Al Qadisiyah-Dentistry

Local Anaesthesia

PHARMACOLOGY
DR. KHETAM ALHILALI

General Introduction of Local Anesthesia in Dentistry

History of Local Anesthesia

The history of local anesthesia started in 1859, when cocaine was isolated by Nieman.

In 1884, the ophthalmologist Koller was the first, who used cocaine for topical anesthesia in ophthalmologic surgery.

In 1884, local anesthesia in the oral cavity was first performed by the surgeon Halsted, when he removed a wisdom tooth without pain.

However, a number of adverse effects were observed with the clinical use of cocaine. Thus, other local anesthetic agents had to be developed.

General Introduction of Local Anesthesia in Dentistry

History of Local Anesthesia

In 1905, Einhorn reported the synthesis of procaine, which was the first ester-type local anesthetic agent. Procaine was the most commonly used local anesthetic for more than four decades.

In 1943, Löfgren synthesized lidocaine, which was the first "modern" local anesthetic agent, since it is an amide-derivate of diethyl amino acetic acid. Lidocaine was marketed in 1948 and has remained one of the most commonly used local anesthetics in dentistry worldwide, although other amide local anesthetics were introduced into clinical use: mepivacaine 1957, prilocaine 1960, bupivacaine 1963.

Definition A local anaesthetic

- Local anesthetic: produce loss of sensation to pain in specific area of the body without the loss of consciousness
- Definition A local anaesthetic can be defined as a drug which reversibly prevents transmission of the nerve impulse in the region to which it is applied, without affecting consciousness
- Definition: Local anesthetics are drugs which upon topical application or local injection cause reversible loss of sensory perception, especially of pain in a localized area of the body. Block generation and conduction of nerve impulses at a localized site of contact without structural damage to neurons.
- Clinically - to block pain sensation from - or sympathetic vasoconstrictor impulses to specific areas of the body - Loss of sensory as well as motor impulses

Pain

Pain

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Introduction

- Pain control in dentistry presents one of the greatest challenges. (Pain leads to increased stress, release of endogenous catecholamines and unexpected cardiovascular responses).
- Before anesthetization

❖ Common Questions To Ask The Patient

1. Allergic to any medications?
2. Have you ever had a reaction to local anesthesia?
3. If yes, describe what happened ?
4. Was treatment given? If so, what?

❖ Preparation Of The Patient

- ❑ Careful Preoperative Assessment
- ❑ History
- ❑ A clear explanation of what to expect

PREOPERATIVE ASSESSMENT

Data should be documented which includes:

- ❑ Baseline vital signs
  - Blood pressure
  - Laboratory values
  - Results of ECG monitoring
  - Any other tests.
- ❑ Weight, height, and age
  - Dosage of some drugs is calculated on the basis of body weight in kilograms (mg/kg).
  - Some drugs are contraindicated for age extremes (i.e., pediatric or geriatric patients).

- Current medical problem(s) past history of medical events, including a history of substance abuse.
- Current medications or drug therapy, such as insulin for diabetes or hypertensive drugs.
- Allergy, or hypersensitivity reactions to previous anesthetics or other drugs.
- Mental status, including emotional state and level of consciousness.
- Communication ability A patient with hearing impairment or language barrier may be unable to understand verbal instructions during the procedure or to respond appropriately.

STRESS REDUCTION PROTOCOL

- ❑Morning appointments are usually best.
- ❑Keep appointments as short as possible.
- ❑Freely discuss any questions, concerns, or fears that the patient has.
- ❑Establish a honest, supportive relationship with the patient.
- ❑Maintain a calm, quiet, professional environment.
- ❑Provide clear explanations of what the patient should expect and feel.

LOCAL ANESTHETIC USE IN MEDICALLY COMPROMISED PATIENTS

Disease	Precautions
Cardiovascular disease	Use stress reduction protocol
Hypertension	Minimize vasoconstrictor use
Pulmonary disease	
Asthma	Stress reduction protocol; minimize vasoconstrictor use
COPD	No special precaution
Renal disease(severe)	Reduced dosage; extend time between injections
Pancreatic disease	
Diabetes	Stress reduction protocol
Blood dyscrasias	
Sickle cell anemia	Stress reduction protocol; minimize vasoconstrictor use

Ideal LA

- Reversible action.
- Non-irritant.
- No allergic reaction.
- No systemic toxicity.
- Rapid onset of action.
- Sufficient duration of action.
- Potent.
- Stable in solutions.
- Not interfere with healing of tissue.
- Have a vasoconstrictor action
- Not expensive

cocaine

- Cocaine: For many dentists and tooth pain patients, cocaine was considered a popular pain relief method for a long time. Carl Koller argued the use of cocaine as local anesthesia for pain, but it is now considered a highly addictive substance that alters the brain structure if used consistently.
- Here's what happens in the body:
- Heart - Cocaine is bad for the heart. Cocaine increases heart rate and blood pressure while constricting the arteries supplying blood to the heart.
- Brain - Cocaine can constrict blood vessels in the brain, causing strokes. This can happen even in young people without other risk factors for strokes. Cocaine causes seizures and can lead to bizarre or violent behavior
- Lungs and respiratory system. Snorting cocaine damages the nose and sinuses. Regular use can cause nasal perforation. Smoking crack cocaine irritates the lungs and, in some people, causes permanent lung damage.
- Sexual function. Although cocaine has a reputation as an aphrodisiac, it actually make you less able to finish what you start. Chronic cocaine use can impair sexual function in men and women. In men, cocaine can cause delayed or impaired ejaculation.

