAP CHOLECYSTECTOMY	1	2	2	2	2	12	108	100	142	77	98	100	138	82
34	55	M	1	CALCULOUS CHOLECYSTITIS	LAP CHOLECYSTECTOMY	1	2	1	1	3	12	96	100	133	64	58	100	121	54

BUPIVACAINE GROUP

S.NO.	AGE	SEX	ASA GRADE	DIAGNOSIS	SURGERY	VAS SCORE.					TIME FOR RESCUE ANALGESIA	PREOP VITALS				POSTOP VITALS			
						2 HOURS	4 HOURS	9 HOURS	18 HOURS	24 HOURS		HR	SPO2	SBP	DBP	HR	SPO2	SBP	DBP
1	20	F	1	APPENDICITIS	LAP. APPENDICECTOMY	1	1	3	3	4	6	78	100	110	70	70	100	120	70
2	60	M	2	APPENDICITIS	LAP. APPENDICECTOMY	1	1	2	3	5	8	82	100	160	100	66	100	140	70
3	24	M	2	APPENDICITIS	LAP. APPENDICECTOMY	0	1	1	3	4	6	77	100	120	80	65	100	130	80
4	51	M	2	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	1	2	2	3	3	6	68	100	100	60	77	100	110	80
5	62	F	2	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	1	2	4	4	3	6	65	100	150	90	68	100	130	70
6	63	F	2	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	1	1	3	4	5	8	78	100	90	60	65	100	120	70
7	28	M	1	APPENDICITIS	LAP. APPENDICECTOMY	0	2	3	3	4	7	76	100	110	80	88	100	120	70
8	44	M	1	MEDIAN ARCUATE LIGAMENT SYNDROME	LAPAROSCOPY & PROCEED	1	1	1	1	3	10	110	97	120	90	88	100	130	70
9	59	F	2	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	2	3	4	4	5	6	97	99	100	70	76	100	110	60
10	62	M	2	INGUINAL HERNIA + CHOLELITHIASIS	LAP CHOLECYSTECTOMY + HERNIA REPAIR	1	1	1	2	3	5	84	98	130	65	64	100	120	90
11	18	F	1	APPENDICITIS	LAP. APPENDICECTOMY	2	3	4	3	3	6	120	99	120	90	100	98	110	65
12	52	M	1	UMBILICAL HERNIA	LAP HERNIA REPAIR	0	1	4	3	3	8	54	96	140	72	62	100	120	84
13	34	M	1	PERFORATED APPENDIX	OPEN APPENDICECTOMY	2	3	3	2	3	6	126	97	140	80	87	100	132	76

14	51	M	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	0	1	3	2	4	6	73	99	126	72	68	99	116	64
15	22	M	1	INGUINAL HERNIA + CHOLELITHIASIS	LAP HERNIA REPAIR	2	2	2	1	3	6	87	97	116	54	79	99	123	67
16	56	F	1	FIBROID UTERUS	TLH	3	2	3	2	4	8	82	97	107	64	88	100	100	70
17	39	F	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	2	3	3	2	3	4	79	99	142	56	84	100	128	86
18	25	F	1	APPENDICITIS	LAP. APPENDICECTOMY	2	3	2	2	3	4	62	100	143	72	66	100	97	64
19	42	M	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	2	3	3	2	4	6	97	100	109	65	82	100	110	62
20	18	F	1	MESENTRIC LYMPHADENITIS	DIAGNOSTIC LAP AND PROCEED	2	2	2	1	3	6	67	100	127	65	62	100	108	64
21	47	F	2	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	2	2	3	3	4	8	84	100	112	74	88	99	122	74
22	30	M	1	APPENDICITIS	LAP. APPENDICECTOMY	2	3	4	3	3	7	98	100	142	67	77	97	132	77
23	40	M	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	2	2	3	3	4	6	83	100	128	77	75	100	132	66
24	27	F	1	CBD CALCULI	LAP CHOLECYSTECTOMY	1	3	3	3	5	4	78	100	128	74	77	100	114	62
25	48	F	1	CHOLECYSTITIS	LAP CHOLECYSTECTOMY	2	3	3	2	4	8	84	100	133	78	76	99	121	74
26	60	M	2	INGUINAL HERNIA	LAP HERNIA REPAIR	2	2	3	3	3	6	74	100	122	72	68	100	109	72
27	35	F	1	UMBILICAL HERNIA	LAP HERNIA REPAIR	2	2	3	4	3	8	72	100	154	77	62	100	128	64
28	55	M	2	CALCULOUS CHOLECYSTITIS	LAP CHOLECYSTECTOMY	1	2	3	3	2	10	100	99	100	68	92	100	112	74
29	45	F	1	UMBILICAL HERNIA	LAP HERNIA REPAIR	2	2	3	3	4	9	92	100	132	76	77	100	114	72
30	33	F	1	APPENDICITIS	LAP. APPENDICECTOMY	2	2	4	2	3	9	112	99	148	82	88	100	128	76
31	38	F	1	APPENDICITIS	LAP. APPENDICECTOMY	1	1	2	3	3	8	66	100	128	64	58	100	116	72
32	32	F	1	CHOLELITHIASIS	LAP CHOLECYSTECTOMY	2	3	3	2	5	4	102	100	138	76	88	100	124	72
33	47	M	2	INGUINAL HERNIA	LAP HERNIA REPAIR	2	2	3	3	3	9	65	100	142	77	58	100	124	65
34	29	M	1	PAIN ABDOMEN UNDER EVALUATION	DIAGNOSTIC LAP AND PROCEED	2	3	3	2	4	9	109	97	142	86	78	100	128	79