"COMPARISON OF ORAL MORPHINE AND NEUROLYTIC CELIAC PLEXUS BLOCK IN PATIENTS WITH UPPER ABDOMINAL MALIGNANCIES"

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In partial fulfilment for the award of the degree of

DOCTOR OF MEDICINE
IN
ANAESTHESIOLOGY

BRANCH X

INSTITUTE OF ANAESTHESIOLOGY AND CRITICAL CARE
MADRAS MEDICAL COLLEGE
CHENNAI- 600003

MAY 2018

CERTIFICATE

This is to certify that the dissertation titled, **"COMPARISON OF ORAL MORPHINE AND NEUROLYTIC CELIAC PLEXUS BLOCK IN PATIENTS WITH UPPER ABDOMINAL MALIGNANCIES**" Submitted by **Dr.ANBARASAN.B** in partial fulfilment for the award of the degree of **DOCTOR OF MEDICINE** in Anaesthesiology by The Tamilnadu Dr.M.G.R Medical University, Chennai is a bonafide record of work done by him in the **INSTITUTE OF ANAESTHESIOLOGY & CRITICAL CARE"** Madras Medical College, during the academic year 2015-2018.

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DECLARATION

I hereby declare that the dissertation titled, **"COMPARISON OF ORAL MORPHINE AND NEUROLYTIC CELIAC PLEXUS BLOCK IN PATIENTS WITH UPPER ABDOMINAL MALIGNANCIES**" has been prepared by me under the guidance of **Prof. Dr.M.Bhavani M.D.,** Professor of Anaesthesiology, Institute of Anaesthesiology & Critical care, Madras Medical college, Chennai, in partial fulfillment of the regulations for the award of the degree of M.D (Anaesthesiology), examination to be held in May 2018.

This study was conducted by me at Institute of Anaesthesiology & Critical care, Madras Medical College, Chennai.

I have not submitted this dissertation previously to any journal or any university for the award of any degree or diploma.

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ABBREVIATIONS

CNS	Central Nervous System
DRG	Dorasal Root Ganglion
Na^+	Sodium ion
Ca^{2+}	Calcium ion
ATP	Adenosine Triphosphate
NGF	Nerve Growth Factor
CA	Carcinoma
CRF	Cortisol Releasing Factor
GABA	Gaba Amino Benzoic Acid
cAMP	Cyclic Adenosine Monophosphate
PLC	Phospholipase-C
PKC	Protein kinase-C
GRK	G protein coupled receptor kinase
NCPB	Neurolytic celiac plexus block
CPB	celiac plexus block
ECG	electrocardiogram
VAS	visual analoge scale
USG	ultrasonogram
ASA/PS	American society of anasthesiologistis / physical status
MAP	Mean arterial pressure

INTRODUCTION

The majority of patients with advanced upper abdominal malignancies suffer from moderate to severe pain because of the disease process and inadequate pain relief given.

Since pain is a predominant symptom in 90% of these patients in advanced stage of the disease good pain management is a necessity . Although most clinicians are well aware of the importance to adequately treat pain, it was found that half of cancer patients have insufficient pain control and many of them die in pain. The sheer potential for suffering from cancer can be a terrible experience for anyone who bears this diagnosis, while pain is probably one of the most frightening of all cancer symptoms for patients and their families which negatively impact their quality of life .Patient and their families tend to be under great distress after the diagnosis of cancer. Suboptimal pain relief can be very debilitating. Although many of these patients carry a very poor prognosis, prompt and effective pain control can prevent needless suffering, improve quality of their lives and spare families the feeling of helplessness and despair. Although cancer is a terminal disease , there should be no reason to deny a patient the opportunity to live free of pain. Severe pain can interfere with the physical rehabilitation, mobility and proper nutrition of the patient. Therefore, the goal of pain control in a patient should be to

optimise the patient's comfort and function while avoiding unwanted adverse effects from medication.

Although there are many modes of pain control, opiates remains the mainstay of pain relief. Morphine is the standard opiate and drug of choice in treatment of moderate to severe cancer pain. Oral Morphine is recommended due to its ease of administration and convenience of use.

Therapeutic nerve blocks are used for patients in the treatment of cancer related pain. In these patients therapeutic nerve blocks extend the treatment range when conservative method failed to achieve tolerable pain levels (or) side effects (or) both. In patients with upper abdominal malignancies like CA stomach, pancreas, gallbladder, neurolysis of celiac plexus may be used for pain treatment.

This study was done to evaluate the role of neurolytic celiac plexus block on pain relief and compare the effects of oral morphine and neurolytic celiac plexus block.

AIMS AND OBJECTIVES

To compare the analgesic efficacy of oral morphine and neurolytic celiac plexus block in patients with upper abdominal malignancies.

Secondary objectives

1. To assess side effects
2. To assess Complications
3. To assess the performance status of the patient

PAIN

International association for study of pain defines, " pain is an unpleasant sensory and emotional experience associated with actual or potential damage or describe in terms of such damage**".** It is often regarded as 5th vital sign. Roughly divided into two broad categories physiologic pain and pathologic pain.

Physiologic (acute nociceptive) pain is an essential early warning sign that usually elicits reflex withdrawal and there by promotes survival by protecting the organism from further injury. In contrast, **pathologic** (eg. neuropathic) pain is an expression of the maladaptive operation of the nervous system.

COMPONENTS OF PAIN:

1.Transduction - Process by which a noxious stimulus(heat, cold, mechanical distortion) is converted to an electrical impulse in sensory nerve endings.

2. Transmission- Conduction of electrical impulses to the CNS.

3. Modulation -Process of altering pain transmission.

4. Perception- likely mediated through the thalamus.

Physiologic changes in persistent pain:

Physiologic pain mediated by sensory system:

- Primary afferent neurons
- Spinal interneurons
- Ascending tract
- Supraspinal areas

Excitatory mechanism:

Trigeminal and DRGs give rise to light threshold pain.

Aδ fibres and C fibres innervates peripheral tissues (skin, muscle, joint, viscera).

These specialised primary afferent neurons also called nociceptors transduce noxious stimuli into action potentials and conduct them to the dorsal horn of the spinal cord.

When peripheral tissue is damaged primary afferent neurons are sensitized or directly activated or both, by a variety of thermal, mechanical and chemical stimuli.

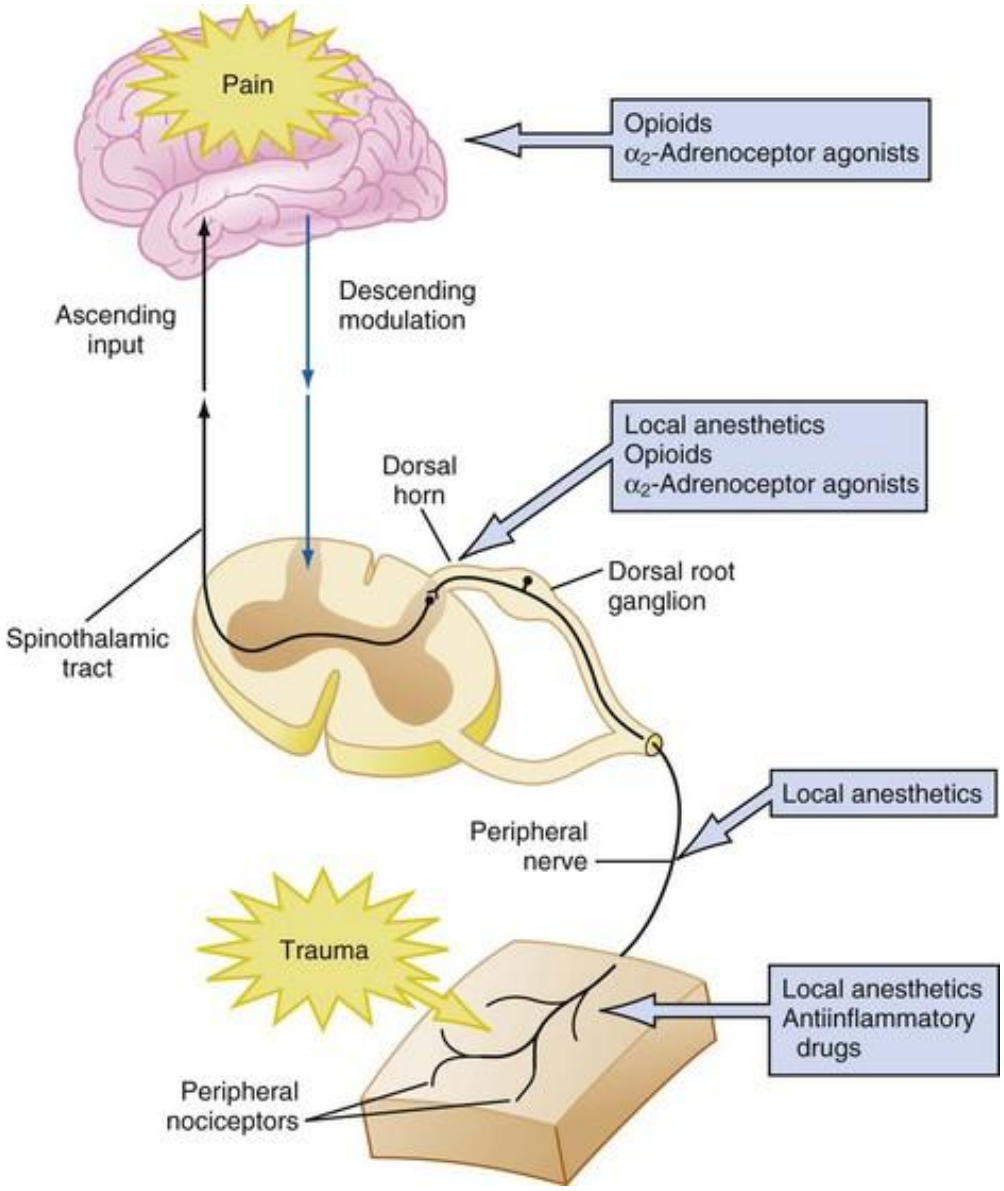
Examples: protons, sympathetic amines, ATP, glutamate, neuropeptides, NGF, bradykinin, chemokines.

These agents lead to opening (GATING) of cation channels in the neuronal membrane.

Gating produces an inward current of Na^{+} and Ca^{2+} ions into peripheral nociceptor terminal.

Thereafter the impulses are transmitted to spinal neurons, brainstem, thalamus and cortex.

Fig.1: PAIN PATHWAY



Inhibitory mechanism:

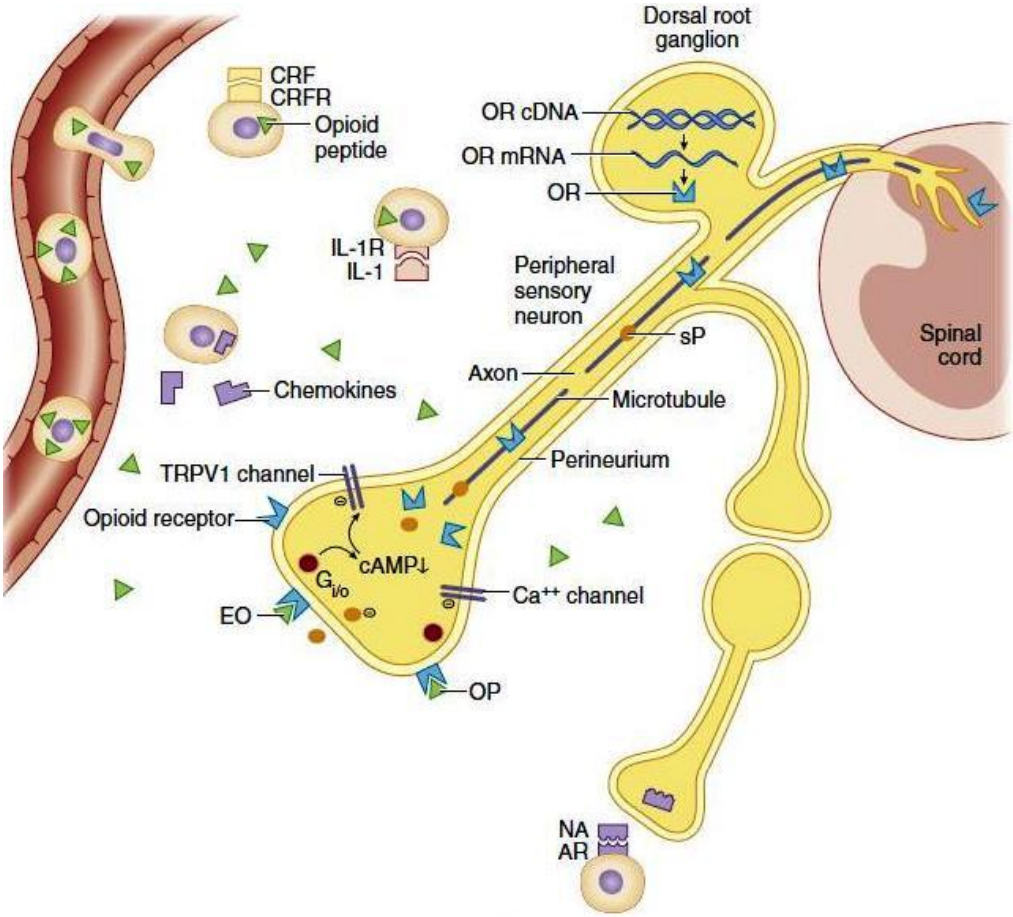
Concurrent with the events described above powerful endogenous mechanisms counteracting pain unfold both in the periphery and in CNS. In injured tissues this process results from interactions between leukocyte derived opioid peptides and peripheral nociceptor terminals carrying opioid receptors and/or by anti inflammatory cytokines.

Inflammation of peripheral tissue leads to increased expression, axonal transport and enhanced G-protein coupling of opioid receptors in DRG neurons as well as enhanced permeability of the perineurium.

These depends on sensory neuron electrical activity, the production of proinflammatory cytokines and the presence of nerve growth factor within the inflamed tissue. In parallel, opioid peptide containing immune cells extravasate and accumulate in the inflamed tissue.

In response to stress, catecholamines, CRF, cytokines, chemokines or bacteria, leukocytes secrete opioids. The latter activate peripheral opioid receptors and produce analgesia by inhibiting the excitability of nociceptor with the release of excitatory neuropeptides or both.In spinal cord, inhibition is mediated by the release of opioids, GABA, glycine from interneurons which activate presynaptic opioids or GABA receptors or both on central nociceptor terminals to reduce excitatory transmitter release. When the intricate balance of biologic, psychological and social factors becomes disturbed, chronic pain develops.

Fig.2: Endogenous antinociceptive mechanisms within peripheral injured tissue



Corticotropin-releasing factor (CRF), interleukin-1β (IL-1),noradrenaline [NA], CRF receptors (CRFR), adrenergic receptors (AR), Exogenous opioids (EO), endogenous opioid peptides (OP), opioid receptors (OR), substance P (sP) *cAMP*, Cyclic adenosine monophosphate

Classification:

Non malignant pain

Examples : neuropathic, musculoskeletal, inflammatory

Frequent symptoms would be spontaneous lancinating, shooting, burning pain, hyperalgesia and allodynia or combination of such pain.

Malignant pain

Originate from invasion of tumour into tissues innervated by primary afferent neurons or directly into peripheral nerve plexus.

Drugs used for chronic pain

Analgesic drugs interfere with generation or transmission or both the impulses following noxious simulation in the nervous system (nociception) examples: NSAIDS

OPIOIDS

TABLE : 1 ANALGESIC DRUGS, TARGETS, MECHANISM & SIDE EFFECTS

Drugs	Targets	Mechanisms	Functional consequences	Side effects
NSAID	Cycloxygenases (COX-1 & 2)	↓Prostaglandins ↓Thromboxanes	↓sensitization of sensory neurons, ↑Inhibition of spinal neurons	GIT ulcers, Perforation, bleeding, renal imparment, Thrombosis, MI, stroke.
OPIOIDS	G-protein coupled μ,κ,δ receptors	↓cAMP, ↓Ca^{2+} currents, ↑K^{+} currents	↓Excitability of peripheral & central neurons ↓Release of excitatory neuro transmitters	**μ,δ**:sedation, nausea, euphoria, respiratory depression, constipation **κ:**dysphoria,diuresis, sedation.