Medical uses

- **Acute pain** : Even though, acute pain may be managed using analgesics, conduction anesthesia may be preferable because of superior pain control and fewer side effects
- **Chronic pain** : Chronic pain is a complex and often serious condition that requires diagnosis and treatment by an expert in pain medicine. LAs can be applied repeatedly or continuously for prolonged periods to relieve chronic pain, usually in combination with medication such as opioids, NSAIDs, and anticonvulsants. Though it can be easily performed, repeated local anaesthetic blocks in chronic pain conditions are not recommended as there is no evidence of long-term benefits.
- **Surgery** : Virtually every part of the body can be anesthetized using conduction anesthesia. However, only a limited number of techniques are in common clinical use. Sometimes, conduction anesthesia is combined with general anesthesia or sedation for the patient's comfort and ease of surgery. However, many anaesthetists, surgeons, patients and nurses believe that it is safer to perform major surgeries under local anesthesia than general anesthesia.[4] Typical operations performed under conduction anesthesia include:

Medical uses / **Surgery**

- Dentistry (surface anesthesia, infiltration anesthesia or intraligamentary anesthesia during restorative operations such as fillings, crowns, and root canals, or extractions, and regional nerve blocks during extractions and surgeries)
- ENT operations, head and neck surgery (infiltration anesthesia, field blocks, or peripheral nerve blocks, plexus anesthesia)
- Shoulder and arm surgery (plexus anesthesia or intravenous regional anesthesia)
- Heart and lung surgery (epidural anesthesia combined with general anesthesia)
- Abdominal surgery (epidural anesthesia/spinal anesthesia, often combined with general anesthesia during inguinal hernia repair or other abdominal surgery)
- Gynecological, obstetrical, and urological operations (spinal/epidural anesthesia)
- Bone and joint surgery of the pelvis, hip, and leg (spinal/epidural anesthesia, peripheral nerve blocks, or intravenous regional anesthesia)
- Surgery of skin and peripheral blood vessels (topical anesthesia, field blocks, peripheral nerve blocks, or spinal/epidural anesthesia)

How Does a Nerve Impulse Occur?

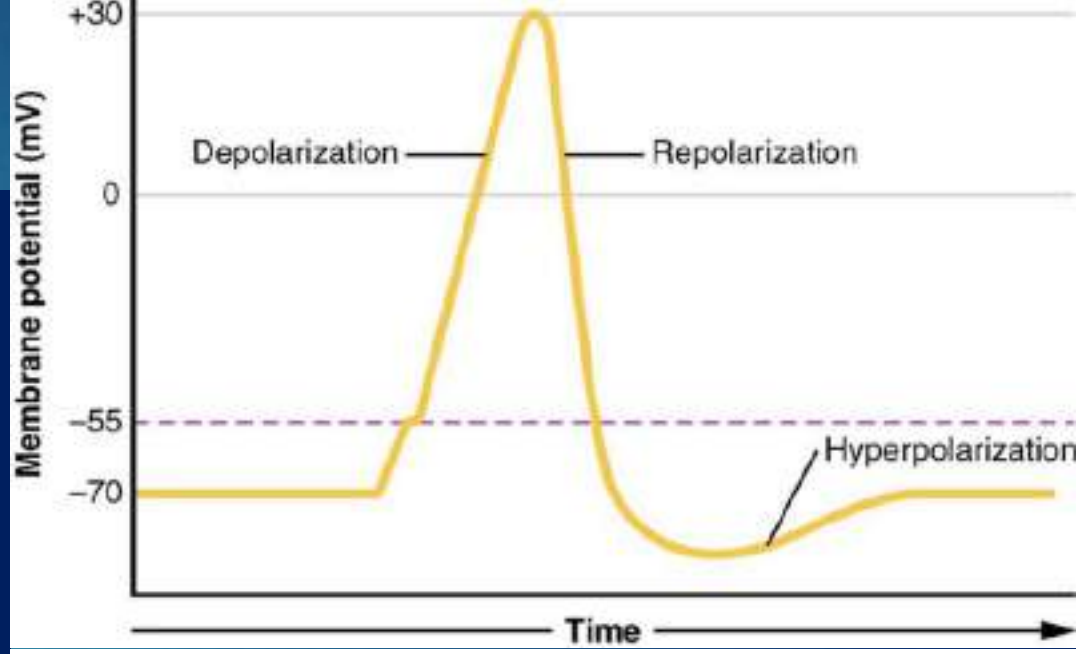
Starts with a **Stimulus** (-90mv_-60mv) →

