NEET SS ANAESTHESIA

REGIONAL ANAESTHESIA

CONTENT

PHARMACOLOGY OF LOCAL ANAESTHETICS AND LAST

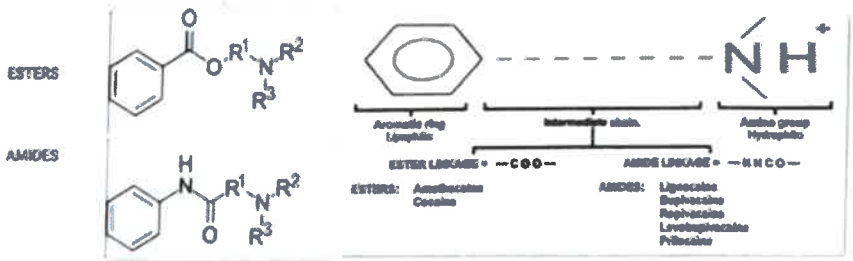
Introduction 00:00:27

History :

- Coca leaves (Cocaine) 3000 years ago found in mummies in south america.
- 1860 → First time coccaine was seperated.
- 1884 → Carl Koller gave it for anaesthesai in ophthal.
- 1885 → Leonard corning injected in spine of dog : Epidural and halted first nerve block.
- 1898 → Spinal anaesthesia by August bier.
- 1909 → IVRA.
- 1943 → Lidocaine.
- 1963 → Bupivocane.
- 1997 → Ropivocane.
- 2000 → Levobupivocane

Structure :

LA's contain an aromatic ring and an amine at opposite ends of the molecule, seperated by a hydrocarbon chain and either an ester or an amide bond.



Structure of local anaesthetic.

Local anaesthetics 00:04:50

Classification :

Esters :

a. Esters of benzoic acid : Cocaine, tetracaine, utacaine, benzocaine, hexyicaine, piperocain (used in dental).
b. Esters of PABA (Para amino benzoic acid) : Chloroprocaine, procaine,

propoxycaine.

Amides :

Articaine (used in dental), bupivacaine, etidocaine, lidocaine, mepivacaine. Prilocain, ropivacaine, levo - bupivacaine.

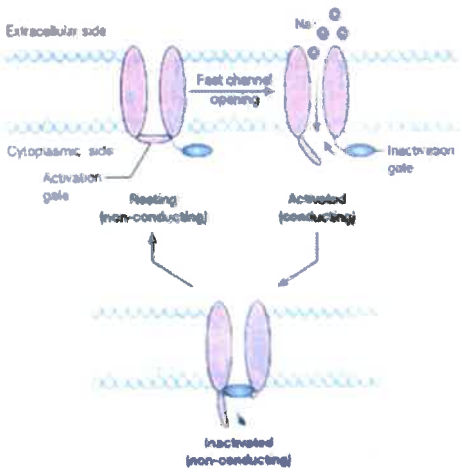
Quinolone : Centbucridine.

Key points :

- Esters are unstable solutions and rapidly hydrolysed by plasma cholinesterases.
- PABA main breakdown product hypersensitivity reactions.
- Amides are relatively stable solutions.
- Slowly metabolised by hepatic amidases.
- Hypersensitivity is rare .

Site of action :

- They act on voltage gated Na channels.
- Blocking of impulses in fibre requires that a defined length of nerve become inexcitable (To prevent jumping of AP).
- Conduction in myelinated fibres proceeds in jumps from one Ranvier node to the next , process termed saltatory conduction.
- To block three successive ranvier nodes which produces anaesthesia.
- Unmyelinated fibres, lacking the saltatory mechanism conduct much more slowly than myelinatd fibres, unmyelinated fibres are relatively resistant to LA.



Voltage gated Na channel.

MOA :

AP → Neuronal Na channel open → Extra cellular to inside flow → Depolarisation followed by Na channel inactivations and with membrane depolarisation → Na channel to resting state.

Na channel isoforms to remember :

- Na V 1.4 in skeletal muscles.
- Na V 1.5 in cardiac muscles.

- Na v IN Neural tissues
- Na v 1.8 in DRG, trigeminal ganglia, brain cells.