OPIOIDS:

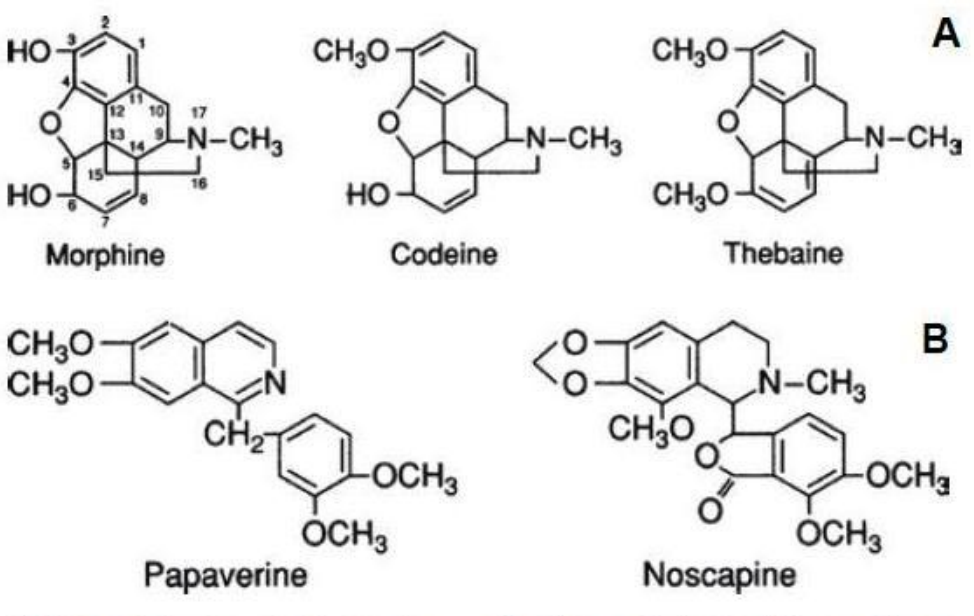
Opioids remain the mainstay of modern perioperative care and pain management Opium derived from Greek word *opion* (poppy juice) Drugs derived from opium are opiates. Morphine is the best known opiate.

Chemical structure of opium alkaloids

Two distinct chemical classes

CHEMICAL STRUCTURE

A.Phenanthrenes and B. Benzylisoquinolines



The principal phenanthrene alkaloids present in opium are morphine, codeine and thebaine

TABLE.2 : CLASSIFICATION OF OPIOIDS

Agonists	Agonists-Antagonists	Antagonists
Morphine	Pentazocine	Naloxone
Morphine-6-glucuronide	Butorphanol	Naltrexone
Meperidine	Nalbuphine	Nalmefene
Sufentanil	Buprenorphine	
Fentanyl	Nalorphine	
Alfentanil	Bremazocine	
Remifentanil	Dezocine	
Codeine	Metazinol	
Hydromorphone		
Oxymorphone		
Hydrocodone		
Oxycodone		
Propoxyphene		
Methadone		
Tramadol		
Heroin		

TABLE.3: CLASSIFICATION OF OPIOID RECEPTORS

	Mu_1	**Mu_2**	**Kappa**	**Delta**
Effect	Analgesia (supraspinal,spinal) Euphoria Low aduse potential Miosis Bradycardia Hypothermia Urinary retention	Analgesia (spinal) Depression of ventilation Physical dependence Constipation (marked)	Analgesia (supraspinal, spinal) Dysphoria, sedation Low abuse potential Miosis Diuresis	Analgesia (supraspinal, spinal) Depression of ventilation Physical dependence Constipation (minimal) Urinary retention
Agonists	Endorphins Morphine Synthetic opioids	Endorphins Morphine Synthetic opioids	Dynorphins	Enkephalins
Antagonists	Naloxone Naltrexone Nalmefene	Naloxone Naltrexone Nalmefene	Naloxone Naltrexone Nalmefene	Naloxone Naltrexone Nalmefene

Mechanism of action:

1. Acts as agonists at specific opioid receptors at presynaptic and postsynaptic sites in CNS (brainstem & spinal cord) as well as in the periphery.
2. Opioid receptors normally are activated by three endogenous peptide opioid receptor ligands known as enkephalins, endorphins & dynorphins. Opioid mimic the action of these endogenous

ligands by binding to opioid receptors resulting in activation of pain modulating (antinociceptive) system.

Pharmacokinetics:

Morphine can be taken orally, sublingually, subcutaneously, rectally, intranasally, intravenously, intrathecally, epidurally & inhaled via nebuliser.

- Intravenous is the most common method of administration.
- Morphine is subject to extensive first pass metabolism. So if taken orally only 40 to 50% of the dose reaches the CNS.
- Levels peak approximately in 30 mins.
- Metabolised primarily in liver into morphine-3-glucuronide (60%) & morphine-6-glucuronide (6-10%) via glucuronidation by phase II metabolic enzyme UDP-glucuronyltransferase 2B7.
- Elimination t1/2-120 mins, 87%excreted in urine within 72 hrs of administration.

Effects:

CVS - orthostatic hypotension, bradycardia in high doses.

RS - respiratory depression, decreased ventilatory response to hypoxia and hypercapnia, anti tussive.

CNS - potent analgesic, cause drowsiness, relieve anxiety, euphoria.

GIT - decreases GI motility, gastric acid secretions and pancreatic secretions, vasospasm of sphincter of oddi, nausea, vomiting and constipation.

GENITOURINARY-increase tone of ureters, bladder detrusor and sphincter, precipitates urinary retention.

OTHERS - diaphoresis, pruritus.

Uses:

1. as premedication
2. as an analgesic in management of moderate to severe pain
3. treatment of left ventricular failure
4. to provide analgesia during terminal care

Side effects

1. respiratory depression
2. nausea and vomiting
3. hallucinations
4. physical dependence

Fig. 3: OPIOID RECEPTOR SIGNALING AND RECYCLING

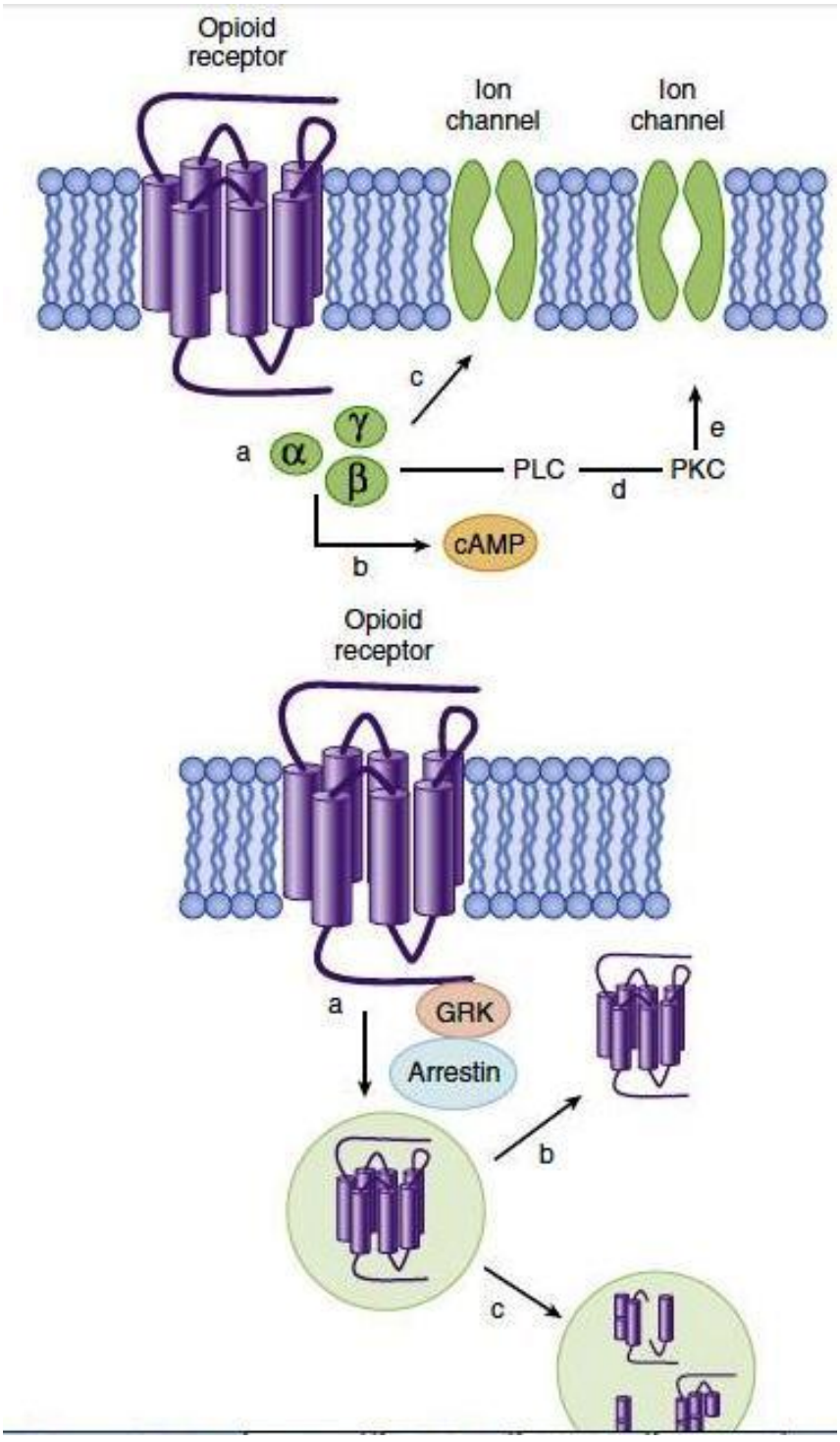


Fig.4: GATE THEORY OF PAIN CONTROL IN SPINAL CORD

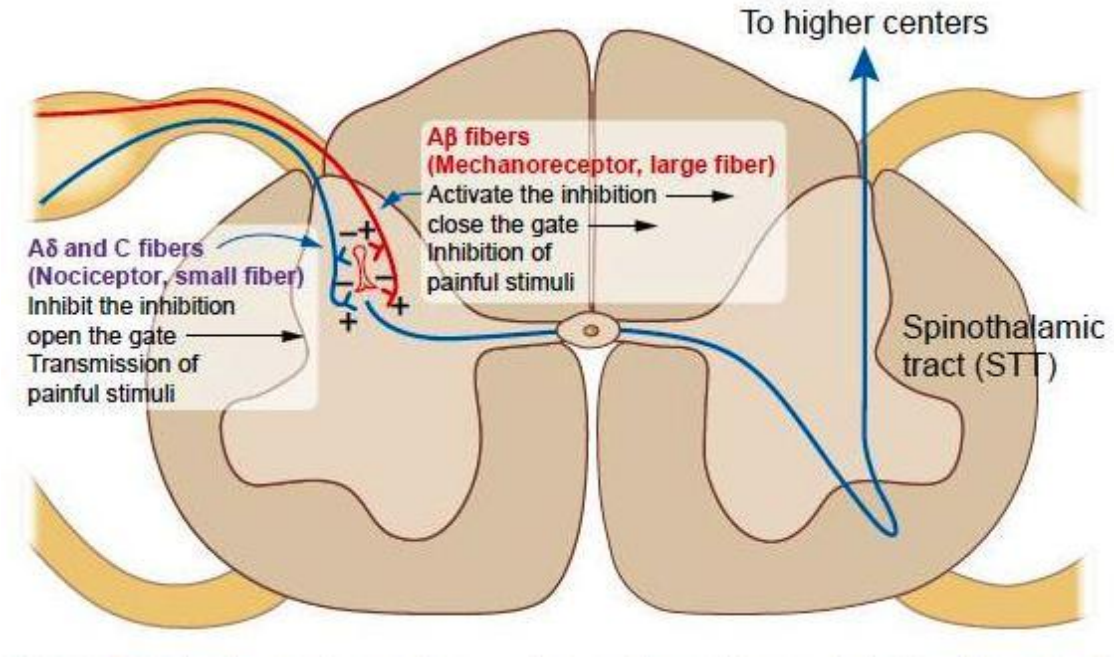
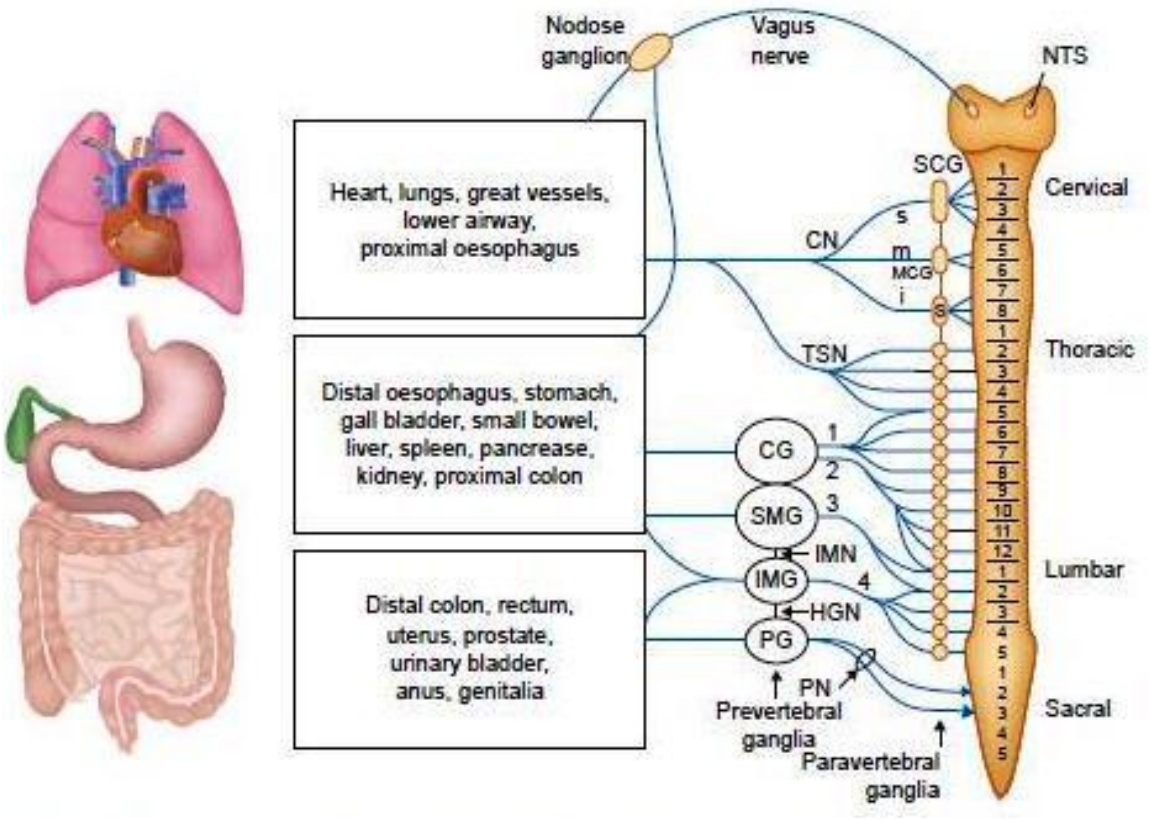


Fig.5: VISCERAL INNERVATION



NTS- nucleus solitarii ,CG-Celiac ganglia, SMG&IMG-superior and inferior mesentric ganglia ,PG-pelvic ganglia, SCG&MCG-superior and middle cervical ganglia, S-stellate ganglia, CN-Cardiac nerves,TSN-thoracic splanchnic nerves[1,2,3&4-greater, least, lesser and lumbar splanchnic nerves respectively], IMN-intermesentric nerve, HGN-hypogastric nerve, PN-pelvic nerve

Fig.6: WHO ANALGESIC LADDER

CELIAC PLEXUS ANATOMY AND SONOANATOMY

Celiac ‘plexus’ (ganglia and interconnecting fibres that converge in a well defined location) is the largest plexus of sympathetic nervous system that innervates the upper abdominal organs (pancreas, diaphragm, liver, spleen, stomach, small bowel, ascending colon and proximal part of transverse colon, adrenal glands, kidneys, abdominal aorta, mesentery) derived from embryonic foregut. The celiac plexus embedded in loose areolar tissue, lies within the retroperitoneal space posterior to the stomach and pancreas close to the celiac axis and is separated from vertebral column by crus of diaphragm. It overlaps the aorta at the level of first lumbar vertebra. It is a dense ganglia around aorta with considerable variability in size (0.5-4.5 cm), number (1-5) and position (T12 -L1 disc space to middle of L2 - vertebral body). Left celiac plexus is located relatively caudal to that of right counterpart.