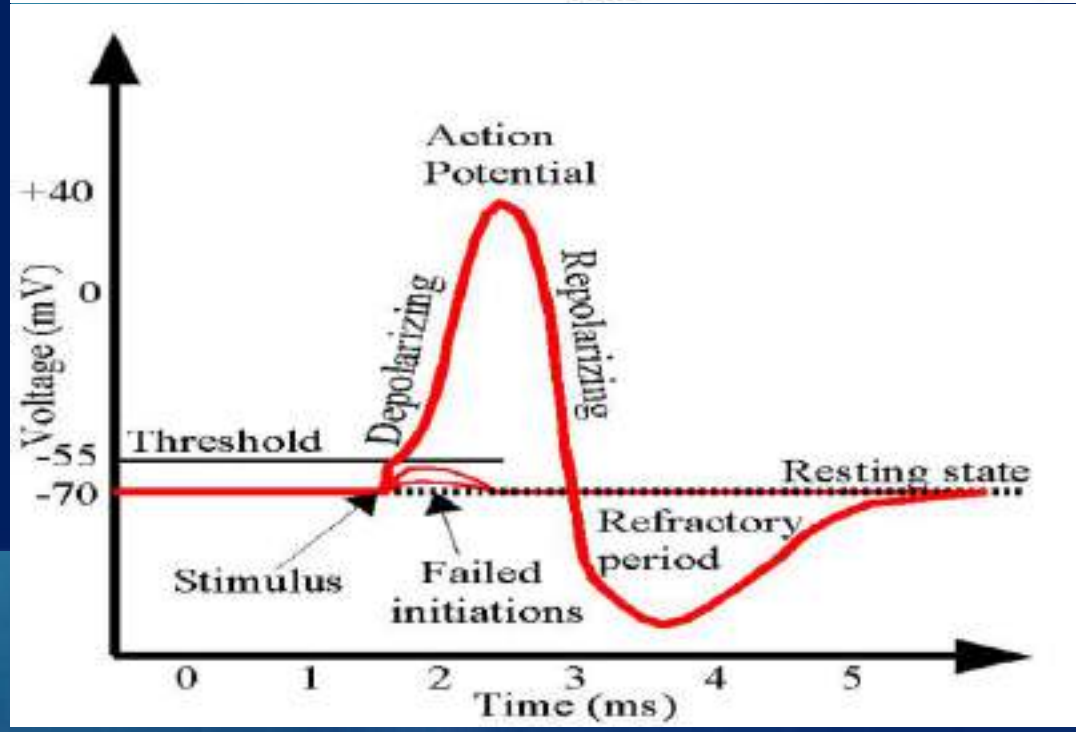
Depolarization of the nerve →

Na+ flows from extracellular to Intra-cellular space →

Repolarization →

K+ flows from Intra-cellular to extra-cellular space





How does a nerve impulse occur .cont

- In the generation of the action potential, stimulation of the cell by neurotransmitters or by sensory receptor cells partially opens channel-shaped protein molecules in the membrane.
- Sodium diffuses into the cell, shifting that part of the membrane toward a less-negative polarization.
- If this local potential reaches a critical state called the threshold potential (measuring about −60 mV), then sodium channels open completely. Sodium floods that part of the cell, which instantly depolarizes to an action potential of about +55 mV. Depolarization activates sodium channels in adjacent parts of the membrane, so that the impulse moves along the fibre.