Properties :

Potency and duration :

- Potency is the dosage of drug required for 50% of its action.
- Potency increases with increase in molecular weight and lipid solubility as more permeability.
- more lipid solubility, highly protein bound, more slowly washed out, longer duration of action.
- Etidocaine and bupivacaine have greater lipid solubility and potency than lidocaine.

Classification	Potency	Onset	Duration after infiltration (min)	Maximum single dose for infiltration (mg)	Toxic plasma concentration (µg/ml)	pK	Protein binding (%)
Esters							
Procaine	1	Slow	45-60	500		8.9	6
Chloroprocaine	4	Rapid	30-45	600		8.7	
Tetracaine	16	Slow	60-180	100 (Topical)		8.5	76
Amides							
Lidocaine	1	Rapid	60-120	300	>5	7.9	70
Prilocaine	1	Slow	60-120	400	>5	7.9	55
Mepivacaine	1	Slow	90-180	300	>5	7.6	77
Bupivacaine	4	Slow	240-480	175	>3	8.1	95
Levobupivacaine	4	Slow	240-480	175		8.1	>97
Ropivacaine	4	Slow	240-480	200	>4	8.1	94

Classification	Fraction nonionized (%) at pH 7.4	Fraction nonionized (%) at pH 7.6	Lipid solubility	Volume of distribution (L)	Clearance (L/ml)	Elimination half-time (min)
Esters						
Procaine	3	5	0.6	65		9
Chloroprocaine	5	7		35		7
Tetracaine	17	11	80			
Amides						
Lidocaine	25	33	2.9	91	0.95	98
Prilocaine	24	33	0.9	191		96
Mepivacaine	39	50	1	84	9.78	114
Bupivacaine	17	24	28	73	0.47	210
Levobupivacaine	17	24		55		158
Ropivacaine	17			59	0.44	108

Dosage needed :

Agent	Onset	Duration	Maximum Dose	Maximum Dose With Epinephrine
Bupivacaine	5-10 min	200 min + (up to 540 min with epinephrine)	2.5 mg/kg	3 mg/kg
Lidocaine	<2 min	30-60 min (longer with epinephrine)	3 mg/kg	5 mg/kg
Articaine	2-3 min	180-360 min	7 mg/kg	7 mg/kg
Mepivacaine	3-5 min	45-90 min	6.6 mg/kg	5 mg/kg
Prilocaine	5 min	30-90 min	5 mg/kg	7 mg/kg
Ropivacaine	5-15 min	200 min +	3 mg/kg	3 mg/kg
Procaine	10-20 min	40 min	7 mg/kg	Not applicable

Fomulations :

- Chloroprocaine : 1%, 2%, 3%.
- Cocaine : 4%, 10%.
- Tetracaine : 0.2%, 0.3%, 1%, 2% amethocaine vial and eye drops.
- Benzocaine : 20% ointment.
- Procaine : 1%, 2%, 10%.
- Lidocaine / lignocaine : 2% (Local infiltration) , 4%,(Trans tracheal injection) 10% spray, 2% gel, 5% patches and gel (PHN).
- Bupivacaine : 0.25%, 0.5% and 0.75%.
- Levo bupivacaine : 0.25 % and 0.5%.
- Ropivacaine : 0.2% and 0.7%.

Note : Concentration of formulations are chosen based on the procedure for which anaesthesia is required.

Speed of onset :

- At any pH the % of LA molecules in unchanged form and available to cross membranes decrease with increase in Pka.
- Chloroprocaine is highly lipid soluble and highest Pka hence has faster acting.
- Bupivacaine and Ropivacaine are sensitive to sensory fibre and Bupivacaine has more rapid onset on sensory fibre.
- Ropivacaine less sensitive to motor fibres.
- Local anesthetics are weak bases with pKa values between 7.6 and 9.2.
- At physiological pH they are mostly in their ionized form.
- Poor health resulting from asthma, emphysema, diabetes, kidney disease, lung disease, gout, infection, shock and hemorrhage can affect blood pH, although acidosis is more common than alkalosis and less effectivity of LA.

Efficacy :

Depends on :

- Dose.
- Site of administration.
- Additives.
- Temperature.
- Pregnancy.