The celiac plexus is composed of pre- and postganglionic sympathetic, parasympathetic and visceral sensory afferent fibres. Pre-ganglionic sympathetic fibres that are carried in greater (T5-10), lesser (T10-11) and least (T12) sphlanchnic nerves, relay in celiac ganglia, which also receives parasympathetic fibres from the celiac branch of right vagus nerve. From there, post-ganglionic fibres travel with blood vessels and subsidiary plexus to innervate the abdominal viscera.

Sphlanchnic nerve block and CPB have often been referred interchangeably. Factually, they are different. When the blockade is limited solely to the pre-ganglionic structures (greater, lesser, least sphlanchnic nerves) that synapse at the celiac ganglia it is called ‘sphlanchnic nerve block’. Anatomically, blockade of celiac plexus, which arises from pre-ganglionic sphlanchnic nerves, vagal pre-ganglionic parasympathetic fibres, sensory fibres from the phrenic nerve and post-ganglionic sympathetic fibres, is termed ‘celiac plexus block’.

Structures surrounding the celiac plexus, unless distorted profoundly by organomegaly or tumor spread, are configuratively consistent and lie within the retroperitoneal space. Aorta lies anterior and slightly to the left of anterior vertebral body margin . The inferior vena cava lies to the right and kidneys are posterolateral to the great vessels. The stomach, left renal vein and pancreas lies anterior to the celiac plexus.

Fig.7: ANATOMY OF CELIAC PLEXUS

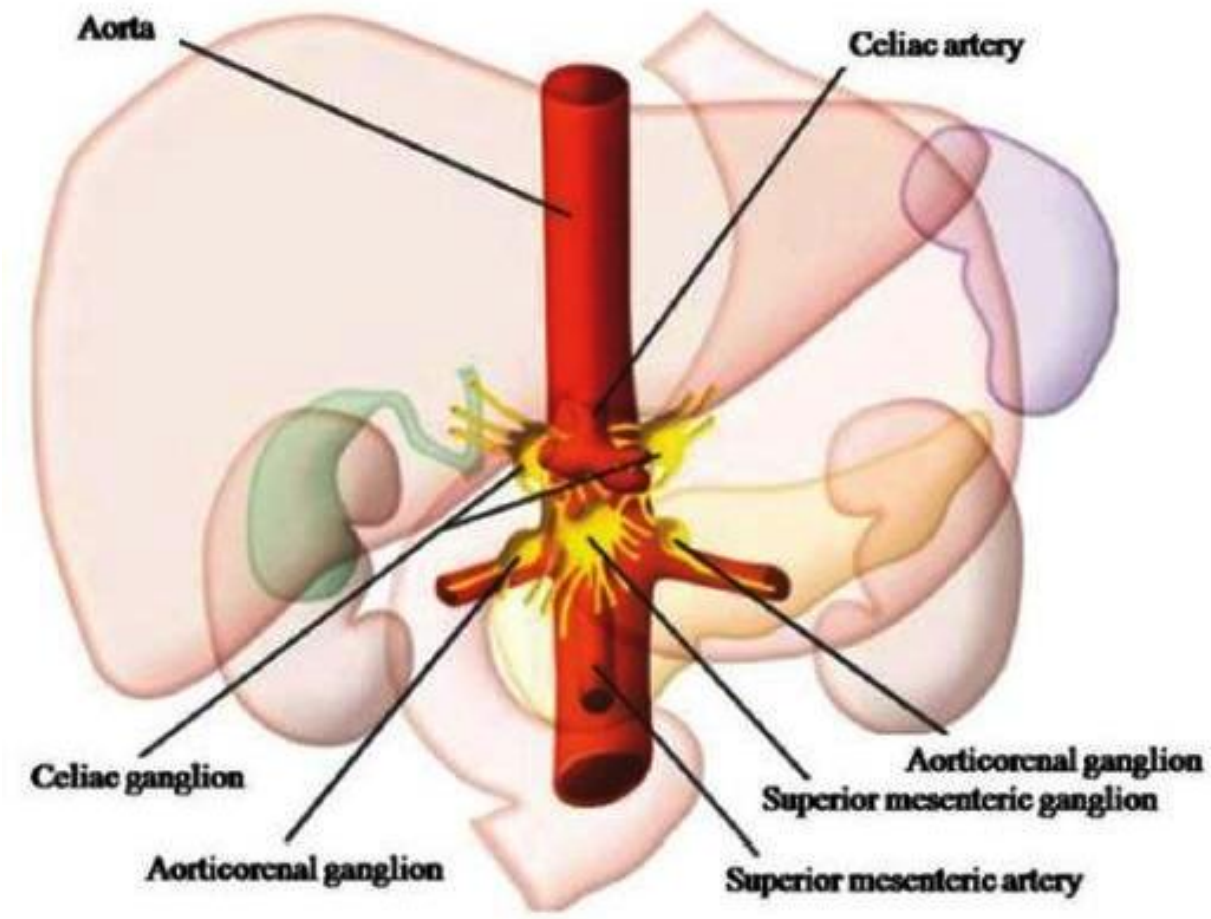


Fig.8: LATERAL VIEW OF CELIAC PLEXUS

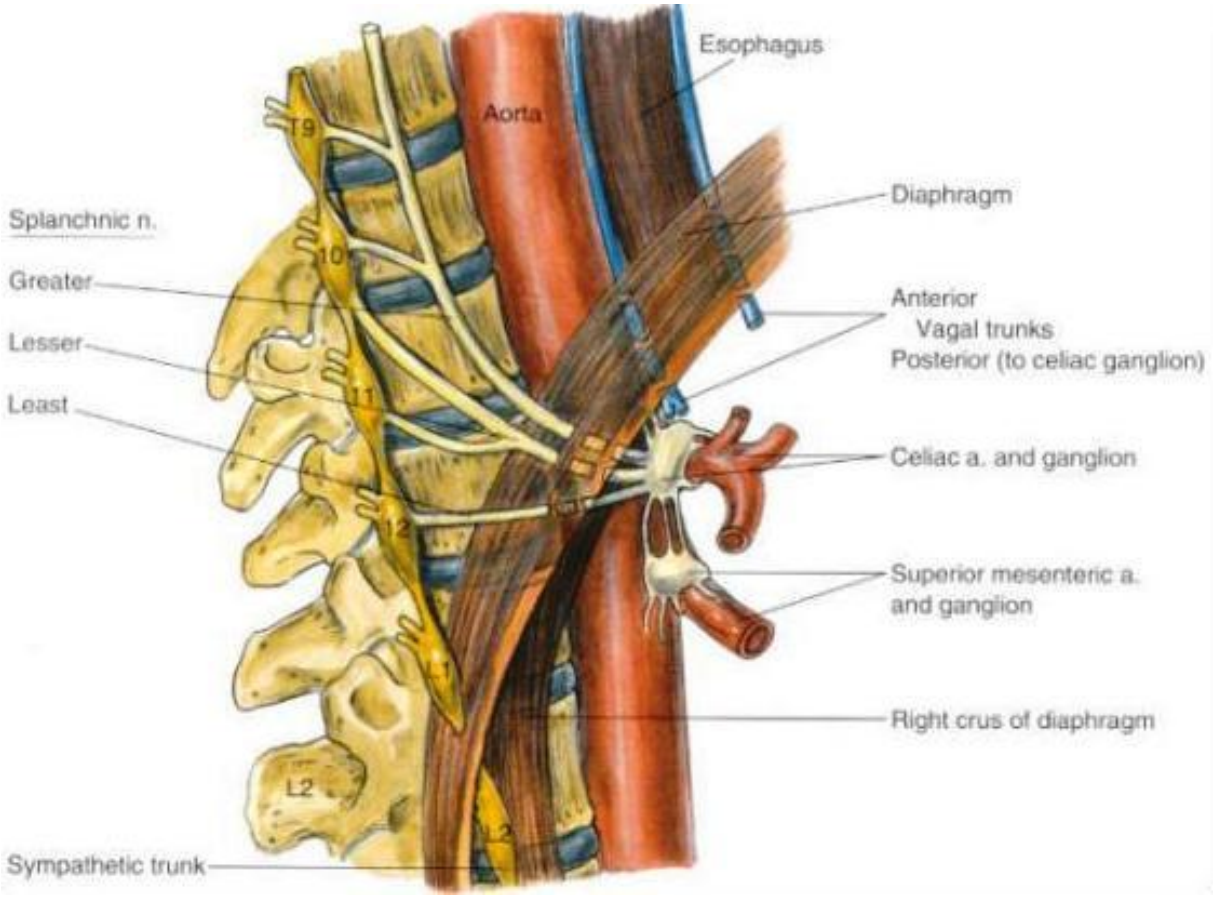
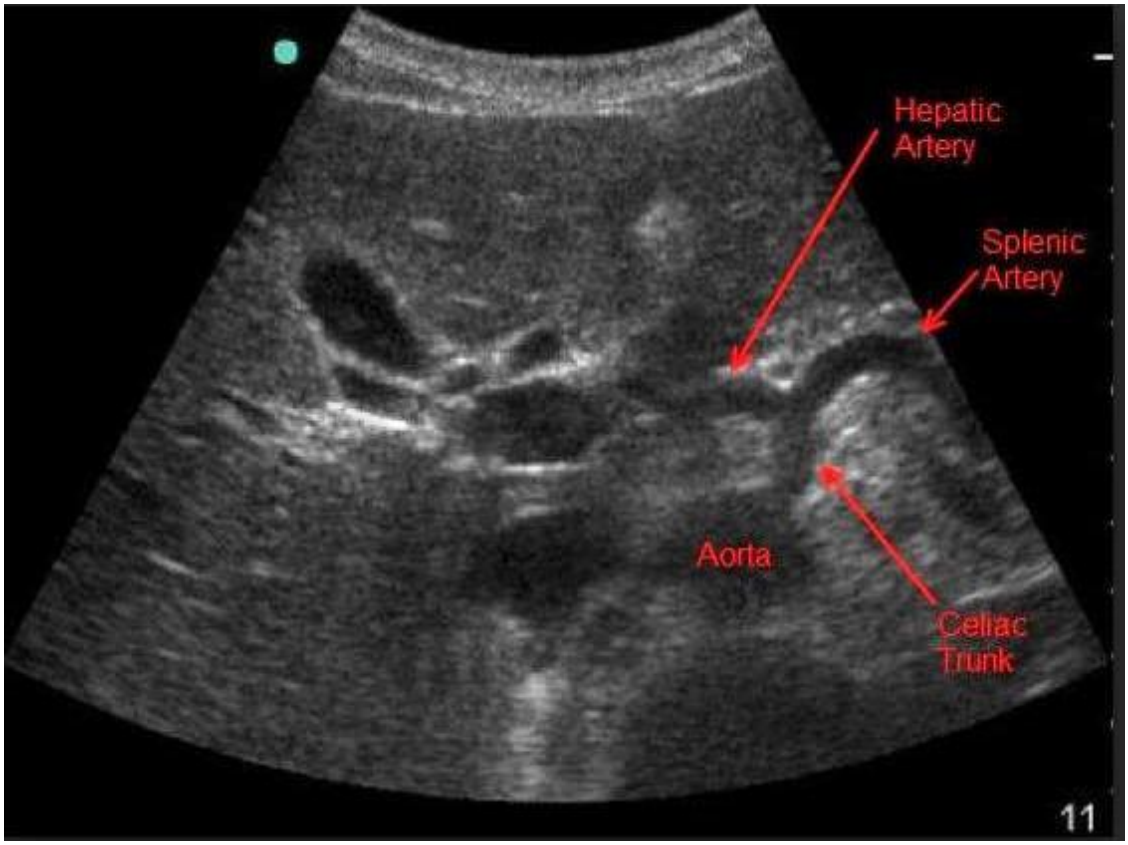


Fig.9: SONOANATOMY OF CELIAC PLEXUS



CELIAC PLEXUS BLOCK

Introduction:

Celiac plexus blockade with local anaesthetic (LA) were introduced as early as in 1914, primarily for surgical anaesthesia. Soon, over the ensuring few decades, the emergence of subarachnoid block, variable results and technical demands of celiac plexus block (CPB) and introduction of neuromuscular blocking drugs into clinical practice of anaesthesia, led to the disfavour of CPB among anaesthesiologist for surgical anaesthesia.

- Mid-20th century saw an expected but atypical crossover of the utility of CPB from a surgical anaesthetic to speciality of pain management. CPB was finally introduced to palliate abdominal pain secondary to a variety of aetiologies.

History:

1914 - Kappis introduced percutaneous technique for sphlanchnic nerve and CPB.

1946 - Pitkin overviewed the status of CPB for surgical anaesthesia and concluded that its utility was not beyond experimental tool.

1947 - Gage and Floyed described use of CPB in alleviating pain of pancreatitis.

1957 - Bridenbaugh and colleagues used CPB to treat pain sencondary to abdominal malignancy, Jones introduced alcohol - neurolysis of sphlanchnic nerves.

1965 - Moore further modified original Kappis's technique and established CPB as an important tool in pain management practice.

1971 - Gorbitz (use of plain X-ray to facilitate CPB) related radiology to pain management practice.

1979 - Hegedeus stressed the importance of fluoroscopic guidance in ascertaining 10 correct needle placement and radiocontrast material spread.

Moore/Hagga recommended CT scan to facilitate CPB.

Indications:

1. Diagnostic tool : CPB with local anaesthetic agent (LA) to determine whether the flank/retroperitoneal/ upper abdominal pain is sympathetically mediated via the celiac plexus.
2. Surgical anaesthetia :When general anaesthesia is contraindicated, CPB along with intercostal nerve block can be used for upper abdominal surgery.
3. Pain relief :

   - Abdominal pain of neoplastic/nonmalignant origin.

- Acute pain secondary to arterial embolisation of the liver (cancer therapy).

- Pain of "abdominal angina" (visceral arterial insufficiency).

4. Neurolysis : of celiac plexus to palliate pain of retroperitoneum /upper abdominal malignancy.
5. Prognostic tool : to assess the responsiveness of celiac plexus prior to neurolysis.
6. Anti-inflammation : CPB with steroid and/or LA markedly reduces morbidity and mortality associated with acute pancreatitis.
7. Visceral anaesthesia : CPB (anterior approach) with LA during interventional radiological procedures.
8. Others : Splenic vein thrombosis, carcinoma stomach.

Contraindications

1. Patients on anticoagulant therapy.
2. Patients with coagulopathy secondary to
    - Congenital abnormality
    - Cancer therapy
    - Liver abnormalities.
3. Local/intra-abdominal infection and sepsis.
4. Bowel obstruction.

5. Patients on disulfiram therapy.
6. Patients with physical dependence and drug seeking behaviour.

TECHNIQUES OF CELIAC PLEXUS BLOCK

1. Posterior (retrocrural) approach
2. Anterior approach
3. Transaortic approach
4. Transcrural approach
5. Transintervertebral disc approach
6. Thoracoscopic dennervation
7. Intraoperative
8. Radiographic guidance
    - fluoroscopy
    - CT guided

Patient preparation:

Positioning : Patient is positioned in supine position for anterior approach.

Preoperative medication:

- Oral anticoagulants must be stopped and coagulation status optimised.

- To continue anti-hypertensives
- Hydration and electrolyte imbalance should be corrected especially in debilitated and elderly patients.
- Opioids should be continued.

Monitoring : is essential during the performance of block in position and include pulse oximetry, NIBP and ECG.

Others : an intravenous cannula should be placed and 0.9% saline started. Supplemental oxygen should be administered via ventimask in obese/elderly patients.

NEURAL ABLATION METHODS

Neural ablation in pain practice can be divided into chemical and physical (thermal and electromagnetic field) neurolysis. Both methods produce nerve injury and result in degeneration of the nerve fibre from the distal to the lesion, along with its myelin sheath, known as "Wallerian degeneration".

The Wallerian degeneration causes a temporary interference in nerve cell transmission, resulting in a nociceptive block. This process does not completely disrupt the nerve cell, but preserves the basal lamina of the Schwann cells (non-neuronal cells that coat the axons in myelin). The Schwann cells in the peripheral nerve

potentially allows for axonal regeneration with reconnection to the proximal end of the nerve fibre.

1. ALCOHOL NEUROLYSIS

The chemical ablative agents are alcohol, phenol, and glycerol. They disrupt the transmission of pain signals for 3-6 months by causing Wallerian degeneration from the distal to the lesion.

