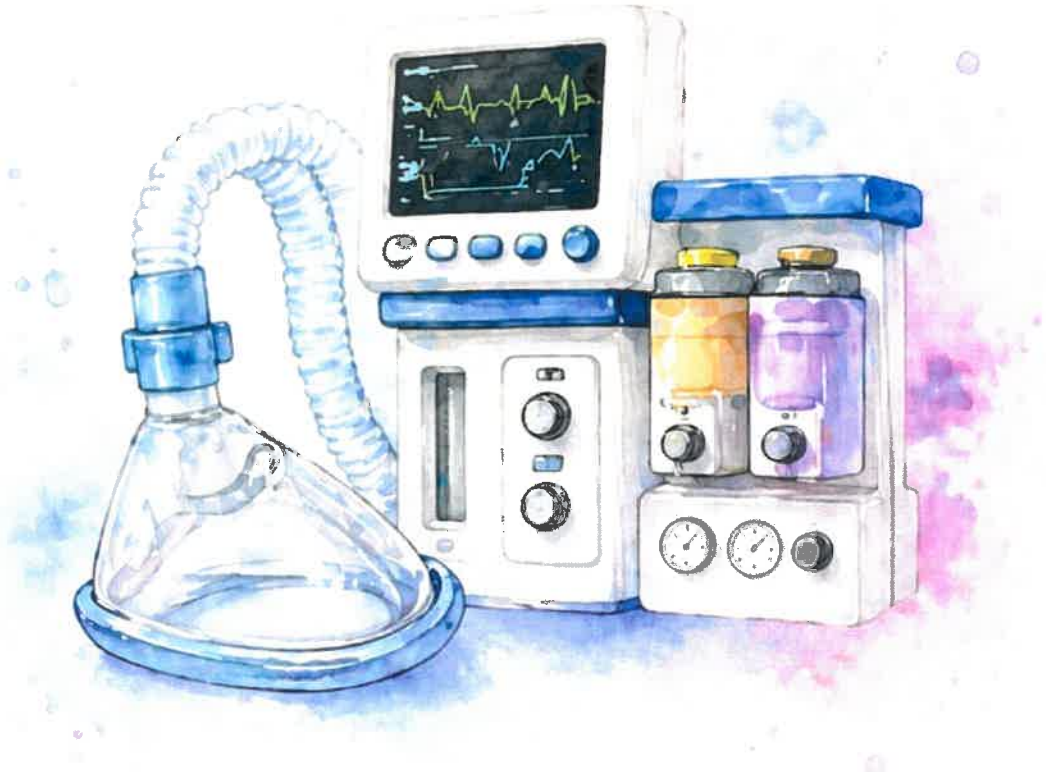
NEET SS ANESTHESIA

Updated Notes 2026



REGIONAL ANAESTHESIA

Contents

Regional Anaesthesia in Specific Populations

Complications and Special Techniques

PHARMACOLOGY OF LOCAL ANAESTHETICS AND LAST

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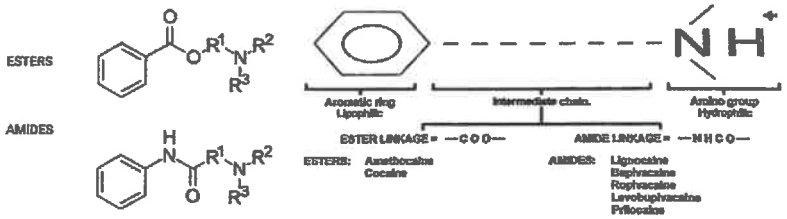
Introduction 00:00:27

History :

- Coca leaves (Cocaine) 3000 years ago found in mummies in South America.
- 1860 : First time cocaine was separated.
- 1884 : Carl Koller gave it for anaesthesia in ophthal.
- 1885 : Leonard Corning injected in spine of dog → Epidural; Halsted first peripheral nerve block.
- 1898 : Spinal anaesthesia by August Bier.
- 1909 : IVRA.
- 1943 : Lidocaine.
- 1963 : Bupivacaine.
- 1997 : Ropivacaine.
- 2000 : Levobupivacaine.

Structure :

- LAs contain an aromatic ring and an amine at opposite ends of the molecule, separated by a hydrocarbon chain and either an ester or an amide bond.



Structure of local anaesthetic

Local anaesthetics 00:04:50

Classification :

Esters :

- Esters of benzoic acid : Cocaine, tetracaine, butacaine, benzocaine, hexylcaine, piperocaine (used in dental).
- Esters of PABA (Para amino benzoic acid) : Chloroprocaine, procaine, propoxycaine.