Additives :

- Opiods, alpha agonists, epinephrine, sodium bicarbonate, dexamethasone etc.

Efficacy in pregnancy :

- Pregnancy increases neural susceptibility to LA.
- LAs are partially protien bound, primarily to alpha 1 acid glycoprotein and secondarily to albumin.
- Affinity for alpha 1 gylcoprotein correlates with LA hydrophobicity and decreases with protonation (Acidity).
- Both protein binding and protein concentration decline during pregnancy.

Systemic absorption :

Order of systemic absorption of LA :

Intravenous > Tracheal > Intercostal > Caudal > Paracervical > Epidural > Brachial plexus > Sciatic > Subcutaneous.

Metabolism :

- Ester : Plasma hydrolysis, non specific esterases.
- Procaine and benzocaine are metabolised to para aminobenzoic acid (PABA)/anaphylaxis. Higher doses of benzocaine, threatening levels of methemoglobin.
- Amides undergo metabolism in the liver.
- Lidocaine undergoes oxidative N dealkylation (By the cytochromes CYP 1A2 and CYP 3A4) and hydroxylation.
- Prilocaine is hydrolyzed to O-toluidine, the agent that causes methemaglobinemia.

Pregnancy :

- Placental transfer depends on three factors :
  - Pka.
  - Maternal and fetal pH.
  - Degree of protein binding.
- Fetal acidosis : Higher fetal to maternal drug ratios because binding of H^+ ions to the nonionized form causes trapping.
- High protein binding drug like Bupivacaine and ropivacaine diffuse poory across placenta.
- Chloroprocaine : Least placental transfer as broken by plasma esterases.
- metabolism altered in pregnancy due to increased cardiac output, hepatic blood flow and clearance as well as the previously mentioned decline in protein binding.
- Renal failure tends to increase Vd of amide LAs and to increase the accu-

mulation of metabolic by products.

- Beta blockers and H2 receptor blockers inhibit CYP 2D6, itraconazole, ketoconazole inhibits CYP 3A4, Fluvoxamine inhibition

LAST

00:28:06

Overview :

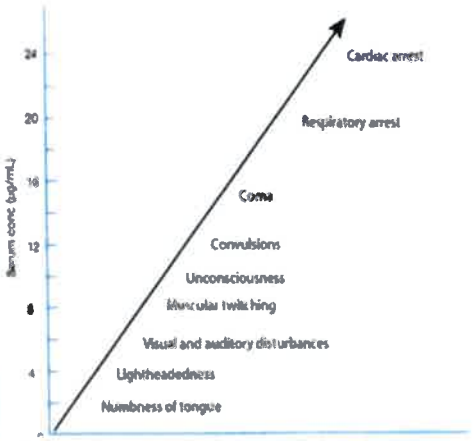
LAST : Local anaesthetic systemic toxicity.

In laboratory experiments, most LAs will not produce CV toxicity until the blood concentration exceeds three times that necessary to produce seizures.

Causes :

- Exceeding maximum dose.
- Inadvertent intravascular injection.
- Disconnection (Epidural to I/V).
- Cumulative effect of infusions and repeated blouses.
- Susceptible patients : Pre-existing cardiac conduction defects, metabolic mitochondrial defects.

Central nervous system	Cardiovascular system
Initial phase	Initial phase
Circumoral paresthesia	Hypertension
Tinnitus	Tachycardia during CNS excitatory phase
Confusion	
Excitatory phase	Intermediary phase
Convulsions	Myocardial depression
	Decreased cardiac output
	Hypotension
Depressive phase	Terminal phase
Loss of consciousness	Peripheral vasodilatation
Coma	Severe hypotension
Respiratory depression	Sinus bradycardia
	Conduction defects
	Dysrhythmias