Alcohol is the most commonly used agent for intractable visceral cancer pain to produce damage to the unmyelinated sympathetic chains and ganglia. However, potential complications arising from chemical neurolysis of the peripheral nerve include necrosis of the skin and other non-target tissue, neuritis, anesthesia dolorosa, and prolonged motor paralysis . Both the American Society of Anesthesiologists task force on chronic pain management and the American Society of Regional Anesthesia and Pain Medicine recommended in 2010 that chemical denervation should not be used in the routine care of non-cancer patients with chronic pain.

Sympathetic neurolysis for visceral cancer pain.

(1) Celiac plexus neurolysis,

(2) Superior hypogastric neurolysis and

(3) Ganglion impair neurolysis**.**

Basic mechanisms of alcohol neurolysis are clearly unknown, but it damages nerves by denaturing proteins and fatty substance extraction. In other words, the mechanisms involve extraction of cholesterol/ phospholipid and cerebroside from the neural membranes, and precipitation of mucoproteins and lipoprotein.

Currently, a recommended application of alcohol neurolysis is sympathetic neurolysis for the treatment of visceral cancer pain. However, the risks of chemical neurolysis to the peripheral nerve are considered to outweigh its benefits.

Table.6: NEUROLYTIC AGENTS

	Aqeous Alcohol	**Phenol**
Mechanism of action	-Cholesterol/phospholipid/ Cerebroside extraction from neural membranes -Precipitation of lipoproteins / mucoproteins	Protein coagulation/ necrosis of neural structures
Concentration used(%)	50-100	5-10
Injection profile Difficulty in injection Pain on injection	 + +	 – –
Onset/duration of block	slow / longer	slow / shorter
Plasma/Expired air concentration	+	–
Adversities Affinity for vascular structures Neuritis	 – +	 + –
Miscibility with radiocontrast media	–	+

Table.7: SIDE EFFECTS/COMPLICATIONS

Technical	Physiological
Damage to somatic nerves Paraesthesia of lumbar somatic nerve Deficit of lumbar somatic nerve	Hypotension (orthostatic) Celiac plexus ablation by neurolysis
Penetration of intervertebral foramen (if initial needle-insertion is too posterior) Dural tap Epidural injection Paraplegia	Urinary abnormality Failure of ejaculation Impotence Diarrhoea (unopposed parasympathetic activity)
Trauma to closely related great vessels Intravascular injection (venous / arterial) Vascular wall trauma Vascular thrombosis / embolism	**Others** Local pain Failure to relieve pain Shoulder tip pain / back pain Groin numbness / dysaesthesia Ethanol intoxication / seizures /loss of consciousness
Necrosis of intervening tissue Aorto-duodenal fistula	
Needle injury Intradiscal injection Renal injury Pneumothorax / chylothorax Perforation of cyst / tumors Injection of psoas muscle Retroperitoneal haematoma	**Infection** Abscess Peritonitis

Fig.10 :ULTRASONOGRAM

USG PRINCIPLES:

Based on principles of piezoelectric effect. This is defined as principle of converting electrical energy into mechanical energy. The reverse of the piezoelectric effect converts energy back into its original form.

This phenomenon was discovered by the Curie in 1880 using natural quartz.

Frequency ranges used in medical ultrasound imaging range from 2-15 MHZ.

PIEZOELECTRIC EFFECT AND ULTRASOUND TRANSDUCERS

Transducers convert one type of energy into another, depending upon pulse echo principle transducers convert

a. Electricity into sound- pulse
b. Sound into electricity- Echo

PULSE

- It is the wave sent to soft tissue
- Interaction of this sound wave with soft tissues said to be bioeffect.

- It is determined by transducers of probe crystal and is not operator controlled.

ECHO

- It is wave produced by soft tissue
- It is received back by transducer, crystals, interpreted and processed by USG machine

FREQUENCY

No. of complete cycles per unit of time.

One cycle per second= one Hertz (HZ)

TRANSDUCER FREQUENCIES

- Thin adult patient 5-10 MHz small footprint hockeystick
- Average sized adults- Linear transducer
- Obese patient curvilinear used

HIGH FREQUENCY WAVES

- Improved resolution with less depth of penetration
- Used for superficial structures

LOW FREQUENCY WAVES

- Poor resolution with full depth of penetration
- For general abdomino pelvic uses.
- Transducer frequency in USG machine is predetermined by design
- Basic sound relationships

WAVELENGTH

Distance between consecutive cycles of sound

BANDWIDTH

A range of frequencies is femoral band width

Broadband width transducers contain more than one operating freqnency.

AXIAL AND LATERAL RESOLUTION

Spatial resolution is the term which describes that two physically close objects can be displayed separately. Axial: along the beam path. Lateral: perpendicular to beampath. Normally used spatial resolution is 1mm or less.

Fig.11: TRANSDUCER

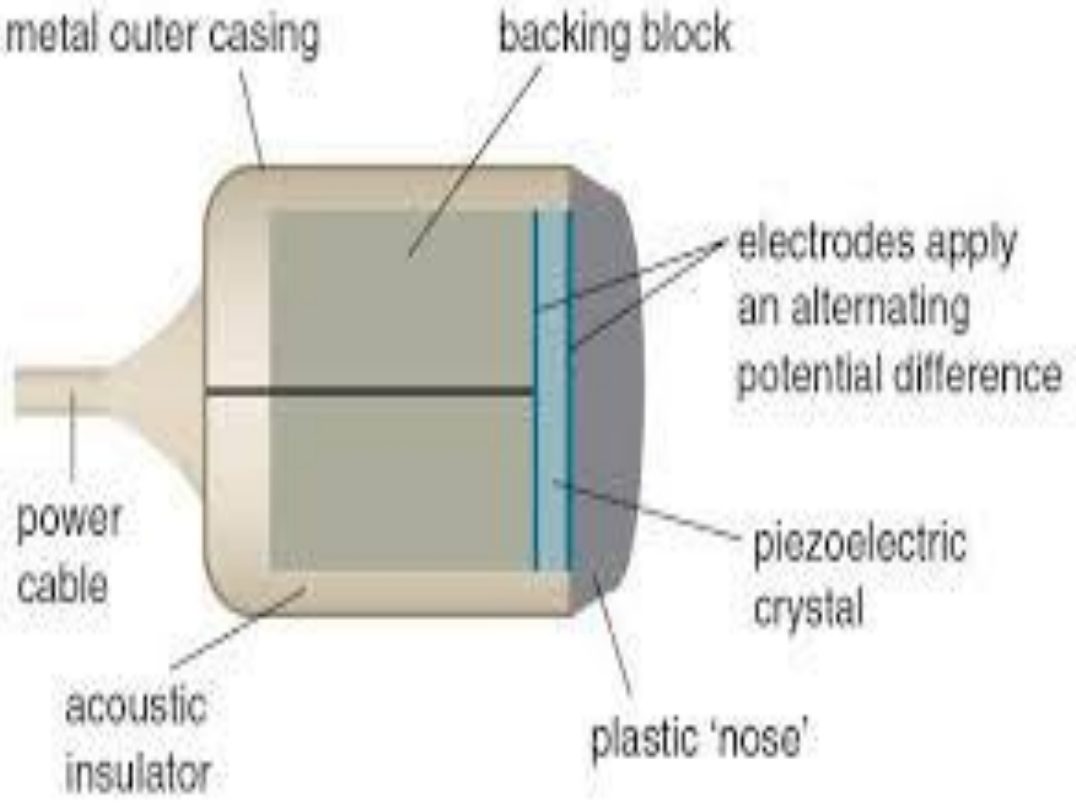
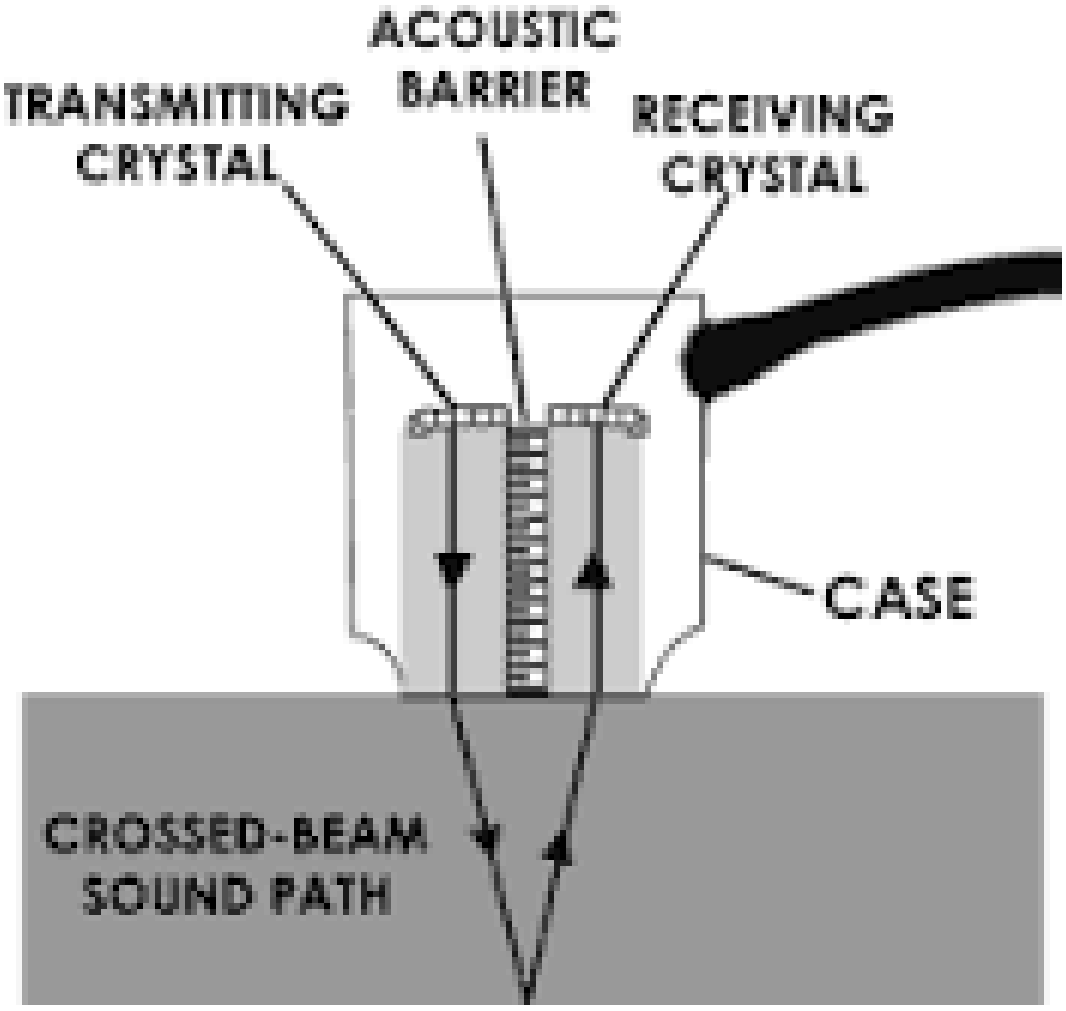


Fig.12: ULTRASOUND GENERATION



Transducers :

Types

Mechanical	Electronic
Oscillating Rotation	Linear assays Curved assays Phased assays

Receiver

Memory

Display

PROBE TYPES

Fig.13: Curvilinear (low frequency probe)

- For imaging deeper structures

Fig.14: Linear (High frequency Probe)

- For imaging superficial structures

DISPLAY MODE

B Mode- 2 dimensional

M Mode- Records moving echoes from heart in display, thus could be interpreted in terms of myocardial and valvular function.

DOPPLER

Here the frequency shift in echo is measured after a certain time.

COLOUR DOPPLER

Uses colour corresponding to frequency shift, red for near to and blue for away from the probe.

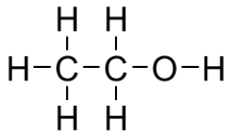
ALCOHOL

Pharmacology

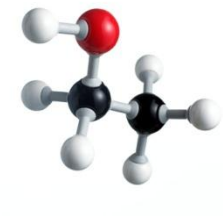
Ethanol, also called alcohol, ethyl alcohol, and drinking alcohol, is a compound and simple alcohol

C2H5OH.

Chemical Formula



STRUCTURAL FORMULA



BALL AND STICK MODEL

SPACE FILLING MODEL OF ETHANOL

Fig.15: ETHANOL

Properties

Molar mass	46.07 g·mol−1
Appearance	Colorless liquid
Density	0.7893 g/cm3 (at 20 °C)
Melting point	−114.14 ± 0.03°C
Boiling point	78.24 ± 0.09 °C
Solubility in water	miscible

Vapor pressure 5.95 kPa (at 20 °C)

Acidity (pKa) 15.9 (H_2O),

Ethanol is a volatile, flammable, colourless liquid with a slight characteristic odour. It is used as a drug and is the principal type of alcohol found in alcoholic drinks.

Ethanol is naturally produced by the fermentation of sugars by yeasts or via petrochemical processes, and is most commonly considered as a popular recreational drug.

It also has medical applications as an antiseptic and disinfectant. The compound is widely used as a chemical solvent, either for scientific chemical testing or in synthesis of other organic compounds, and is a vital substance utilized across many different kinds of manufacturing industries.

Ethanol is also used as a clean energy burning fuel source.

LIGNOCAINE

Pharmacology

Local anesthetics are used to provide analgesia and anesthesia for various surgical and nonsurgical procedures (acute and chronic pain management, reduce perioperative stress, improve perioperative outcomes, treat dysrhythmias).

Local anesthetics produce reversible conduction blockade of impulses along central and peripheral nerve pathways. With progressive increases in concentrations of local anesthetics, the transmission of autonomic, somatic sensory and somatic motor impulses is interrupted, producing autonomic nervous system blockade, sensory anesthesia, and skeletal muscle paralysis in the area innervated by the affected nerve.

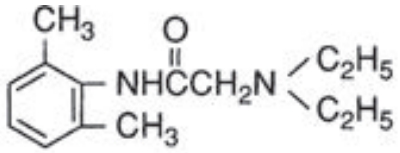
MOLECULAR STRUCTURE

A. Local anesthetics consist of a lipophilic and a hydrophilic portion separated by a connecting hydrocarbon chain.

In almost all instances, an ester (–CO–) or an amide (–NHC–) bond links the hydrocarbon chain to the lipophilic aromatic ring.

B. The nature of the connecting hydrocarbon chain is the basis for classifying drugs that produce conduction blockade of nerve impulses as *ester* local anesthetics or amide local anesthetics . The important

differences between ester and amide local anesthetics relate to the site of metabolism and the potential to produce allergic reactions.



Lidocaine

Fig.16: LIGNOCAINE

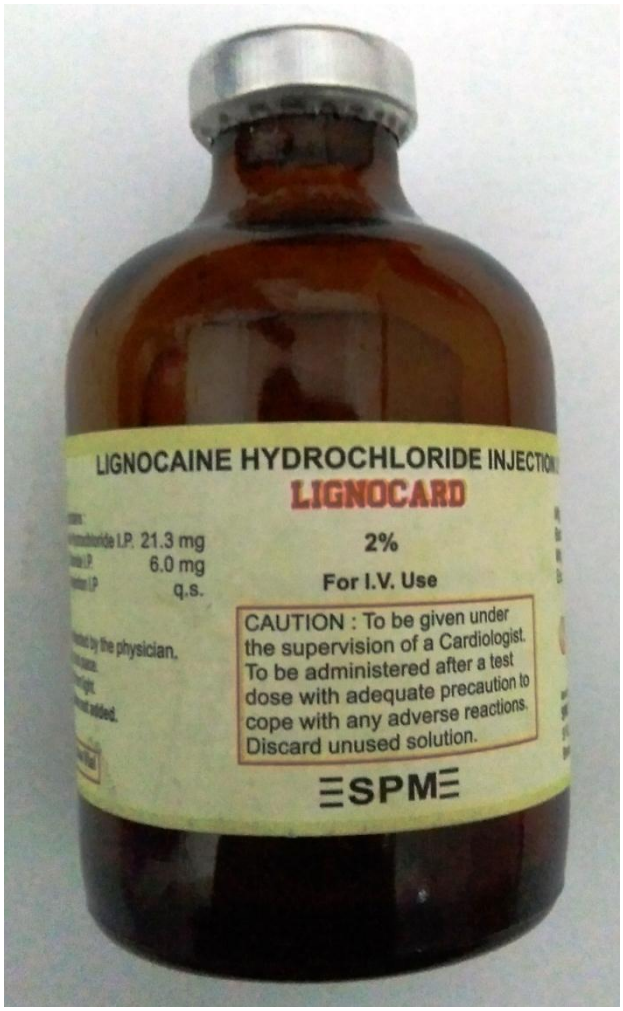


TABLE 4: FEATURES OF LIGNOCAINE

Classification	Amides
Potency	1
Onset	Rapid
Duration after Infiltration(min)	60-120
Maximum Single Dose for Infiltration(mg)	300
Toxic PlasmaConcentration(_g/mL)	>5
pK	7.9
Protein binding (%)	70
Fraction Nonionized(%) at pH 7.4	25
Fraction Nonionized(%) at pH 7.6	33
Lipid Solubility	2.9
Volume of Distribution(L)	91
Clearance(L/min)	0.95
Elimination Half-Time (min)	96

Mechanism of Action:

Local anesthetics prevent transmission of nerve impulses (conduction blockade) by inhibiting passage of sodium ions through ion-selective sodium channels(a specific receptor for local anesthetic molecules) in nerve membranes.