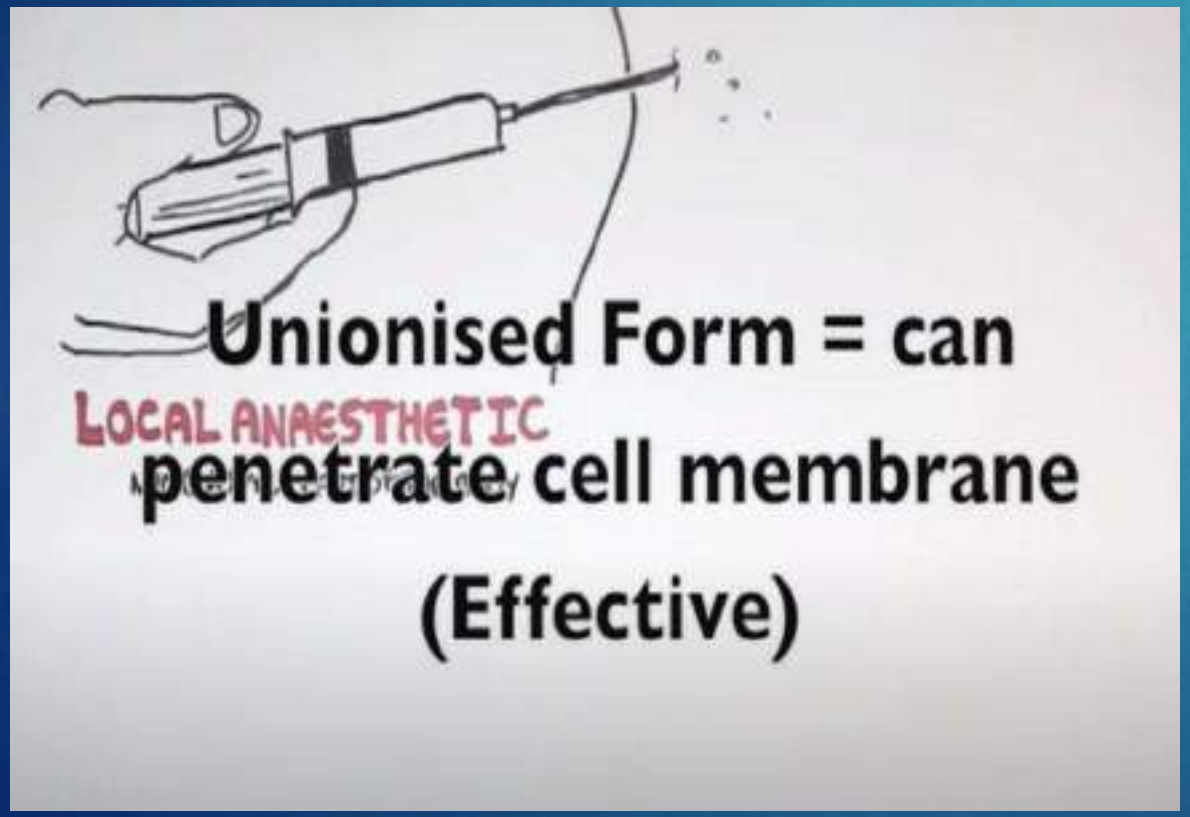
Composition

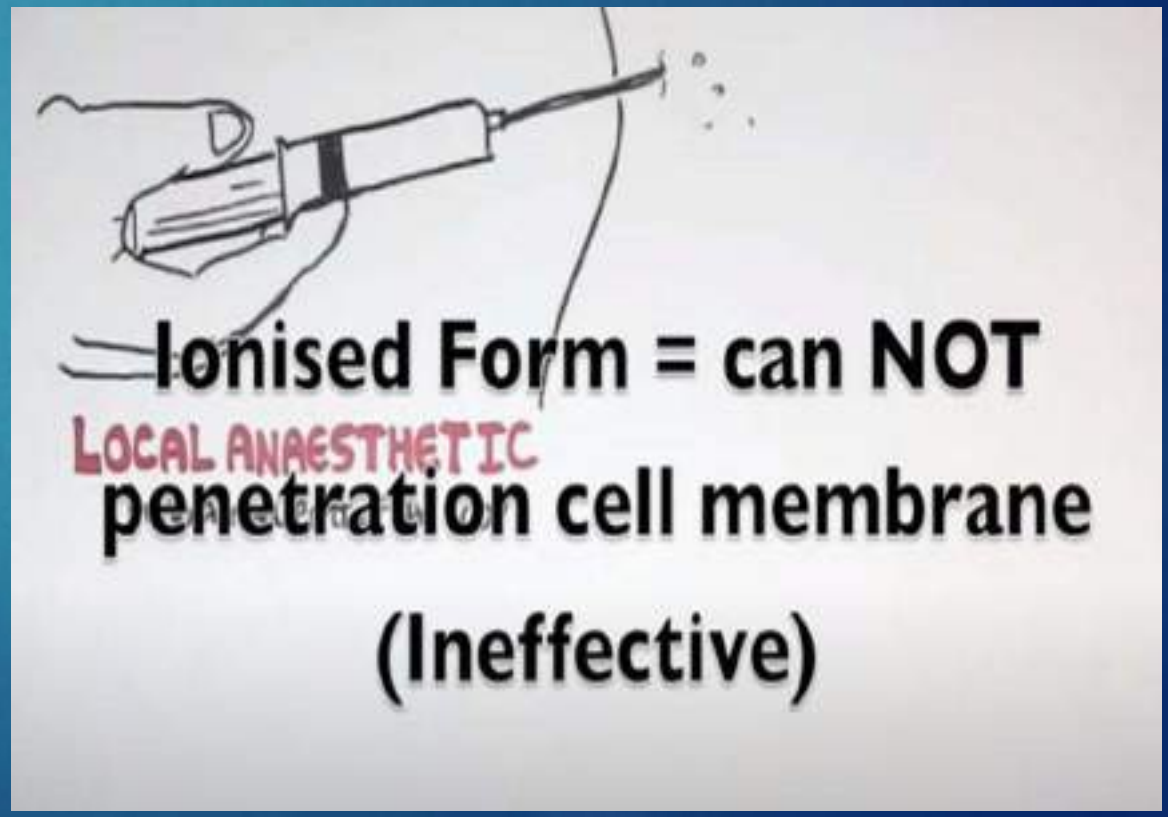
- **Local anaesthetic solution contains:**
- **1. Local anaesthetic agent.**
  - Ester linkage- procaine,cocaine,tetracaine
  - Amide linkage-Lignocaine, prilocaine, bupivacaine , mepivacaine
- **2. Vasoconstrictor**

Adrenaline- a synthetic substance similar to that secreted in human body.
Concentration from 1:50,000-1:250,000 (optimal 1:200,000)