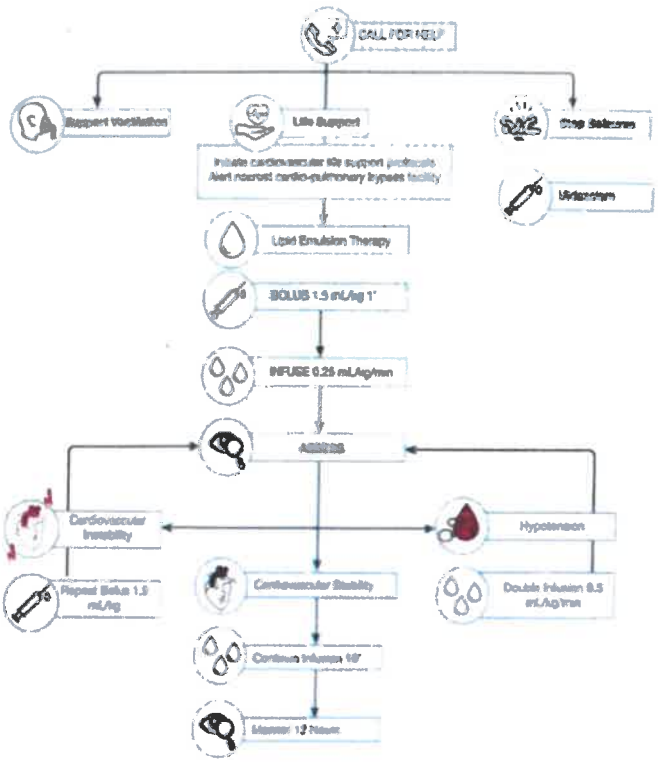


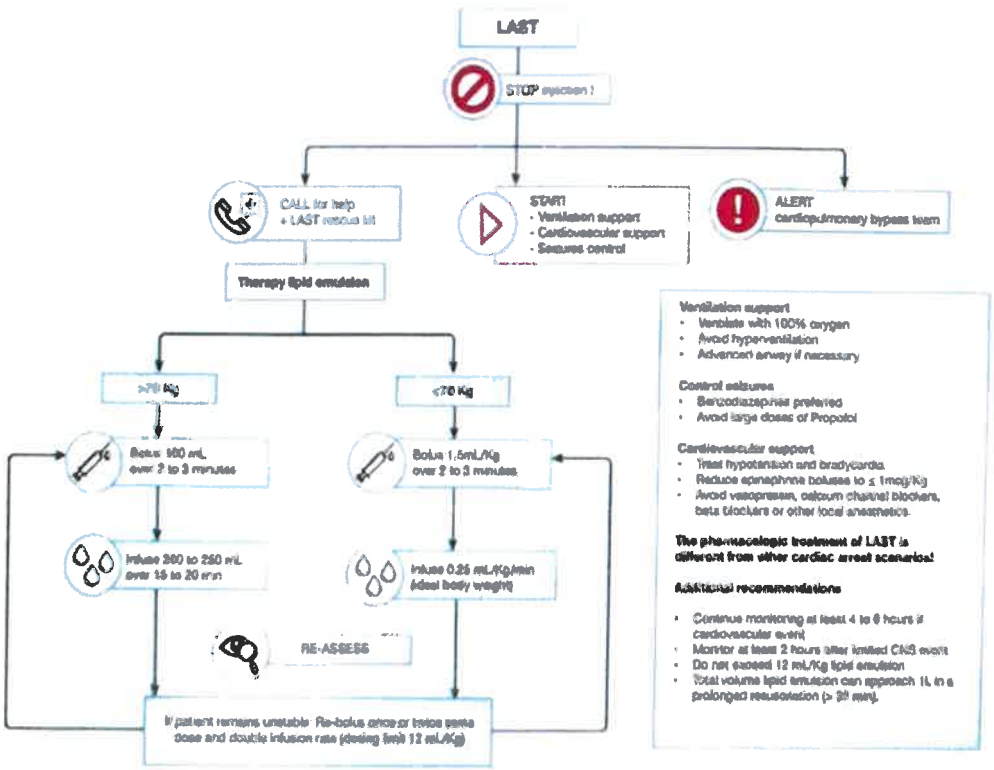
Symptoms of LAST.

- There is also controversy about transient neurologic symptoms and persistent sacral deficits after lidocaine spinal anesthesia.
- 2 chloroprocaine (at that time formulated with sodium metabisulfite at a relatively acidic pH) occasionally produced cauda equina syndrome following accidental large dose intrathecal injection.
- True anaphylaxis appears more common with ester LAs that are metabolized directly to PABA than other LAs.