Failure of sodium ion channel permeability to increase slows the rate of depolarization such that threshold potential is not reached and thus an action potential is not propagated .

Absorption and Distribution:

1. Absorption of a local anesthetic from its site of injection into the systemic circulation is influenced by the site of injection and dosage, use of epinephrine, and pharmacologic characteristics of the drug .

2. The ultimate plasma concentration of a local anesthetic is determined by the rate of tissue distribution and the rate of clearance of the drug.

Lung Extraction

The lungs are capable of extracting local anesthetics (lidocaine, bupivacaine, prilocaine) from the circulation.

Renal Elimination and Clearance

1. The poor water solubility of local anesthetics usually limits renal excretion of unchanged drug to less than 5% (exception is cocaine, of which 10% to 12% of unchanged drug can be recovered in urine).

2. Water-soluble metabolites of local anesthetics, such as para-aminobenzoic acid resulting from metabolism of ester local anesthetics, are readily excreted in urine.

Metabolism :

Amide local anesthetics undergo varying rates of metabolism by microsomal enzymes located primarily in the liver.

Lidocaine is intermediate.

Compared with that of ester local anesthetics, the metabolism of amide local anesthetics is more complex and slower. This slower metabolism means that sustained increases of the plasma concentrations of amide local anesthetics, and thus systemic toxicity, are more likely than with ester local anesthetics. Furthermore, cumulative drug effects of amide local anesthetics are more likely than with ester local anesthetics.

The principal metabolic pathway of lidocaine is oxidative dealkylation in the liver to monoethylglycinexylidide (approximately

80% of the activity of lidocaine for protecting against cardiac dysrhythmias) followed by hydrolysis of this metabolite to xylidide.

Xylidide has only approximately 10% of the cardiac antidysrhythmic activity of lidocaine.

Table.5: Dose-Dependent Effects of Lidocaine

Plasma Lidocaine Concentration (_µg/mL)	**Effects**
1–5	Analgesia
5–10	Circumoral numbness Tinnitus Skeletal muscle twitching Systemic hypotension Myocardial depression
10–15	Seizures Unconsciousness
15–25	Apnea Coma
>25	Cardiovascular depression

REVIEW OF LITERATURE

1. P.N.JAIN et.al had undertaken a study to evaluate the role of neurolytic celiac plexus block and oral morphine on pain and quality of life in patiens with upper abdominal malignancies.NCPB provided statistically significant(P=0.000) better pain relief and reduced morphine consumption in 100 consecutive patients.Patients with oral morphine had more side effects(94%vs58%)compared to NCPB

2. Donald F.Romenelli ,Carl F.Beckmann,Fredrick W.Heiss et.al performed celiac plexus block via anterior approach in 17 consecutive patients and found better than posterior approach which had shorter procedure time,less discomfort to the patients,and less risk of neurological complications.

3. Gilbert Y.Wong,Darrell R.Schroeder et .al conducted a randomised controlled trial for 4 years from October 1997 to January for 100 patients and found although NCPB improves pain relief it does not affect quality of life or survival rate.

4. Mikito Kawamata et.al evaluated 21 patients with pancreatic cancer the effectiveness of NCPB compared to the traditional NSAID –morphine treatment and found NCPB does not directly improve QOL but it may

prevent deterioration in QOL by the long lasting analgesic effect,limitation of side effects and decrease in morphine consumption .

5. Byambasuren Yondonjamts et al studied 56 patients with advanced abdominal cancers taking oral morphine and performed NCPB and found NCPB had significantly reduced pain intensity opiod consumption and improved quality of life.

6. Mariam Hameed et al performed a metaanalysis of 8 RCTs and found various techniques ,timing ,intensity of pain,efficacy of the celiac plexus block,anatomical location of disease and injectate spread all influence in the outcome of patients with upper abdominal malignancies. And also found NCPB as a relatively safe procedure. Most common adverse effects reported are local pain(96%),diarrhoea(44%),and hypotension(10%).

7. E.Polati et.al reviewed the various techniques classic retrocrural technique, bilateral splanchinectomy, transintervertebral disc approach, echoendoscopic guidance and concluded that NCPB is an effective and safe tool but could not establish which kind of radiologic guidance is the best to reach the target and avoid major neurologic complications.

8. Sankalp Sehgal and Ahmed Ghaleb reviewed the treatment of cancer pain and advances in techniques of performing NCPB. And also analysed the incidence of complications and quality of pain relief . Concluded that

NCPB is effective low incidence of complications and should be used often in patients with pancreatic cancers.

9. Hrachya Nersesyan and Konstantin V Slavin reviewed about the different treatment options available for treating cancer pain focusing mainly on pharmacological therapy,invasive modalities for pain control,side effects and ways to increase the effectiveness of treatment and its compliance.

MATERIAL AND METHODS

60 patients ASA gradeI, II, III of both sexes aged 80 years or less (range 18-80 years) were included in study. After getting ethical committee clearance, study was conducted. Informed written consent was obtained from patients included in the study.

STUDY DESIGN

The study was prospective, randomized, comparative study.60 patients with complaints of chronic upper abdominal pain, to whom surgery was not possible due to inoperability

SELECTION OF CASES

Inclusion Criteria

1)Patients of age 18 years to 80 years.

2)ASA I, II,III

3)Chronic abdominal pain due to cancer

4)Refractory to analgesics

5)Who had given valid informed consent.

Exclusion criteria

1)Not satisfying inclusion criteria

2)Patient refusal

3)Cases with history of surgery

4)Cases with severe disability

5)Allergy to local anaesthetics ,alcohol, bleeding diathesis

6)Patient with severe cardiovascular, respiratory, renal and hepatic diseases.

MATERIALS REQUIRED

1)18 G Venflon needle

2) USG machine

3)10cm 3 way extension catheter

4)10ml, 5ml, 2ml syringes

DRUGS

1)2% xylocard preservative free

2)50% ethyl alcohol

3)Emergency drugs

4)Distilled water

MONITORS

NIBP, HR, SpO2, 5 LEAD ECG

OUTCOMES MEASURED

1.to measure the efficacy of USG guided neurolytic celiac plexus block via anterior approach

2.to assess the analgesic effect of 50% alcohol

3.to assess and compare the side effects of NCPB and morphine

4.to assess the complications

METHODOLOGY

Ethical committee approval

↓

Patient satisfying inclusion criteria

↓

Informed consent obtained

↓

Ramdomization by closed envelope Method

↓

Group OM –Patients on oral morphine

Group NCPB –Patients undergoing neurolytic celiac plexus block

↓

Routine labarotory investigations done

[CBC,PT,INR,RBS]

↓

Peripheral venous line accessed

↓

NIBP,ECG,SpO2 Probe measurement done

↓

↓

Cleaning of abdomen and draping done under strict aseptic precautions in supine position

↓

Under USG guidance over the epigastric region aorta visualised and celiac plexus visualised

Local skin infiltration was done using 3 ml of inj.2% xylocaine .After 3 minutes 18G 9cm venflon needle was inserted under USG guidance ,position of needle confirmed near celiac plexus and inj.1%xylocard 10 ml injected around the plexus after negative aspiration.Then 10 ml 50% ethyl alcohol is injected around the celiac plexus after negative aspiration and finally needle was flushed with 5 ml of 1%xylocard

↓

Patient was observed for changes in HR,BP,SpO2,pain scores

↓

Patient was observed in post anaesthesia care unit for 24 hrs

↓

Data compilation

↓

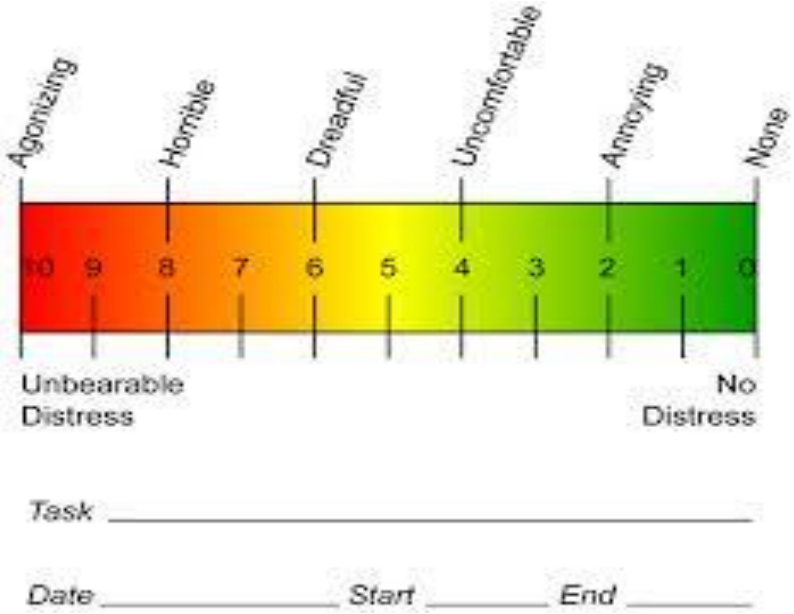
Statistical analysis

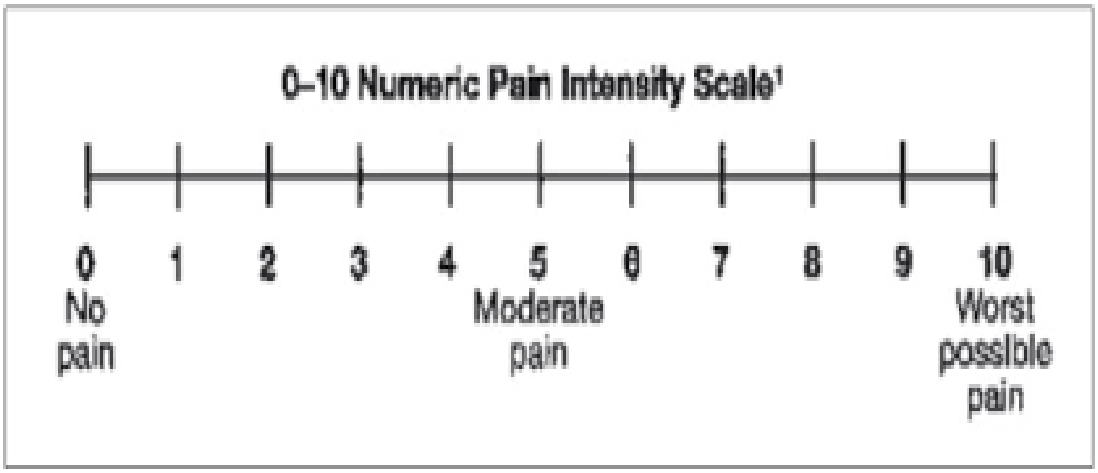
↓

Conclusion

OBSERVATION AND INTERPRETATION

Fig.17: ASSESSMENT OF PAIN USING VISUAL ANALOGUE SCALE





PERFORMANCE STATUS

SCORE

0	no symptoms , normal life
1	able to carry on normal activities but has returned to parttime or less strenuous work
2	unable to work but is able to cre for personal needs
3	limited in care for self
4	unable to care for self and confined to bed

SIDE EFFECTS

NAUSEA AND VOMITING, CONSTIPATION, TRANSIENT DIARRHOEA

SCORE	
1	No symptoms
2	Moderate
3	Severe and tolerable
4	Severe and intolerable

RESULTS

The collected data were analysed with IBM.SPSS statistics software 23.0 Version. To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups the Unpaired sample t-test and the Mann-Whitney U test was used. To find the significance in categorical data Chi-Square test was used. In all the above statistical tools the probability value .05 is considered as significant level.

P - Value	** Highly Significant at P ≤ .01

P -Value	# No Significant at P >.05

TABLE.8:. DEMOGRAPHIC DATA

AGE	CPB (n=30)	MORPHINE (n= 30)
MEAN ±SD	57.3 ± 8.59	61.2 ± 7.87

TABLE.9:GENDER

		GROUPS		Total
		CCPB	**OM**	
F	count	**7**	**8**	**15**
	%within groups	**23.3%**	**26.7%**	**25.0%**
M	count	**23**	**22**	**45**
	%within groups	**76.7%**	**73.3%**	**75.0%**

The mean age of the study participants was 57.3 years and 61.2 years .

25% (n=15)were females and 75%(n=45) were males.

Fig.18: GENDER

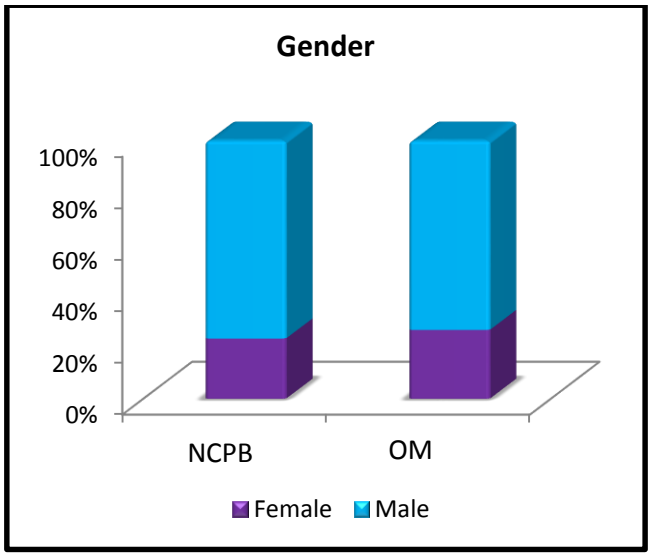


TABLE.10: ASA/PS CLASSIFICATION

		GROUPS		Total
		CCPB	**OM**	
III	Count	**30**	**16**	**46**
	% Within groups	**100.0%**	**53.3%**	**76.7%**
IV	Count	**0**	**14**	**14**
	% Within groups	**0.0%**	**46.7%**	**23.3%**