Ionised and unionised

- **Local anesthetic is a weak base with a Pka 8-9 with unionized form can penetrate the cell membrane it is quite effective.**
- **But when enter physiological PH which is slightly acidic below 7 it will become ionized and so can not penetrate the cell membrane.**

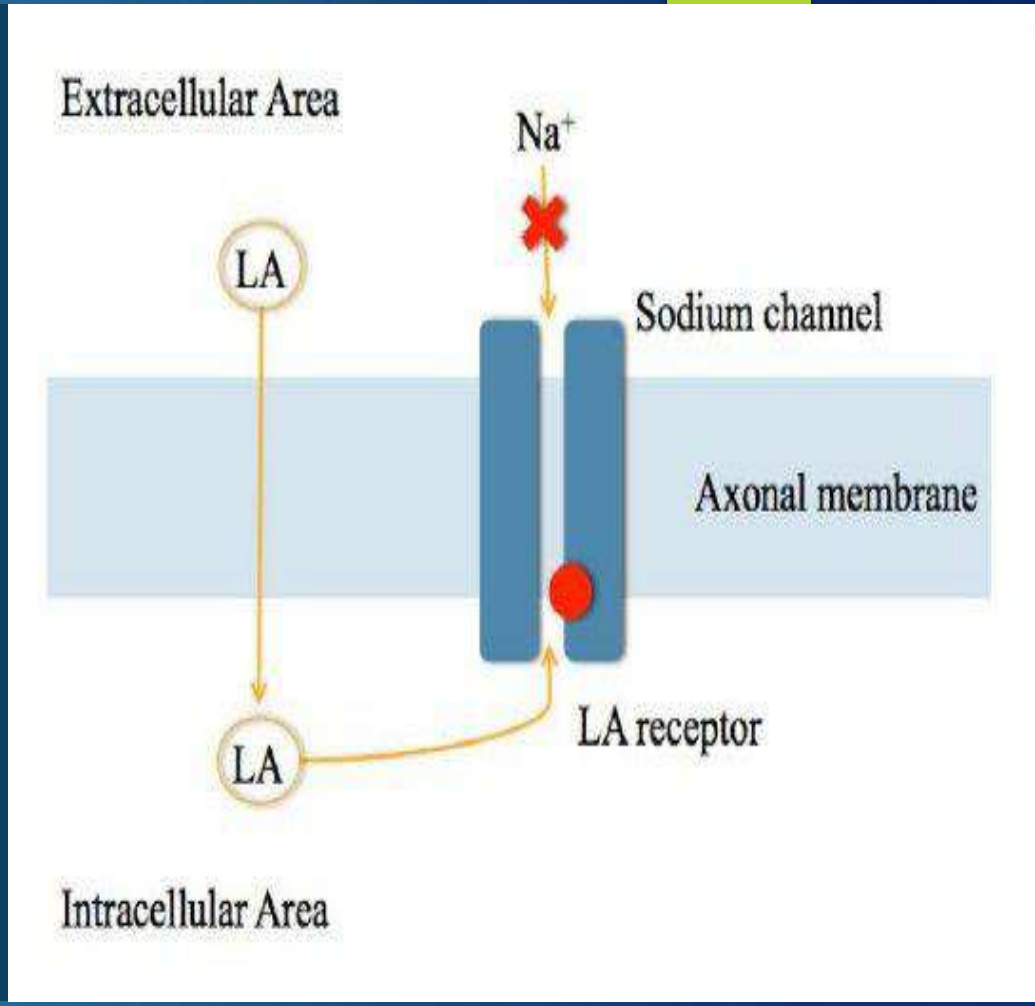




Mechanism of Action Local anesthetic drugs

- Mechanism of Action Local anesthetic drugs act mainly by inhibiting sodium influx through sodium-specific ion channels in the nerve cytoplasm
  - Sodium ions cannot flow in, so potassium ions cannot flow out, thereby preventing the depolarization of the nerve.

- To do this the anesthetic molecules must actually enter through the cell membrane of the nerve. This is where the differences in the time of onset and duration of the various local anesthetics lies.