- Other allergens : Sodium metabisulphite, Methyparabens etc.
- Methemoglobinemia seen with Prilocaine, Benzocaine, Articaine

Treatment :





Doses to treat complications :

- Seizures : IV midazolam (0.05–0.10 mg/kg) or propofol (0.5–1.5 mg/kg) or a paralytic dose of succinylcholine (0.5–1 mg/kg).
- CV depression manifested by moderate hypotension, iv fluids (phenylephrine 0.5–5 µg/kg/min, norepinephrine 0.02–0.3 µg/kg/min, or vasopressin 40 µg IV).
- myocardial failure : epinephrine (1–5 µg/kg IV bolus) may be required.
- with unresponsive bupivacaine cardiac toxicity cardiopulmonary bypass should be considered.

Management of Severe Local Anaesthetic Toxicity - AAGBI

1 Recognition

Signs of severe toxicity:
- Sudden alteration in mental status, severe agitation or loss of consciousness, with or without tonic-clonic convulsions
- Cardiovascular collapse: sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias may all occur
- Local anaesthetic (LA) toxicity may occur some time after an initial injection

2 Immediate management

- Stop injecting the LA
- Call for help
- Maintain the airway and, if necessary, secure it with a tracheal tube
- Give 100% oxygen and ensure adequate lung ventilation (hyperventilation may help by increasing plasma pH in the presence of metabolic acidosis)
- Confirm or establish intravenous access
- Control seizures: give a benzodiazepine, thiopental or propofol in small incremental doses
- Assess cardiovascular status throughout
- Consider drawing blood for analysis, but do not delay definitive treatment to do this

3 Treatment

IN CIRCULATORY ARREST
- Start cardiopulmonary resuscitation (CPR) using standard protocols
- Manage arrhythmias using the same protocols, recognising that arrhythmias may be very refractory to treatment
- Consider the use of cardiopulmonary bypass if available

WITHOUT CIRCULATORY ARREST

Use conventional therapies to treat
- hypotension,
- bradycardia,
- tachyarrhythmia

GIVE INTRAVENOUS LIPID EMULSION
(following the regimen overleaf)

- Continue CPR throughout treatment with lipid emulsion
- Recovery from LA-induced cardiac arrest may take >1 h
- Propofol is not a suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

CONSIDER INTRAVENOUS LIPID EMULSION
(following the regimen overleaf)

- Propofol is not a suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

4 Follow-up

- Arrange safe transfer to a clinical area with appropriate equipment and suitable staff until sustained recovery is achieved
- Exclude pancreatitis by regular clinical review, including daily amylase or lipase assays for two days
- Report cases as follows:
  - in the United Kingdom to the National Patient Safety Agency (via www.npsa.nhs.uk)
  - in the Republic of Ireland to the Irish Medicines Board (via www.imb.ie)

If Lipid has been given, please also report its use to the International registry at www.lipidregistry.org. Details may also be posted at www.lipidrescue.org

Your nearest bag of Lipid Emulsion is kept

This guideline is not a standard of medical care. The ultimate judgement with regard to a particular clinical procedure or treatment plan must be made by the clinician in the light of the clinical data presented and the diagnostic and treatment options available

MIXTURES OF LOCAL ANAESTHETICS AND ADJUVANTS

Local anaesthetic mixtures 00:00:21

Ideal local anaesthetic :

- Faster onset.
- Longer duration.
- Less toxicity.
- No perfectly ideal anesthetic exists.

Usage of mixtures :

So as to combine best quality of each component.

Eg : Intermediate acting faster onset (Lignocaine) with late onset longer duration (Bupivacaine) to get the desired effect.

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Ropivacaine	17			59	0.44	108

- A common misconception is that block duration is related to protein binding.
- More important is the extent to which local anesthetic remains in the vicinity of the nerve which is affected by three factors :

- Lipid solubility.
- The degree of vascularity of the tissue.
- Presence of vasoconstrictors that prevent vascular uptake.