76.7% belonged to ASA/PS III and 23.3% belonged to ASA/PS IV categories

Fig.19: ASA/PS COMPARISON

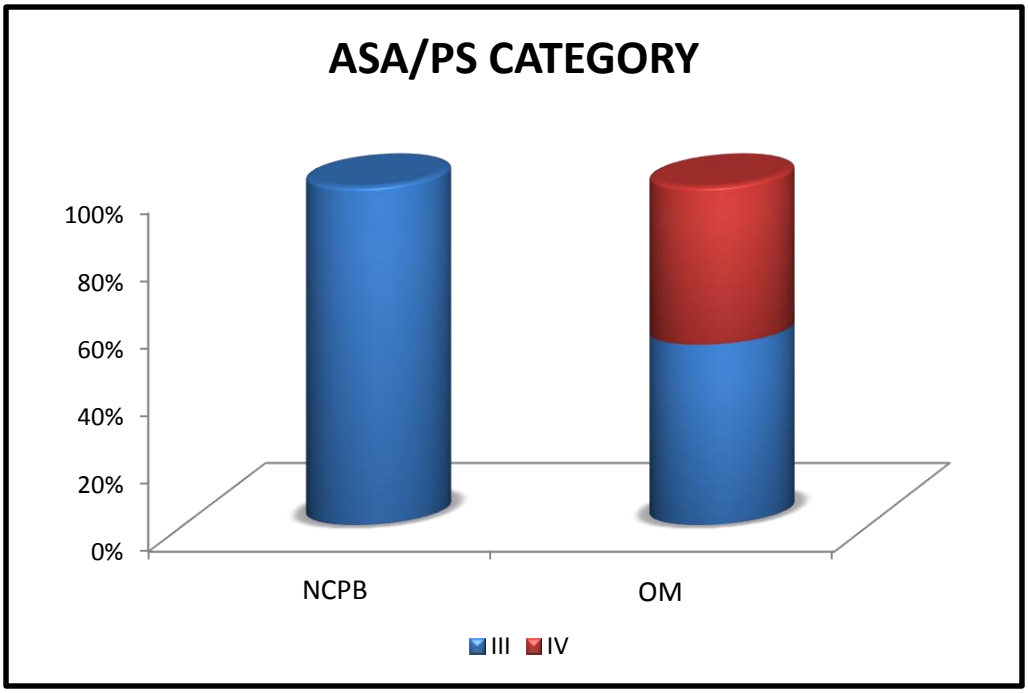


FIG.20: HEMODYNAMICS DURING AND AFTER PROCEDURE

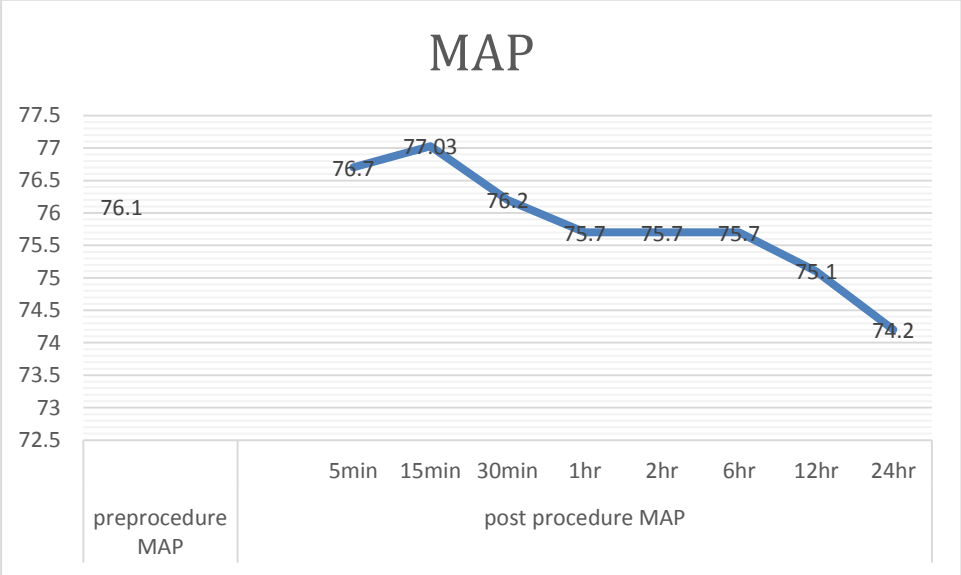


Fig.21: Heart rate

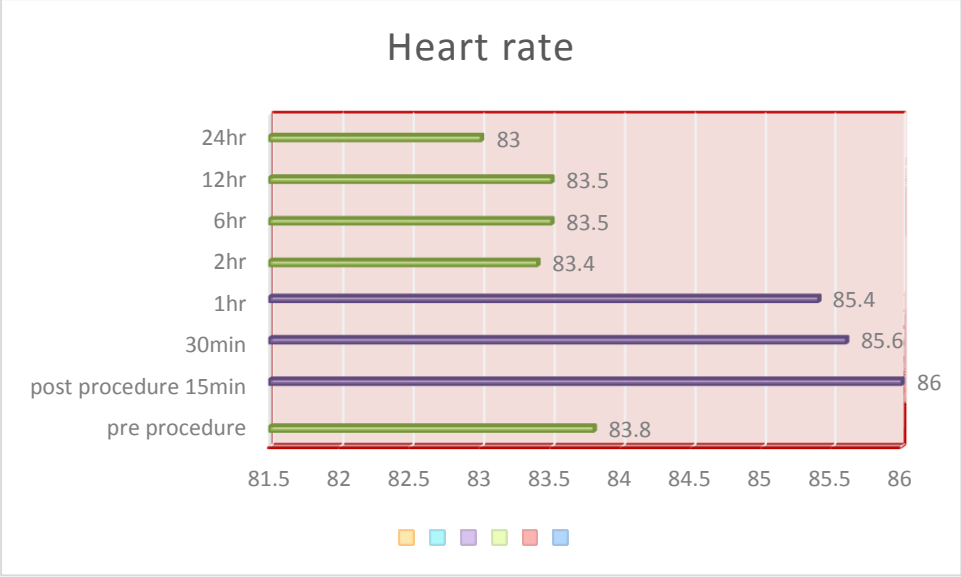


Fig.22: TRENDS IN VAS SCORE OF NCPB

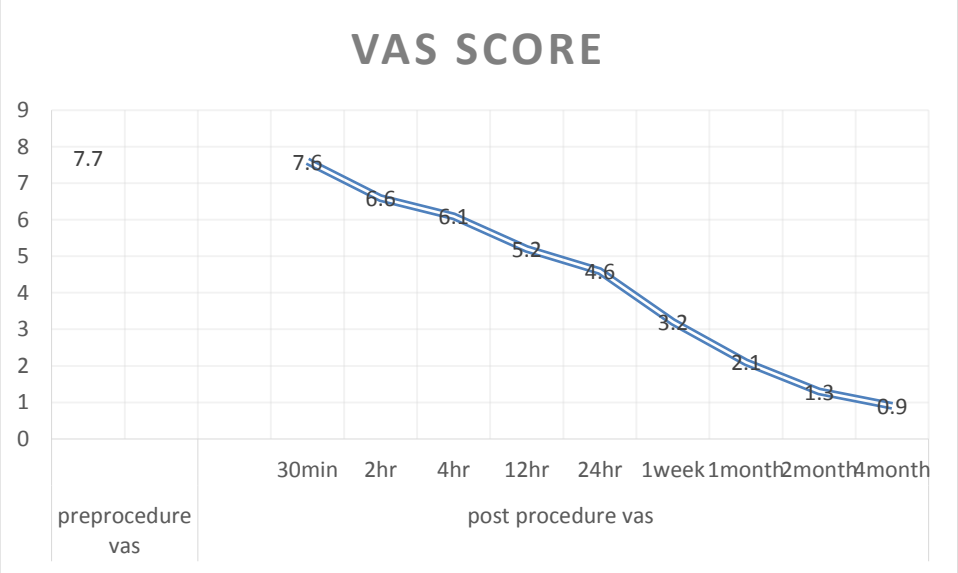


Table.11:Mann-WhitneyTest

GROUPS		N	Mean Rank	Sum of Ranks
VAS 1WK	NCPB	30	16.02	480.50
	OM	30	44.98	1349.50
	Total	60		
VAS 1MTH	NCPB	30	15.90	477.00
	OM	30	45.10	1353.00
	Total	60		
VAS 2MTH	NCPB	30	15.80	474.00
	OM	30	45.20	1356.00
	Total	60		
VAS 4MTH	NCPB	30	16.35	490.50
	OM	30	44.65	1339.50
	Total	60		

Table.12: COMPARISON OF MEAN VAS scores

GROUPS		N	Mean	Std. Deviation
VAS 1 wk	NCPB	30	3.27	1.112
	OM	30	7.07	1.143
VAS 1 mth	NCPB	30	2.13	1.167
	OM	30	6.33	1.295
VAS 2 mth	NCPB	30	1.33	1.213
	OM	30	5.60	1.221
VAS 4 mth	NCPB	30	.93	1.143
	OM	30	5.27	1.701

The mean VAS scores after NCPB decreased from 3.27 at 1st week to .93 at 4 months, the mean VAS scores in oral morphine group decreased from 7.07 at 1st week to 5.27 at 4 months. There was a great reduction in VAS scores in NCPB group compared to oral morphine group.

Table.13:VAS SCORES P value

	Mann-Whitney U	Asymp. Sig. (2-tailed)
VAS 1 WK	15.500	.0005
VAS 1 MTH	12.000	.0005
VAS 2 MTH	9.000	.0005
VAS 4 MTH	25.500	.0005

There was a significant difference **(p value .0005)** between VAS scores of two groups

Fig.23: VAS SCORE

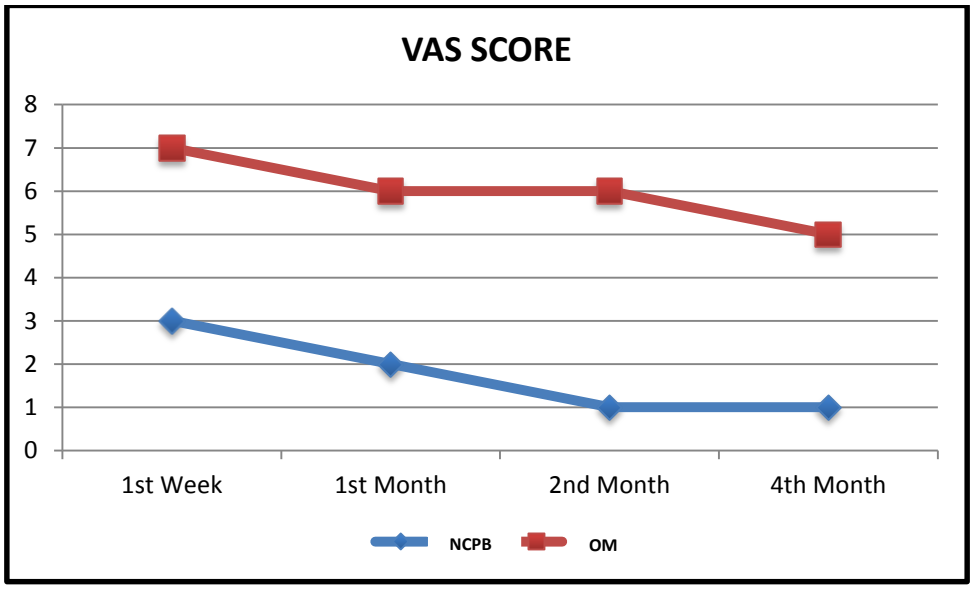


Table.14:PERFORMANCE STATUS BEFORE NCPB

		GROUPS		Total
		NCPB	OM	
1	Count	1	0	1
	% Within groups	3.3%	0.0%	1.7%
2	Count	10	7	17
	% Within groups	33.3%	23.3%	28.3%
3	Count	13	20	33
	% Within groups	43.3%	66.7%	55.0%
4	Count	6	3	9
	% Within groups	20.0%	10.0%	15.0%

Table.14.1 Chi-Square test

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.014[a]	3	.260

There was no significant difference**(p=.260)** between two groups in performance status before NCPB

TABLE.15: PERFORMANCESTATUS AFTER 4 MTHS

		GROUPS		Total
		NCPB	OM	
0	Count	1	0	1
	% within GROUPS	3.3%	0.0%	1.7%
1	Count	11	0	11
	% within GROUPS	36.7%	0.0%	18.3%
2	Count	14	13	27
	% within GROUPS	46.7%	43.3%	45.0%
3	Count	4	16	20
	% within GROUPS	13.3%	53.3%	33.3%
4	Count	0	1	1
	% within GROUPS	0.0%	3.3%	1.7%

Table.15.1 Chi-Square test

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	20.237[a]	4	.0005

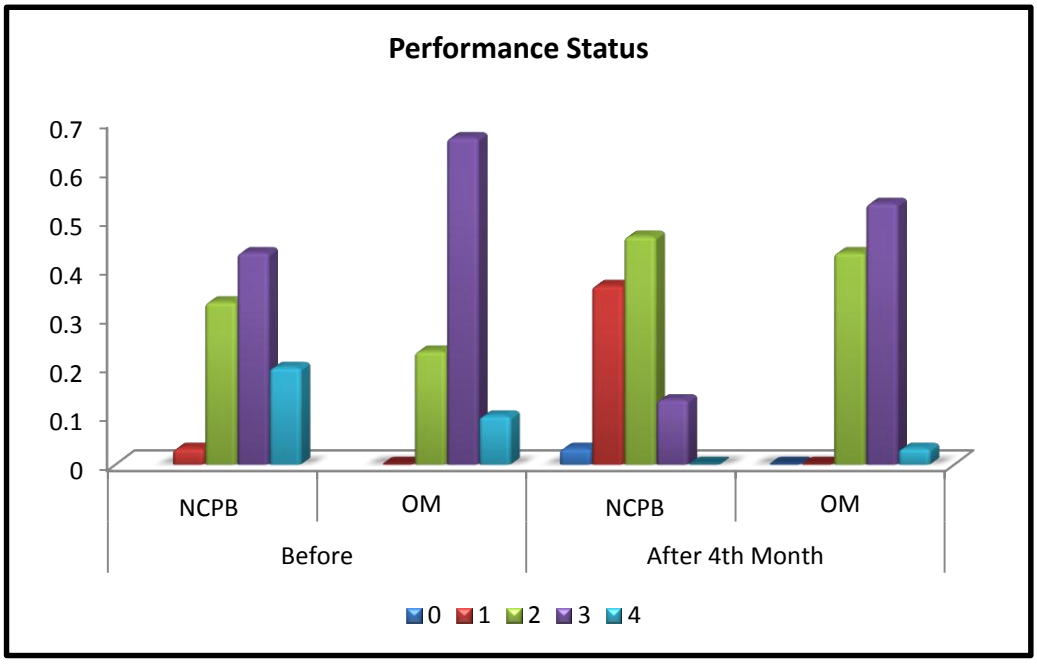
There was a statistically significant difference **(p=.0005)** between two groups after NCPB

Table.16: PERFORMANCE STATUS

SCORE	Before		After 4th Month	
	NCPB	OM	NCPB	OM
0			3.3%	0.0%
1	3.3%	0.0%	36.7%	0.0%
2	33.3%	23.3%	46.7%	43.3%
3	43.3%	66.7%	13.3%	53.3%
4	20.0%	10.0%	0.0%	3.3%

The overall performance status after 4 months improved gradually in patients who had undergone NCPB than who took oral morphine.

FIG.24: PERFORMANCE STATUS



SIDE EFFECTS

Table.17:CONSTIPATION

			GROUPS		Total
			NCPB	OM	
	No symptoms	Count	30	2	32
		% within GROUPS	100.0%	6.7%	53.3%
	Moderate	Count	0	22	22
		% within GROUPS	0.0%	73.3%	36.7%
	Severe & Tolerable	Count	0	6	6
		% within GROUPS	0.0%	20.0%	10.0%

Table.17.1 Chi-Square test

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	**52.500[a]**	**2**	**.0005**

There was a significant difference**(p value .0005)** between two groups.Patients with NCPB had no constipation and those were on oral morphine 73.3% had moderate constipation and 20% had severe constipation and 6.7% had no constipation.

Fig.25: CONSTIPATION

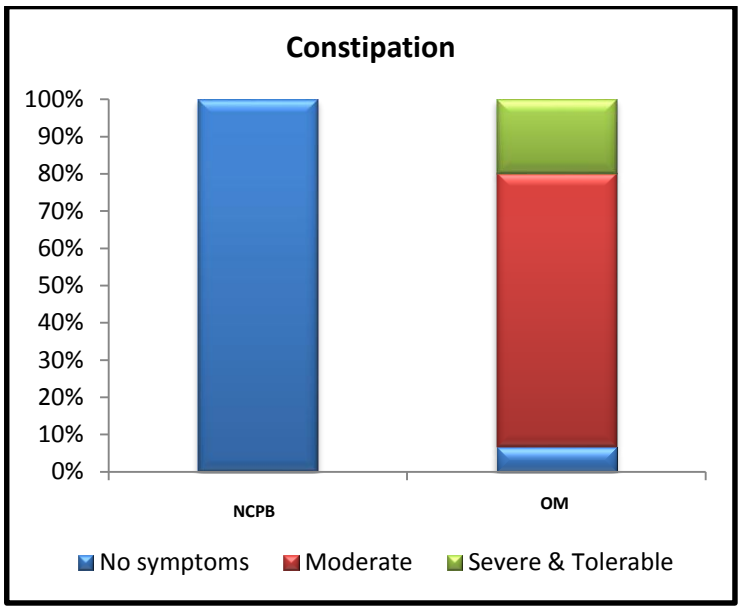


Table.18: NAUSEA AND VOMITTING

		GROUPS		Total
		NCPB	OM	
No symptoms	count	10	9	**19**
	% within groups	33.3%	30.0%	**31.7%**
Moderate	count	17	16	**33**
	% within groups	56.7%	53.3%	**55.0%**
Severe & Tolerable	count	2	5	**7**
	% within groups	6.7%	16.7%	**11.7%**
Severe & Intolerable	count	1	0	**1**
	% within groups	3.3%	0.0%	**1.7%**

Table.18.1:Chi-Square Test

	Value	**Asymp. Sig. (2-sided)**
Pearson Chi-Square	**2.369[a]**	**.499**

There is no significant difference **(p value .499)** between two groups .Both had nausea and vomiting .But patients on oral morphine16.7% had severe and tolerable nausea and vomiting compared to 6.7% in NCPB group.