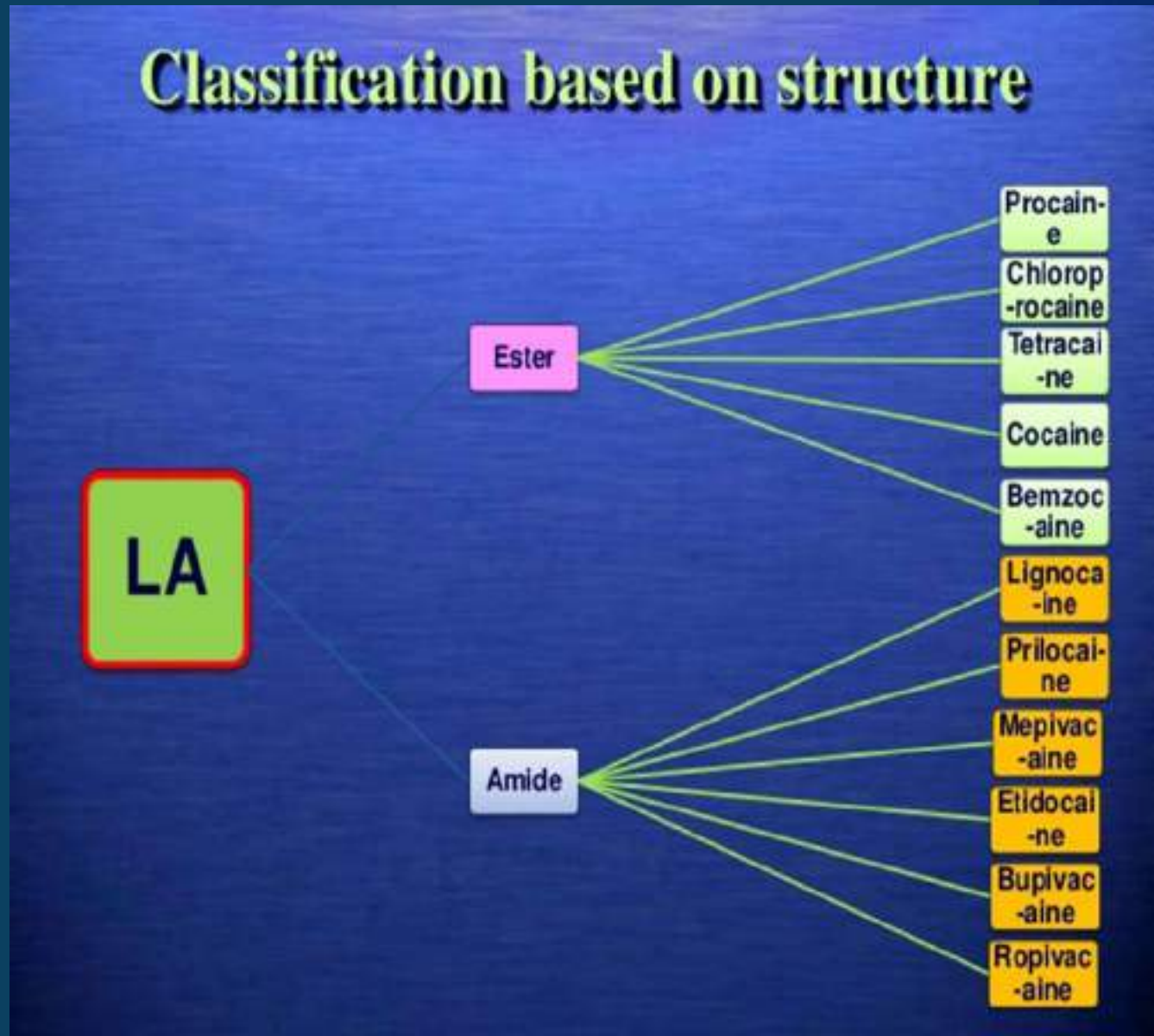
What are the drugs Classification

- **Injectable anaesthetic:**
- – Low potency, short duration – Procaine and Chlorprocaine
- – Intermediate potency and duration – Lidocaine (Lignocaine) and Prilocaine
- High potency and long duration – Tetracaine, Bupivacaine, Ropivacaine, Etidocaine, Mepivacaine and Dibucaine (Cinchocaine)
- **Surface anaesthetic:**
- Soluble – Cocaine, Lidocaine, Tetracaine and Benoxinate –
- Insoluble – Benzocaine, Butylaminobenzoate and Oxethazine •
- **Miscellaneous drugs**: – Clove oil, phenol, chlorpromazine and diphenhydramine etc.

A Local anesthetics are also classified according to Chemical Structure

- Ester-linked
  - Short acting
  - Metabolized in the plasma and tissue fluids
  - Excreted in urine
- Amide-linked
  - Longer acting
  - Metabolized by liver enzymes
  - Excreted in urine