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Amides :

- Articaine (used in dental), bupivacaine, etidocaine, lidocaine, mepivacaine, prilocaine, ropivacaine, levobupivacaine.

Quinolone : Centbucridine.

Key points :

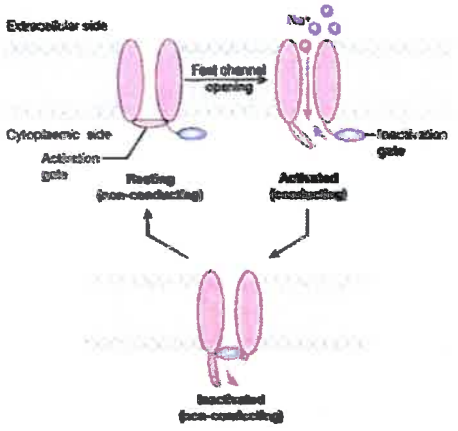
- Esters are unstable solutions and rapidly hydrolysed by plasma cholinesterases.
  - PABA is a main breakdown product → Hypersensitivity reactions.
- Amides are relatively stable solutions.
  - Slowly metabolised by hepatic amidases.
  - Hypersensitivity is rare .

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 myelinated fibres; unmyelinated fibres are relatively resistant to LA.

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.



Voltage gated Na channel

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 :

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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 there is 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/m)	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		156
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	5-6 mg/kg	6 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

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Formulations :

- 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 is less sensitive to motor fibres.
- Local anaesthetics 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 :

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

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Efficacy in pregnancy :

- Pregnancy increases neural susceptibility to LA.
- LAs are partially protein bound, primarily to alpha 1 acid glycoprotein and secondarily to albumin.
- Affinity for alpha 1 glycoprotein 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 can lead to life threatening levels of methemoglobinemia.
- 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 non-ionized form causes trapping.
- High protein binding drug like bupivacaine and ropivacaine diffuse poorly 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 accumulation of metabolic by products.
- Beta blockers and H2 receptor blockers inhibit CYP 2D6, itraconazole, Ketoconazole inhibits CYP 3A4, Fluvoxamine inhibition.

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LAST

00:28:06

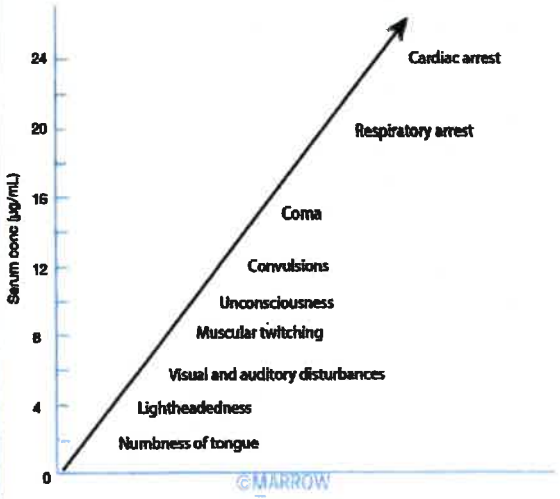
Overview :

- 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 IV).
- Cumulative effect of infusions and repeated boluses.
- 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 anaesthesia.
- 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, methylparabens, etc.
- methemoglobinemia seen with prilocaine, benzocaine, articaine.

Treatment :

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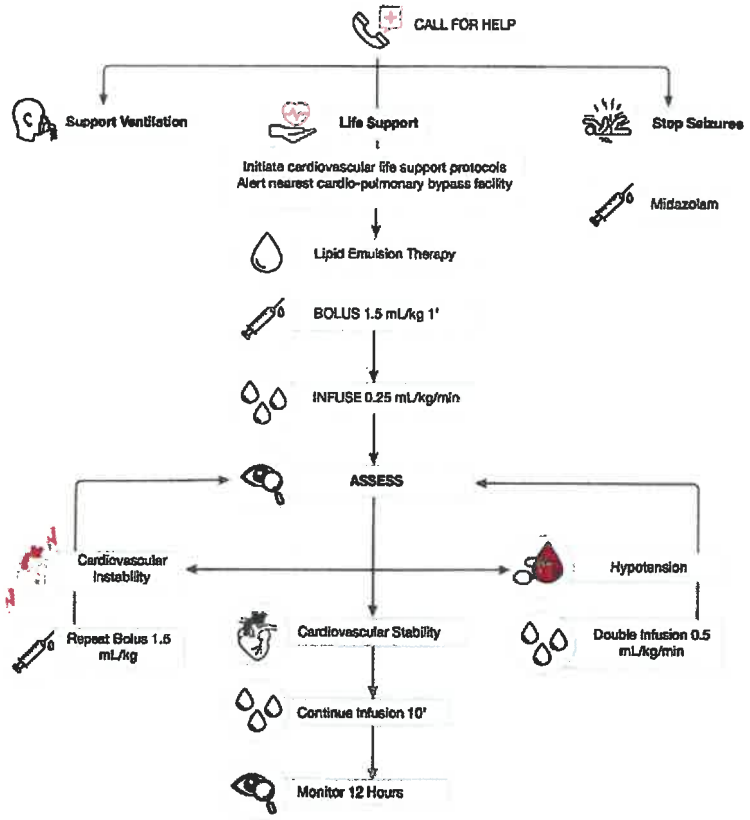