Mixtures :

Lignocaine plus bupivacaine :

- Faster onset but only moderate duration of action.
- Latency was determined by the faster acting component, while duration tended to reflect but did not equal the longer acting component.
- moreover, duration of the local anesthetic mixture varied and was less predictable.
- No difference in either latency or duration of action was observed when mixing bupivacaine 0.5% or lidocaine 2% versus either alone, although there was a slight trend toward prolonged duration in the bupivacaine alone group.

Chlorprocaine and bupivacaine in epidural :

- Acted independent of each other, providing analgesia with rapid onset and long duration.

Bupivacaine ± Lignocaine OR Ropivacaine ± Lignocaine in sciatic block :

- Shorter latency by 33% to 50% with mixtures containing lidocaine.
- Equal volume mixtures of lidocaine 2% with bupivacaine or ropivacaine resulted in significantly shorter duration than bupivacaine or ropivacaine alone by up to 4 to 9 hours.

mepivacaine first then bupivacaine :

- For faster and long duration interscalene block.
- But it was found that the onset times and duration for both groups were identical, showing that, if a mixture is administered, it does not matter which drug is injected first or last.

Mixtures toxicity potential :

- Bupivacaine exhibits a narrow therapeutic window leading to more chances of LAST.
- Local anesthetic toxicity is additive. When mixing local anesthetics, individual fractional contributions to overall maximum recommended dose limits should be considered.
- USG use can decrease the amount of drug and thus toxicity.

- Recent advances in encapsulation of local anesthetic in liposomes offer promise, as these could extend the analgesic profile well beyond the usual 16–24 hours typically seen with our longest acting agents.

Euthetic mixture topical :

- 2.5% Lignocaine +2.5% Prilocaine (5% emulsion).
- Diffuse through intact skin.
- Dose : 1–2 gm/10 cm^2 under occlusive dressing.
- Applied 45–60 min prior to procedure.
- Used in IV cannulation, circumcision, skin graft harvest and cauterising genital warts.
- Commonly K/a EMLA (Eutectic mixture of local anesthetics).

Additives/ Adjuvants

00:10:39

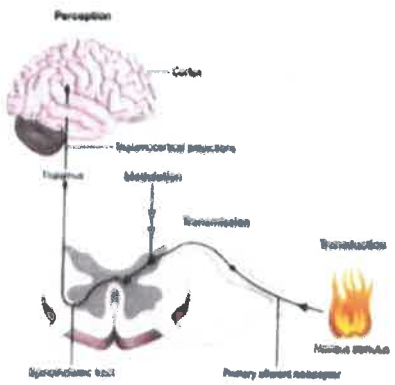
Ideal additive (Any drug increasing potency and efficacy) is not available, has the following characteristics :

- Faster speed onset, prolong effect, and reduce total required dose.
- Can enhance postoperative analgesia without prolonging adverse effects of local anesthetics.
- Act at peripheral sites without central effects, thereby optimizing analgesia with minimal CNS side effects.

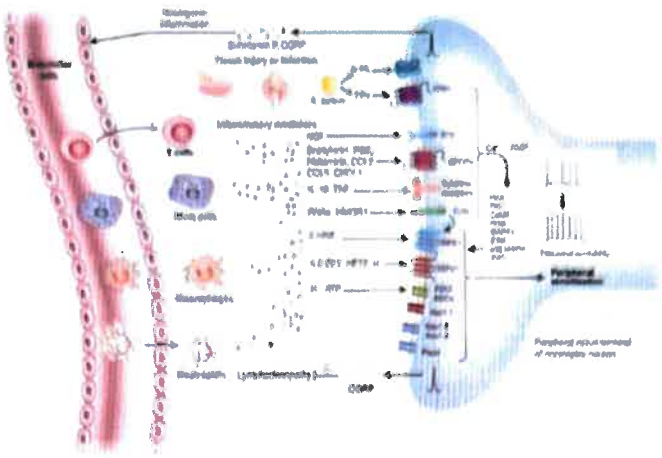
Opioids :

mechanism of action :