Fig.26: NAUSEA & VOMITING

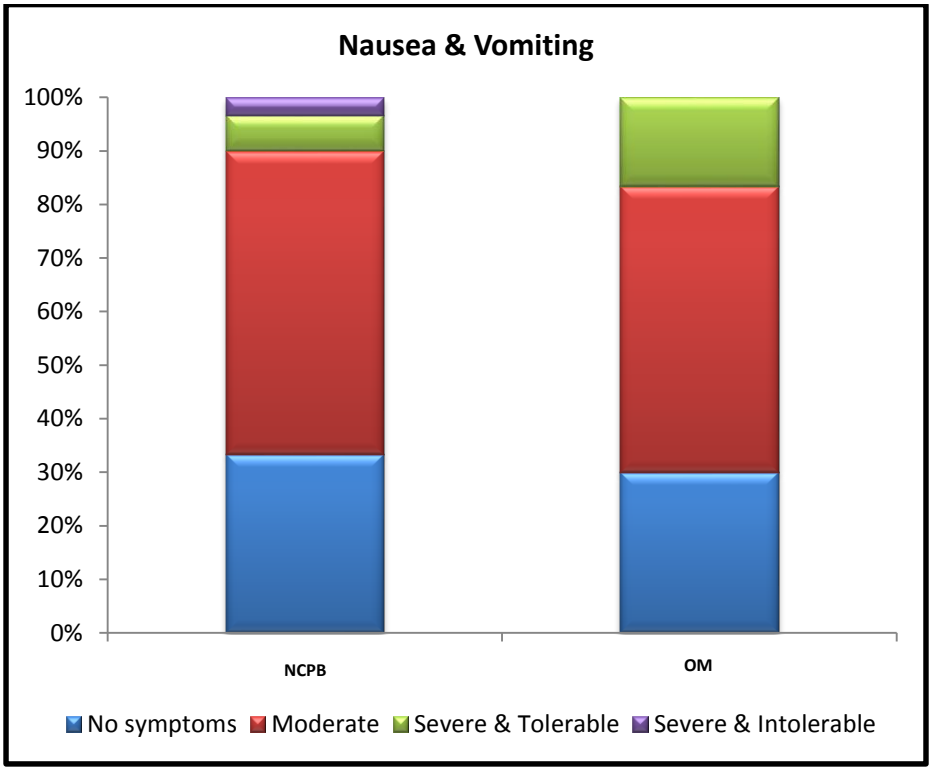


Table.19: TRANSIENT DIARRHOEA

		GROUPS		Total
		CCPB	OM	
No symptoms	Count	7	30	37
	%within GROUPS	23.3%	100.0%	61.7%
Moderate	Count	12	0	12
	%within GROUPS	40.0%	0.0%	20.0%
Severe & Tolerable	Count	8	0	8
	%within GROUPS	26.7%	0.0%	13.3%
Severe & Intolerable	Count	3	0	3
	% within GROUPS	10.0%	0.0%	5.0%

TABLE.19.1: Chi-Square test

	Value	Asymp. Sig. (2-sided)
Pearson Chi-Square	**37.297[a]**	**.0005**

There was a significant difference (p value .0005) between two groups.

Patients who had undergone NCPB had transient diarrhoea.40% had moderate diarrhoea, 26.7% had severe diarrhoea, 23.3% had no symptoms and 10% had severe intolerable diarrhoea.

Patients on oral morphine had no symptoms of transient diarrhoea.

Fig.27: TRANSIENT DIARRHOEA

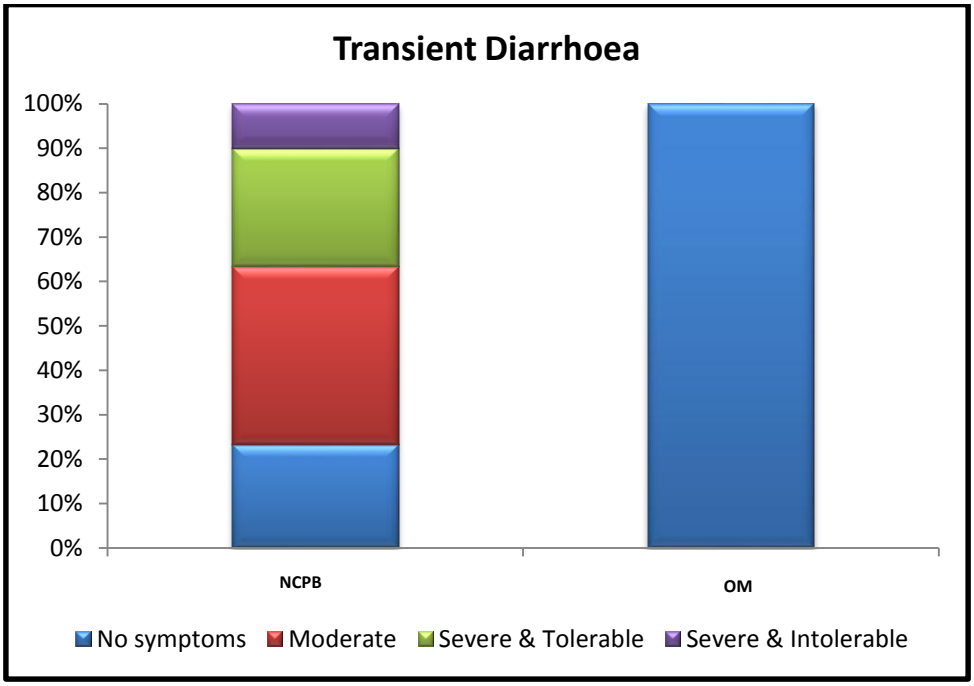


Table.20:MORPHINE CONSUMPTION (mg/day)

GROUPS		N	Mean	Std. Deviation	Std. Error Mean
MC 1WK	NCPB	30	31.00	8.030	1.466
	OM	30	22.67	4.498	.821
MC 1MTH	NCPB	30	22.33	6.261	1.143
	OM	30	31.00	4.807	.878
MC 2MTH	NCPB	30	13.83	6.909	1.261
	OM	30	36.00	4.983	.910
MC 4MTH	NCPB	30	8.00	7.611	1.390
	OM	30	38.33	3.790	.692

The mean morphine consumption per day in NCPB group decreased from 31.00 at 1st week to 8.00 at 4 months.But the mean consumption of morphine per day in Oral morphine group increased from 22.67 at 1st week to 38.33 at 4 months.

Table.20.1:MORPHINE CONSUMPTION p value

	t-test for Equality of Means			
	t	**Sig. (2-tailed)**	**Mean Difference**	**Std. Error Difference**
MC 1st wk	**4.959**	**.0005**	**8.333**	**1.680**
MC 1st mth	**-6.014**	**.0005**	**-8.667**	**1.441**
MC 2nd mth	**-14.253**	**.0005**	**-22.167**	**1.555**
MC 4th mth	**-19.540**	**.0005**	**-30.333**	**1.552**

There is a significant difference **(p value .0005)** in morphine consumption between two groups

Fig.28:Morphine consumption

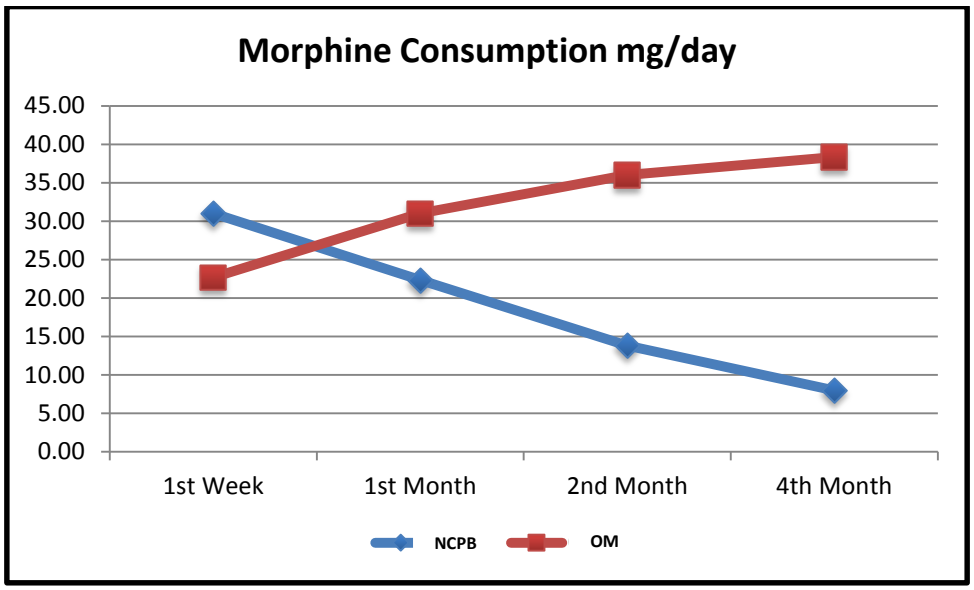


Table.21:DIAGNOSES BY CANCER TYPES

CANCER TYPE	NCPB n=30 (%)	MORPHINE n=30 (%)
Ca Pancreas	10 (33.33)	9 (30.0)
Ca Stomach	5 (16.66)	10 (33.33)
Ca Gall Bladder	5 (16.66)	4 (13.33)
Ca Trans Colon	3 (10.0)	3 (10.0)
Peri Amp Ca	2 (6.66)	2 (6.66)
Ca Lower 1/3 Esophagus	3 (10.0)	2 (6.66)
Ca Stomach With Mets	1 (3.33)	0 (0)
Gist Mets	1 (3.33)	0 (0)

Overall Ca pancreas and Ca stomach were more prevalent in both groups than other malignancies**.**

DISCUSSION

Pain is the predominant symptom in 80-85% of the patients with advanced stages of disease. Adequate pain management and improving quality of life is of utmost importance. This is to enable the patients to live as productively as possible in the stage of impending death.

In this study group, all patients suffered a continuous chronic visceral type of pain of VAS scores ranging from 9-6 deep in the right hypochondrium or epigastrium radiating to the back. It is a well known fact that pain strongly influences the quality of life in advanced stage of cancer.

Since long back, numerous studies tried to evaluate the efficacy of NCPB and all studies showed that there was significant improvement in pain relief and showed reduced side effects and decreased opioid consumption. Extensive study of literatures revealed only a few randomised studies assessing the efficacy of anterior approach by USG.

This study demonstrates that USG guided neurolytic CPB by anterior approach improves pain relief and reduces morphine consumption and its side effects. There was a significant improvement in performance status after NCPB compared to morphine groups.Patients in

NCPB groups had transient diarrhoea which resolved spontaneously after a few days.

We assessed the efficacy of neurolytic celiac plexus block for 30 patients and they responded well to the procedure. Recovery from the pain was evaluated by VAS score and was observed from 30 minutes following the procedure. The maximum pain relief was reported during the 1st week itself and gradual decrease from there onwards. Our results supported the evidence of long term pain relief upto 4 months compared to morphine which required increase in dose of morphine within one month and associated with increase in side effects.

No complications/accidental aortic puncture occurred in our study.

In our study, the advantages of USG assisted anterior approach neurolysis over fluoroscopic guided (posterior approach) are as follows: patient comfort in supine position, less time consuming, less vascular injury and no radiation exposure. Following injection, patients are sent home early thereby frequent visit to hospital for getting morphine tablets is reduced. Moreover many patients became ambulatory and could resume their daily activities without the help of others.

CONCLUSION

In advanced stage of malignancy neurolytic celiac plexus block is more beneficial, with decrease in dosage of oral morphine and its side effects compared to oral intake of morphine alone .

Life threatening complications like the accidental intravascular injection, long term neurological deficits, loss of bladder and bowel control, its potential risks and benefits should be considered in context of a suffering cancer patient with poor prognosis.

So patients who are at the end stage of disease having intractable pain can be benefited from this technique.

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INSTITUTIONAL ETHICS COMMITTEE
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EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

MADRAS MEDICAL COLLEGE
15 MAR 2017
CHENNAI-600 003

CERTIFICATE OF APPROVAL

To
Dr.Anbarasan.B
II Year Post Graduate in MD Anaesthesiology
Institute of Anaesthesiology & Critical Care
Madras Medical College
Chennai 600 003

Dear Dr.Anbarasan.B,

The Institutional Ethics Committee has considered your request and approved your study titled "**COMPARISON OF ORAL MORPHINE VS CELIAC PLEXUS BLOCK ON PAIN RELIEF IN PATIENTS WITH ABDOMINAL MALIGNANCIES" - NO.14022017 (II)**

The following members of Ethics Committee were present in the meeting hold on **21.02.2017** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD., Prof.of Pharmacology.,MMC,Ch-3	: Member
5.Prof.K.Ramadevi,MD.,Director,Inst.of Bio-Che,MMC,Ch-3	: Member
6.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
7.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
8.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

PROFORMA

DATE: ROLL NO:

NAME:

AGE: SEX:

IP NO:

DIAGNOSIS:

PROCEDURE DONE:

Ht: CVS:

HB:

Wt: RS:

AIRWAY:MMS - IID -

DENTITION -

PRE OP ASSESSMENT:

HISTORY: Any Co-morbid illness

H/O Documented Difficult Airway

H/O previous surgeries

MEASURES OF STUDY OUTCOME:

COMPLICATIONS DURING PROCEDURE:

COMPLICATIONS AFTER DRUG INTAKE /NEUROLYSIS:

INFORMATION TO PARTICIPANTS

Investigator **:** Dr. ANBARASAN B

Name of the Participant:

Title. Comparison of Oral morphine and celiac plexus block on pain relief in patients with abdominal malignancies (A Randomized comparative study)

You are invited to take part in this research study. We have got approval from the IEC. You are asked to participate because you satisfy the eligibility criteria. We want to compare and study the efficacy and safety of oral morphine vs celiac plexus block

What is the Purpose of the Research:

For abdominal malignancies with intractable pain , analgesia using oral morphine and celiac plexus block were compared

1. To compare the duration of analgesia.
2. To assess side effects
3. To asses Complications.
4. To evaluate performance status

The Study Design:

All the patients in the study will be divided into two groups.

Group OM- oral medication with morphine
Group NCPB- Celiac plexus block with alcohol via anterior approach under USG guidance

Benefits:

Neurolytic celiac plexus block reduces opioid requirement, side effects of morphine usage and provides long term pain relief.

Discomforts and risks

Intravascular alcohol injection

Damage to neuro vascular structure

This intervention has been shown to be well tolerated as shown by previous studies. And if you do not want to participate you will have alternative of setting the standard treatment and your safety is our prime concern.

Date :
Place :

Signature / Thumb
Impression of Patient
Patient Name:

Signature of the Investigator : _____

Name of the Investigator : _____

PATIENT CONSENT FORM

Study title : Comparison of Oral morphine and celiac plexus block on pain relief in patients with abdominal malignancies
(A Randomized comparative clinical study)

Study center: **INSTITUTE OF ANAESTHESIOLOGY AND CRITICAL CARE,**

RAJIV GANDHI GOVT. GENERAL HOSPITAL,

MADRAS MEDICAL COLLEGE,

CHENNAI-0 3.

Participant name: Age: Sex: I.P.No:

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the pitfall in the procedure. I have been explained about the safety, advantage and disadvantage of the technique.

I understand that my participation in the study is voluntary and that I am free to withdraw at anytime without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

Time:

Date: Signature / thumb impression of patient

Place: Patient name:

Signature of the investigator:

Name of the investigator:

சுய ஒப்புதல் படிவம்

ஆய்வு தலைப்பு :

பெயர் : வயது : தேதி : வெளிநோயாளி எண்:

..... என்பவராகிய நான் இந்த ஆய்வின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக அறிந்து கொண்டேன். எனது சந்தேங்கள் அனைத்திற்கும் தகுந்த விளக்கம் அளிக்கப்பட்டது. இந்த ஆய்வில் முழு சுதந்திரத்துடன் மற்றும் சுயநினைவுடன் பங்கு கொள்ள சம்மதிக்கிறேன்.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தெரிவிக்கிறேன். இச் சுய ஒப்புதல் படிவத்தை பற்றி எனக்கு விளக்கப்பட்டது.