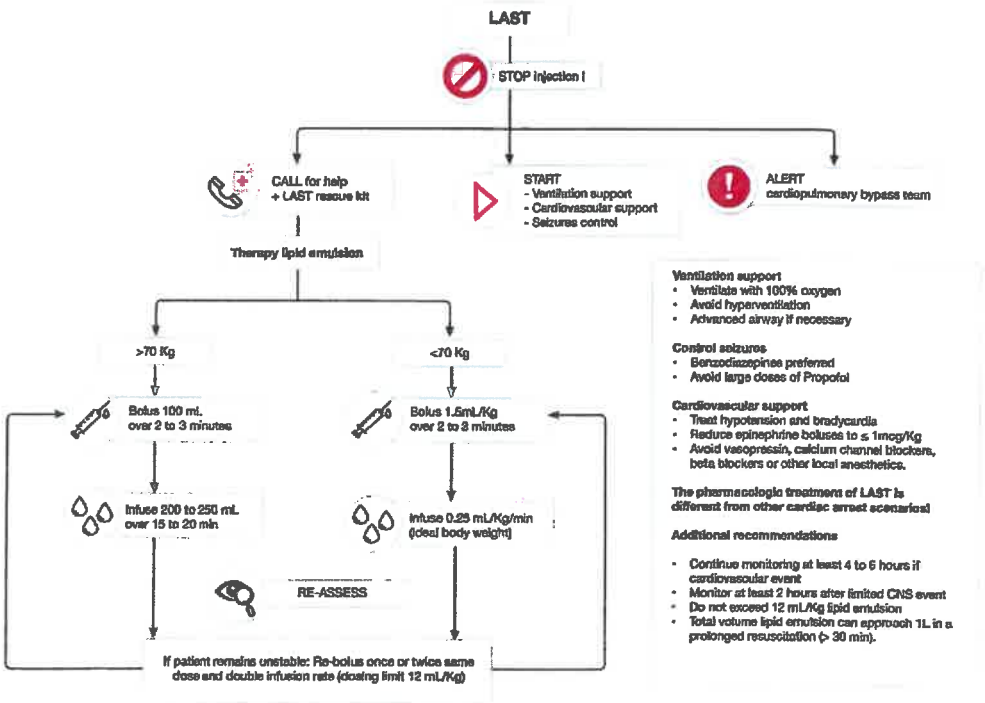

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Doses to treat complications :

- 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 or norepinephrine 0.02–0.2 µg/kg/min or vasopressin 40 µg IV.
- Myocardial failure : Epinephrine (1–5 µg/kg IV bolus) may be required.
- 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 **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	**WITHOUT CIRCULATORY ARREST** Use conventional therapies to treat: • hypotension, • bradycardia, • tachyarrhythmia **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

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Local anaesthetic mixtures 00:00:16

Properties of ideal local anaesthetic :

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

Usage of mixtures :

- So as to combine best quality of each component.
- E.g. : 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

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- A common misconception is that block duration is related to protein binding.
- More important is the extent to which local anaesthetic 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 + 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 anaesthetic 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.

Chloroprocaine 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 anaesthetic toxicity is additive : When mixing local anaesthetics, individual fractional contributions to overall maximum recommended dose limits should be considered.

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- USG use can decrease the amount of drug and thus toxicity.
- Recent advances in encapsulation of local anaesthetic 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.

Eutectic mixture topical :

- 2.5% Lignocaine +2.5% Prilocaine (Total 5% emulsion).
- Diffuse through intact skin.
- Dose : 1–2 g/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 anaesthetics).