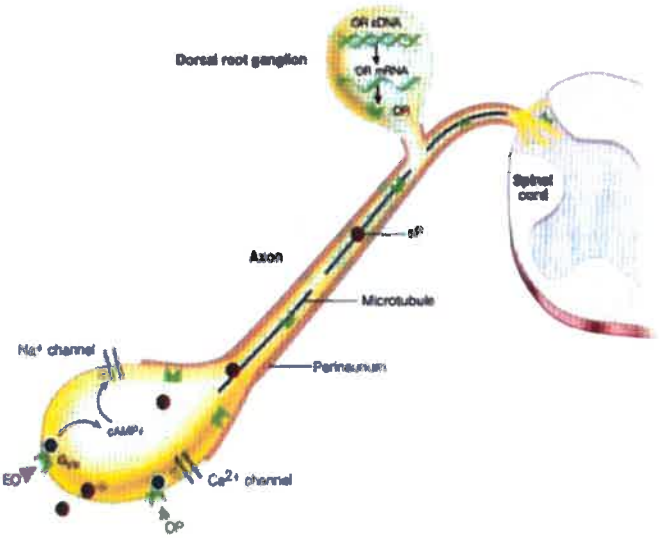
- Act on opioid receptors both PNS and CNS.
- At level of transmission, transduction and perception.
- At the terminals, opioid receptors are incorporated into the neuronal membrane and become functional receptors.
- The permeability of the perineurium is increased within inflamed tissue, enhancing the ability of opioids to reach target receptors.



Opioids MOA



- On activation by exogenous or endogenous opioids (Released by immune cells), opioid receptors couple to inhibitory G proteins.
- This leads to direct or indirect (Through decrease of cAMP) suppression of Ca^{2+} or Na^{+} currents and subsequent attenuation of substance P release.



- Opioid receptors and neuropeptides (Eg, substance P) are synthesized in the dorsal root ganglion.
- They are transported along intraaxonal microtubules into central and peripheral processes of the primary afferent neuron.

Opiod drugs used :

- Buprenorphine : Partial μ-receptor agonist with a very high receptor affinity and it has intermediate lipid solubility, which allows it to cross the neural membrane.
- Tramadol : Weak opioid agonist with some selectivity for the μ-receptor that also inhibits NE reuptake and stimulates serotonin release in the intrathecal space and are transmitters for the descending control pathway in

the spinal cord and enhance analgesia.

- Acts neuraxially at Epidural/intrathecal/perineural, intraarticular.
- Opioid agonists administered into inflamed tissue will bind to opioid receptors on sensory terminals and induce analgesia. Animal studies indicated that these peripheral opioid receptors are expressed 9% hours after the initial inflammatory injury.
- Adding opiods with local anaesthesia provides prolonged analgesia.

Dosage for opiods :

- Tramadol : 100-200 mg (Intrathecally).
- Buprenorphine : 150-300 mcg (Intrathecally).
- morphine : 50-300 mcg intrathecal/2-5 mg epidurally (Newer additive).
- Fentanyl : 10-25 mcg/50-100 mcg epidurally (Newer additive).

Drug	Intrathecal dose	Epidural loading dose
Fentanyl	10-25 µg	50-100 µg
Sufentanil	2.5-10 µg	10-50 µg
morphine	50-300 µg	2-5 mg
Diamorphine	300-400 µg	2-3 mg
Pethidine	Not recommended	25-50 mg

Alpha 2 agonist / clonidine :

- α_2-receptors exist in the dorsal horn of the spinal cord, and stimulation of these receptors produces analgesic effects by inhibiting the presynaptic release of excitatory transmitters, including substance P and glutamate.
- Intrathecal clonidine mediates analgesia by increasing acetylcholine levels, which in turn stimulates muscarinic receptors. Muscarinic excitation increases γ-amino butyric acid levels onto the primary afferent fiber, inhibiting the release of the excitatory neurotransmitter glutamate.
- Clonidine is alpha 2 with alpha 1 stimulatory effects :
  - It acts as a local anesthetic and blocks AP at C fibres and A delta.
  - Also due to alpha 1 activity of vasocontrictive effect leads to prolonged action.
- Dexmedetomidine may be expected to produce not only more profound analgesia but also greater adverse effects because of the selectivity of action (Alpha 2).
- Side Effects : Hypotension, bradycardia, and sedation at higher doses, and

these effects may outweigh any analgesic benefits.