- REMEMBER / All are weak Bases

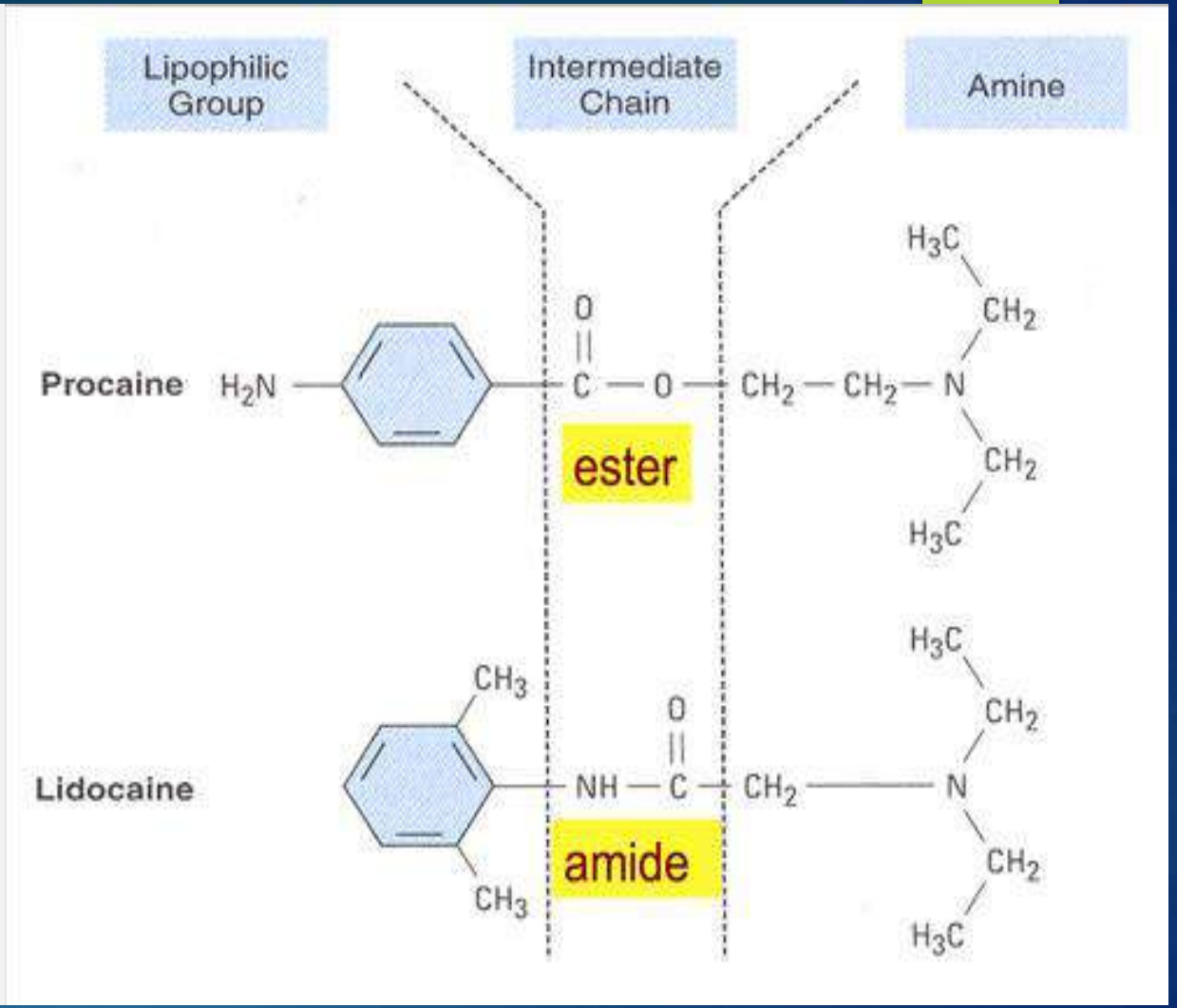


LA: Structure Activity Relationship (SAR)

General Structures There are two types of anesthetics amino-esters and amino-amides. All anesthetics are weak bases and are active in the cationic form

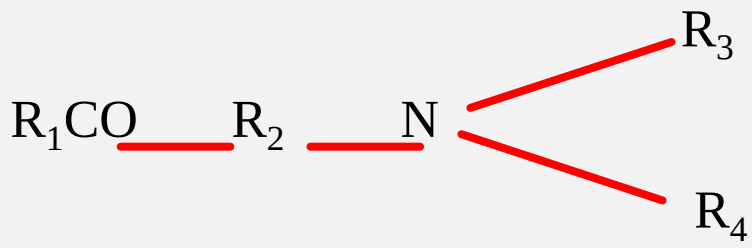
LOCAL ANESTHETICS :AMIDES VS. ESTERS

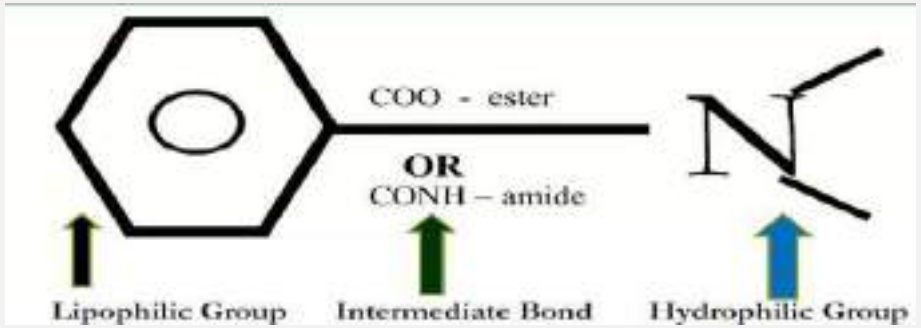
- ❑ Common structure
  - o Aromatic ring
  - o Tertiary amine
  - o Alkyl chain
- ❑ Linking bond
  - o Amide bond (see lidocaine)
  - o Ester bond (see procaine)



Chemistry of LAs

- Chemistry:
  - They are weak bases, insoluble in water
    - converted into soluble salts by adding Hcl for clinical use.
  - They are composed of three parts:
    - *Aromatic* (lipophilic) residue with acidic group R_1.
    - *Intermediate* aliphatic chain, which is either ester or amide link R_2.
    - Terminal *amino* (hydrophilic) group R_3 and R_4.