Additives/ Adjuvants 00:10:38

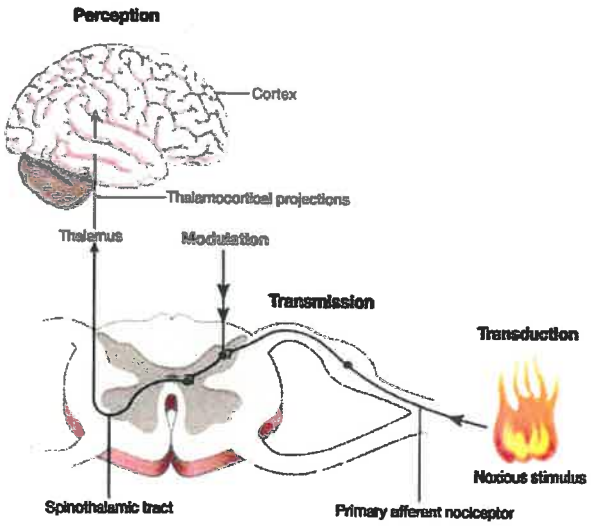
Ideal additive (Any drug increasing potency and efficacy) is not available, has the following characteristics :

- Faster speed onset, prolong effect, and reduce total required dose.
- Can enhance postoperative analgesia without prolonging adverse effects of local anaesthetics.
- 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

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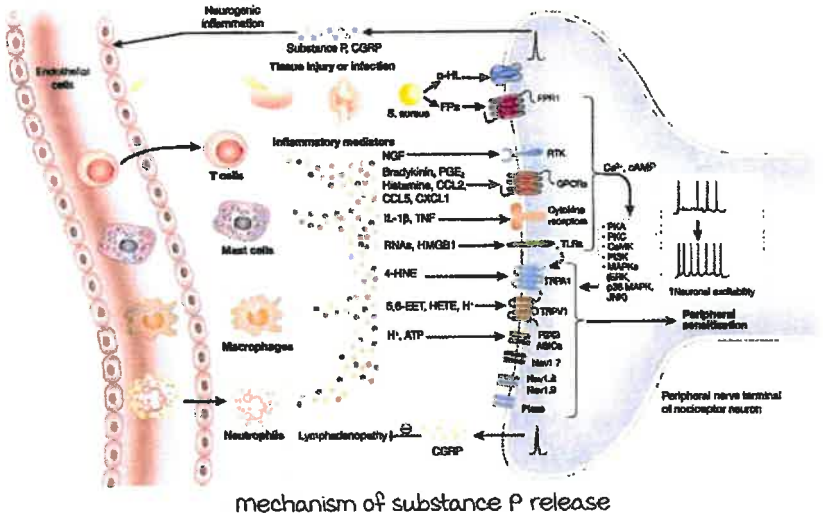
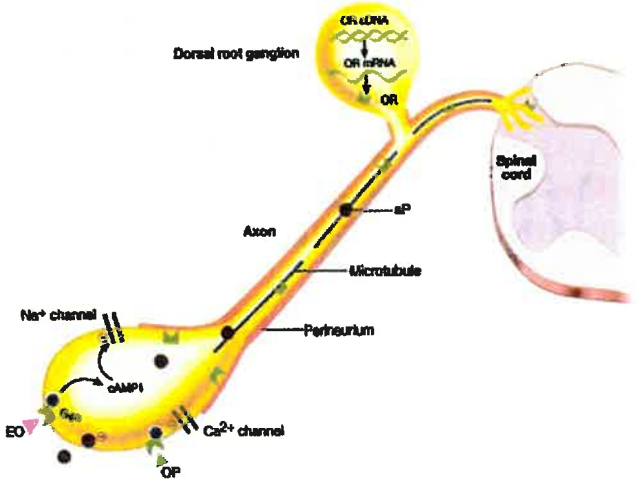


Mechanism of substance P release

- On activation by exogenous or endogenous opioids (Released by immune cells), opioid receptors couple to inhibitory Gi 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 receptor production, transport and signaling in primary afferent neurons

- Opioid receptors and neuropeptides (E.g., 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.

Opioid 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.

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- Tramadol : Weak opioid agonist with some selectivity for the μ-receptor that also inhibits NE re-uptake and stimulates serotonin release in the intrathecal space and are transmitters for the descending control pathway in the spinal cord and enhance analgesia.
- Neuraxial action 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 96 hours after the initial inflammatory injury.
- Adding opioids with local anaesthesia provides prolonged analgesia.

Dosage for opioids :

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

Clonidine :

- α2-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.
- MOA : Intrathecal clonidine → Increase acetylcholine levels → Stimulates muscarinic receptors. → Increases γ-amino butyric acid levels onto the primary afferent fiber → Inhibit the release of the excitatory neurotransmitter glutamate.
- Clonidine is alpha 2 with alpha 1 stimulatory effects :
  - It acts as a local anaesthetic and blocks AP at C fibres and A delta.
  - Also due to alpha 1 activity : 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).

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- Side effects : Hypotension, bradycardia, and sedation at higher doses, and these effects may outweigh any analgesic benefits.

Doses :

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

Magnesium :

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

Ketamine :

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