இந்த ஆய்வினை பற்றிய அனைத்து தகவல்களும் எனக்கு தெரிவிக்கப்பட்டது. இந்த ஆய்வில் எனது உரிமை மற்றும் பங்கினை பற்றி அறிந்து கொண்டேன்.

இந்த ஆய்வில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

இந்த ஆய்வில் கலந்து கொள்வதன் மூலம் என்னிடம் பெறப்படும் தகவலை ஆய்வாளர் இன்ஸ்டிட்யூசனல் எத்திக்ஸ் கமிட்டியினரிடமோ, அரசு நிறுவனத்திடமோ தேவைப்பட்டால் பகிர்ந்து கொள்ளலாம் என் சம்மதிக்கிறேன்.

இந்த ஆய்வின் முடிவுகளை வெளியிடும்போது எனது பெயரோ, அடையாளமோ வெளியிடப்படாது என அறிந்து கொண்டேன். இந்த ஆய்வின் விவரங்களைக் கொண்ட தகவல் தாளைப் பெற்றுக் கொண்டேன். இந்த ஆய்விற்காக இரத்தப் பரிசோதனை செய்துகொள்ள சம்மதம் தெரிவிக்கின்றேன்.

இந்த ஆய்வில் பங்கேற்கும் பொழுது ஏதேனும் சந்தேகம் ஏற்பட்டால், உடனே ஆய்வாளரை தொடர்பு கொள்ள வேண்டும் என அறிந்து கொண்டேன்.

இச்சுய ஒப்புதல் படிவத்தில் கையெழுத்திடுவதன் மூலம் இதிலுள்ள அனைத்து விஷயங்களும் எனக்கு தெளிவாக விளக்கப்பட்டது என்று தெரிவிக்கிறேன் என்று புரிந்து கொண்டேன். இச்சுய ஒப்புதல் படிவத்தின் ஒரு நகல் எனக்கு கொடுக்கப்படும் என்று தெரிந்து கொண்டேன்.

பங்கேற்பாளர் /பாதுகாவலர் கையொப்பம் தேதி :

ஆய்வாளர் கையொப்பம் தேதி :

HEMODYNAMCS

EVENTS	MAP	HEART RATE	SpO2
BASE LINE			
DURING PROCEDURE			
AFTER PROCEDURE			

TIME	0min	5min	15min	30min	1 hr	2hr	6hr	12hr	24hr
HR									
MAP									
SpO2									

POST PROCEDURE

TIME	30min	2hr	4hr	12hr	24hr	1wk	1mth	2mth	4mth
VAS score									

GROUP ORAL MORPHINE																				
SL.NO.	NAME	AGE	SEX	IP NO.	DIAGNOSIS	ASA/ PS	VAS SCORE				MORPHINE CONSUMPTION MG/DAY				QOL		PERFORMANCE STATUS		SIDE EFFECTS		
							1 WK	1 MTH	2 MTH	4 MTH	1 WK	1 MTH	2 MTH	4 MTH	BEFORE	AFTER4MTH	BEFORE	AFTER 4 MTH	CONSTIPATION	NAUSEA &VOMITING	TRANSIENT DIARRHOEA
1	KAMARAJ	63	M	103/17	CA PANCREAS	III	6	6	4	6	30	40	40	40	0	0	3	3	2	2	1
2	PERIYASAMY	67	M	2601/16	CA GALL BLADDER	III	8	6	6	8	20	30	40	40	0	0	3	2	3	2	1
3	PADMANABAN	56	M	395/16	CA GALL BLADDER	IV	6	6	8	6	20	30	40	40	0	0	3	2	2	1	1
4	LALITHA	50	F	673/16	CA STOMACH	IV	8	6	6	4	20	30	30	30	0	0	3	3	2	2	1
5	SUNDARAM	71	M	872/16	CA STOMACH	III	4	4	4	6	20	20	40	40	0	0	2	2	3	2	1
6	SARANGAPANI	53	M	540/16	CA LOWER 1/3 ESOPHAGUS	IV	8	8	6	6	20	30	30	40	0	0	2	2	2	1	1
7	NAGAPPAN	75	M	671/16	CA PANCREAS	III	8	6	6	8	20	30	30	40	0	0	3	2	3	2	1
8	SRINIVASAN	51	M	651/16	CA TRANSVERSE COLON	IV	6	6	8	8	20	30	30	40	0	0	2	2	2	3	1
9	CHELLAIAH	69	M	716/16	CA PANCREAS	III	8	8	6	6	20	30	40	40	0	0	3	3	2	1	1
10	PERUMAL	59	M	797/16	CA PANCREAS	III	8	6	6	6	20	30	40	40	0	0	4	3	3	2	1
11	VEERAMMAL	79	F	801/16	PERI AMP CA	IV	8	6	6	8	20	30	30	40	0	0	3	4	2	2	1
12	AHMED BASHA	61	M	790/16	CA TRANSVERSE COLON	IV	6	6	4	4	20	30	30	40	0	0	3	3	2	1	1
13	PANJATHA	65	F	851/16	CA GALL BLADDER	IV	6	8	8	6	30	20	40	40	0	0	2	3	2	2	1
14	SENI	56	M	201/17	CA STOMACH	III	8	6	6	6	20	30	40	40	0	0	2	3	2	2	1
15	BATHRAKALI	62	F	239/17	CA STOMACH	III	6	6	4	2	30	30	40	40	0	0	3	3	1	3	1
16	RAJA MOHAMED	57	M	298/17	CA STOMACH	III	8	8	6	4	20	30	40	40	0	0	3	2	1	2	1
17	VIJAYA	60	F	342/17	CA STOMACH	III	8	6	6	4	20	30	30	30	0	0	3	2	2	2	1
18	KRISHNAN	66	M	666/17	CA PANCREAS	IV	6	4	4	2	20	40	40	40	0	0	3	2	2	1	1
19	SHANKARAN	60	M	671/17	CA STOMACH	III	8	6	6	6	20	30	30	40	0	0	3	3	2	1	1
20	RAJARAM	52	M	628/17	CA PANCREAS	IV	8	6	6	4	20	30	40	40	0	0	4	3	2	2	1
21	PANDIYAN	54	M	734/17	CA STOMACH	III	6	6	4	4	30	40	40	40	0	0	3	3	2	2	1
22	SEETHALAKSMI	49	F	782/17	PERI AMP CA	III	8	8	6	6	20	30	30	40	0	0	3	2	3	3	1
23	SAMPATH	63	M	794/17	CA TRANSVERSE COLON	IV	8	8	6	6	20	30	40	40	0	0	3	3	2	1	1
24	KALIAPPAN	59	M	804/17	CA PANCREAS	IV	8	8	6	6	30	30	40	40	0	0	3	2	2	2	1
25	UMAR	72	M	842/17	CA PANCREAS	III	6	6	6	4	20	30	30	30	0	0	2	2	2	1	1
26	AROKIYA MARY	60	F	892/17	CA LOWER 1/3 ESOPHAGUS	IV	6	4	4	4	20	30	30	30	0	0	3	3	3	2	1
27	SAVARIMUTHU	54	M	1002/17	CA GALL BLADDER	III	8	8	6	6	30	30	40	40	0	0	3	3	2	3	1
28	MUNIAPPAN	75	M	1013/17	CA STOMACH	IV	6	6	4	4	30	40	40	40	0	0	2	2	2	2	1
29	NAYAGI	62	F	1080/17	CA STOMACH	III	6	4	4	2	30	40	30	30	0	0	3	3	2	1	1
30	NANJUNDAN	56	M	1134/17	CA PANCREAS	IV	8	8	6	6	20	30	40	40	0	0	4	3	2	3	1

GROUP NCPB											
					PRE PROCEDURE						
SL.NO.	NAME	AGE	SEX	IP NO.	DIAGNOSIS	ASA/PS	VAS SCORE	MORPHINE CONSUMPTION	PERFORMANCE STATUS	HR	MAP
								MG/DAY			
1	ARUMUGAM	65	M	1794/16	CA PANCREAS	III	8	20mg	4	96	72
2	MURUGAN	43	M	416/16	CA STOMACH	III	10	40mg	4	90	76
3	KAMARAJ	50	M	883/16	CA GALL BLADDER	III	6	30mg	3	84	78
4	BHASKAR	52	M	215/16	CA PANCREAS	III	8	20mg	4	90	71
5	KANNIYAMMAL	45	F	1016/16	CA STOMACH	III	8	40mg	3	78	74
6	KATHAVARAYAN	49	M	4658/16	CA TRANS COLON	III	6	20mg	2	80	76
7	LALITHA	40	F	470/16	CA PANCREAS	III	8	40mg	2	76	74
8	BASKAR	60	M	841/16	PERI AMP CA	III	8	40mg	3	88	73
9	SURESH	48	M	534/16	CA PANCREAS	III	6	30mg	2	96	78
10	VASANTHAN	62	M	243/16	GIST METS	III	6	20mg	3	70	82
11	JAGAN MOHAN	50	M	145/17	CA STOMACH	III	8	30mg	2	78	77
12	KALIMUTHU	56	M	386/17	CA STOMACH	III	6	20mg	1	86	81
13	MURUGESAN	52	M	513/16	CA LOWER 1/3 ESOPHAGUS	III	8	30mg	2	82	74
14	VENKATESAN	61	M	476/16	CA GALL BLADDER	III	10	40mg	4	98	76
15	GUNASEKARAN	70	M	1723/16	CA STOMACH WITH METS	III	10	40mg	4	92	79
16	MARAN	68	M	328/16	CA PANCREAS	III	8	30mg	3	85	78
17	VALLI	56	F	459/16	CA GALL BLADDER	III	6	20mg	2	80	82
18	MARIAPPAN	60	M	230/17	CA TRANS COLON	III	8	40mg	3	84	77
19	GANESAN	51	M	141/17	CA PANCREAS	III	8	30mg	2	78	79
20	GNANAPANDI	56	M	265/17	CA PANCREAS	III	8	30mg	3	80	78
21	USHARANI	52	F	322/17	PERI AMP CA	III	6	20mg	2	74	76
22	SIVARAMAN	67	M	452/17	CA LOWER 1/3 ESOPHAGUS	III	8	40mg	3	86	75
23	RAJAN	73	M	431/17	CA STOMACH	III	8	30mg	2	80	72
24	RENUGADEVI	66	F	491/17	CA PANCREAS	III	10	40mg	4	94	78
25	ANGAPPAN	65	M	502/17	CA TRANS COLON	III	8	40mg	3	82	76
26	VELAPPAN	62	M	618/17	CA GALL BLADDER	III	8	30mg	3	80	71
27	AYYAPPAN	50	M	589/17	CA PANCREAS	III	8	40mg	3	87	75
28	ANJALAI	69	F	712/17	CA PANCREAS	III	8	30mg	3	82	78
29	GOWRI	62	F	769/17	CA GALL BLADDER	III	8	40mg	2	76	70
30	KARUPPUSAMY	59	M	891/17	CA LOWER 1/3 ESOPHAGUS	III	6	30mg	3	84	79

POST PROCEDURE																				
HEART RATE								MAP								VAS SCORE				
5MIN	15 MIN	30MIN	1 HR	2 HR	6HR	12 HR	24 HR	5MIN	15 MIN	30MIN	1 HR	2 HR	6HR	12 HR	24 HR	30 MINS	2 HRS	4 HRS	12 HRS	24 HRS
95	94	92	86	80	82	80	78	78	78	76	72	75	76	72	78	8	6	6	6	6
86	89	85	90	87	84	85	86	76	77	79	76	74	76	78	76	8	8	8	6	6
84	90	86	85	83	82	87	84	79	78	78	79	76	74	76	79	6	6	6	4	4
92	87	88	84	85	84	82	87	76	77	79	70	78	77	76	77	8	6	6	4	4
82	80	81	79	84	86	82	86	72	76	73	72	71	72	73	73	8	8	8	6	4
83	81	76	78	81	80	79	78	76	74	74	73	74	76	77	76	6	6	4	4	4
87	80	76	78	80	78	79	80	74	73	76	74	73	72	75	73	8	8	6	6	6
78	82	84	81	80	82	80	82	73	74	72	71	77	78	74	73	8	6	6	6	6
91	92	89	86	88	86	90	86	78	77	76	78	77	78	72	74	8	8	8	6	6
76	74	78	76	78	78	78	74	74	83	80	83	76	82	74	78	6	6	4	4	4
78	79	84	76	78	80	82	78	76	76	73	74	73	72	73	71	8	6	6	6	6
87	88	90	85	84	83	85	86	88	86	83	87	82	80	79	61	6	6	6	4	4
88	86	87	89	83	83	82	80	78	77	76	74	75	72	78	62	8	8	6	6	6
92	90	94	97	91	90	88	82	80	78	76	80	78	78	78	76	10	8	8	8	6
90	91	88	90	87	92	86	84	73	78	75	76	74	79	78	76	10	8	8	6	6
89	88	90	92	86	87	84	86	79	80	78	77	79	80	81	78	8	6	6	6	4
87	89	90	86	88	85	87	87	80	78	79	76	73	72	78	75	6	4	4	4	2
86	87	89	90	84	86	83	86	78	80	82	76	75	78	75	74	8	6	6	6	4
86	82	84	89	85	87	85	88	76	78	76	76	77	74	78	76	8	6	6	4	4
86	85	84	89	83	82	81	87	76	75	70	72	72	73	74	76	8	8	6	6	6
78	80	84	78	76	78	86	79	78	76	78	78	76	78	74	75	6	6	6	4	4
78	86	84	87	88	81	83	82	76	78	77	76	75	70	71	72	8	8	6	6	4
86	87	89	89	83	85	85	87	80	78	81	79	78	76	78	79	8	6	6	6	4
93	92	94	90	87	86	84	87	82	78	76	76	79	80	74	76	8	8	8	6	6
87	89	83	86	78	80	87	80	76	78	76	75	78	74	76	78	8	6	6	6	6
85	87	80	89	87	84	82	84	71	70	72	73	74	72	73	76	8	6	6	6	4
86	87	88	87	85	86	83	82	78	76	75	74	78	76	72	73	8	6	6	4	4
85	80	78	76	76	80	83	85	72	77	72	79	74	76	75	73	8	6	6	4	4
86	88	82	89	80	82	85	81	73	72	75	72	74	76	70	71	8	6	6	4	4
89	90	92	87	88	86	83	80	76	75	73	74	76	75	73	72	6	6	4	4	2

									SIDE EFFECTS		
VAS SCORE				MORPHINE CONSUMPTION				PERFORMANCE STATUS	CONSTIPATION	NAUSEA &VOMITING	TRANSIENT DIARRHOEA
1 WK	1 MTH	2MTH	4 MTH	1 WK	1MTH	2 MTH	4 MTH		1	1	1
2	2	0	0	20	10	5	0	1	1	1	3
4	2	2	2	30	20	20	10	3	1	2	3
2	0	0	0	30	20	10	0	2	1	1	4
2	0	0	0	20	10	0	0	1	1	2	1
2	2	0	0	40	30	10	10	1	1	2	1
4	4	4	2	20	20	10	10	2	1	2	3
6	4	2	2	40	30	30	20	2	1	1	2
4	2	2	2	40	20	10	10	1	1	2	2
4	4	2	2	30	20	20	20	2	1	1	1
4	4	2	2	20	20	20	20	3	1	2	1
4	2	2	2	30	20	20	20	2	1	1	1
4	2	2	0	20	20	10	0	1	1	2	3
4	2	0	0	20	20	10	0	0	1	1	2
4	2	2	2	40	30	20	20	3	1	2	3
4	4	4	4	30	20	20	20	2	1	2	2
4	2	2	0	30	20	10	0	1	1	1	2
2	0	0	0	20	10	0	0	1	1	2	1
4	2	2	2	40	30	10	10	2	1	1	3
2	2	0	0	40	30	20	10	1	1	2	1
4	4	2	2	30	30	20	10	2	1	1	2
2	2	0	0	20	20	10	0	1	1	1	2
4	2	2	2	40	20	20	10	2	1	2	3
2	2	2	0	30	20	10	0	2	1	2	2
4	2	2	2	40	30	10	10	3	1	2	2
4	2	0	0	40	30	20	10	2	1	3	2
4	2	0	0	30	20	10	10	2	1	3	3
2	2	2	0	40	30	20	0	2	1	2	4
2	2	2	0	30	30	20	10	2	1	2	2
2	2	0	0	40	20	10	0	1	1	2	2
2	0	0	0	30	20	10	0	1	1	4	4