Doses :

- PNB : Clonidine in doses up to 1.5 µg/kg prolongs sensory block and analgesia when added to LA.
- Can be added intathecally/intraarticular/IVRA.
- Clonidine intrathecal of 30-200mcg and epidural of 6-8 mcg/kg bolus.
- Dexmedetomidine prolongs the analgesic effects of brachial plexus blocks by 284 minutes.
- Intathecal dose used is 3 to 5 mcg and epidural dose is 1mcg/kg.

Dexamethasone :

- Mechanism : Attenuating the release of inflammatory mediators, reducing ectopic neuronal discharge, and inhibiting potassium channel mediated discharge of nociceptive C fibers.
- Dose used is 8 mg perineural/systemic.
- Prolongation in both dexamethasone groups from 12 hours to approximately 20 and 22 hours for systemic and perineural administration, respectively.

NMDA antagonists :

NMDA receptors on dorsal horn is involved in nociceptive signally and central senstisation.

Magnesium :

- Perineural/intrathecal/epidural/IV.
- Perineural dose : 1.5 gm.
- Intrathecal dose : 50 to 100 mg.
- Epidural dose : 50 to 600 mg.

Ketamine :

- Additive for IVRA at 0.5 mg /ml for prolonged effect but side effects are seen.
- Given as intraarticular injection 0.5 mg/kg or local infiltration of 3mg/ml.

Others :

Cyclooxygenase inhibitor (Ketorolac) :

- For IVRA dose : 20mg.
- For infiltration dose : 30 to 60 mg.

- L-acetylsalicylate dose of 90 mg.

Neostigmine :

- muscarinic receptors mediate analgesia in the dorsal horn of the spinal cord, and neostigmine has produced analgesia when administered to both the intrathecal and epidural space,not much effective.
- Neostigmine as an analgesic adjuvant for intra-articular use after knee arthroscopy in a dose of 500 mcg.

Calcitonin :

- used as Epidural/intrathecal at 100 IU.

Midazolam (BDZ) :

- Acts in substantia gelatinousa on BDZ receptors.
- Facilitates inhibitory action on GABA.
- Acts on delta opioid receptors.
- 1-2 mg intrathecally prolong effect by 2-6 hours.
- Perineural : 50 mcg/Kg dose.

Key points :

Perineural :

- Dexamethasone of 4-10 mg.
- Buprenorphine for 0.3 mg.
- Clonidine for 1-2 mcg/Kg.
- Tramadol of 200 mg.

IVRA :

- magnesium at 1.5 gm and dexmed at 0.5 mcg/Kg.

Intraarticular :

- Clonidine 150 mcg and morphine 5 mg.

Local infiltration :

- Ketamine 3 mg/ml.

EQUIPMENTS IN REGIONAL ANESTHESIA

Needles

00:00:21

Features :

- Needles for single-shot nerve blocks are generally short bevelled, attached to extension tubing to enable drug administration and an electrical cord with a female connector for nerve stimulation.
- These are available in varying lengths (25-150 mm) and calibre (20-25 gauge, most commonly 22G needles are used) depending on the specific needs of the desired block.
- These needles will have markings along the shaft to monitor the depth of needle penetration.
- Generally, the shortest possible needle length (that still enables access to desired site) is preferred, as this allows better handling and control of the needle.

Stimulator needles :

- Stimulator needles have an insulated shaft such that electrical conductivity occurs only at the bevel, allowing a smaller current to be used for nerve stimulation and more accurate positioning of the needle relative to the target area.
- With the advent of ultrasound-guided regional techniques, echogenic needles have been crafted in an effort to improve visibility on ultrasound imaging.
- These reflect ultrasound beams and enhance needle visualisation generally either by including an echogenic layer (which traps micro air bubbles creating specular reflectors on the needle surface) or indentations on the needlesurface, which act as reflectors.
- 30 Degree level, 50 mm and 100 mm length, most commonly used

Advantages :

- Puncture and handling characteristics with 30° level
- Consistent feel : Enables user to appreciate the needle's passage through different tissue planes.
- Optimal glide resistance : For smooth progression of needle.