Chemistry of LAs

- Chemistry of LAs (Clinical Chemistry of LAs (Clinical significance)
  - Cross sensitivity (allergy with ESTER LINKAGE)
    - Occurs with drugs in the same chemical class
    - Esters are metabolized to common metabolite PABA
    - Allergy rarely occurs with amide linkage class
  - Biotransformation/duration of action
    - ESTERS are rapidly metabolized in the plasma by a cholinesterase
    - AMIDES are more slowly destroyed by liver microsomal P450 enzymes.

Physiochemical properties

These are very important for local anaesthetic activity.

Ionization:

- They are weak base and exist partly in an unionized and partly in an ionized form.
- The proportion depend on:
  - The pK_a or dissociation constant
  - The pH of the surrounding medium.
- Both ionizing and unionizing are important in producing local anaesthesia.

The difference between Esters and Amides.

Differences

1. Acid dissociation constant (pKa)
2. Onset of action
3. Chemical stability
4. Hypersensitivity
5. Drug interactions
6. Tissue toxicity

1. Acid dissociation constant (PKa)

Local anaesthetics → *Weak Base*

$$pH = pKa + \log(base/salt)$$

Henderson hasselbach equation

pH ≈ pKa

[base] ≈ [salt]

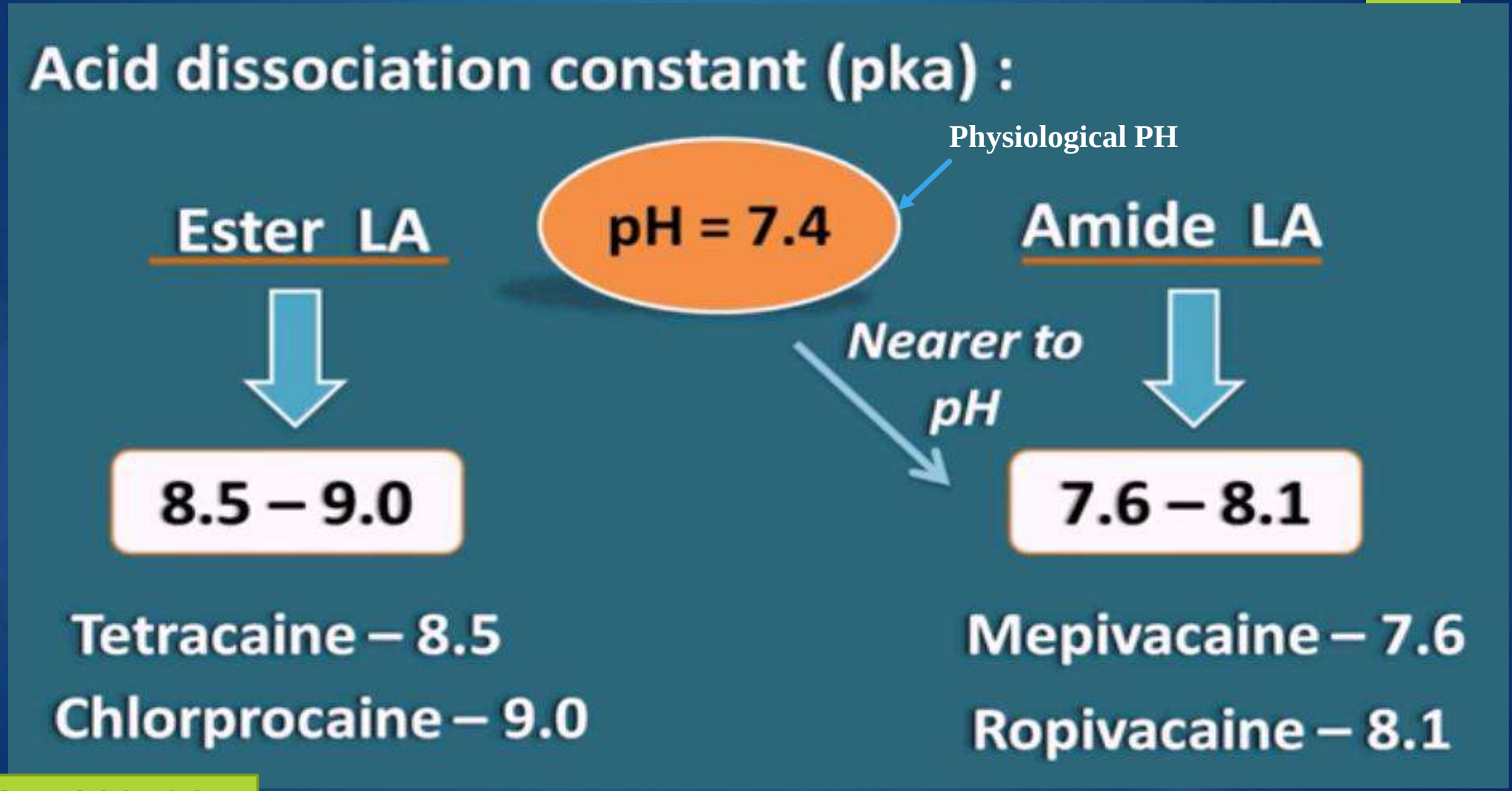
50 % Ionization

pH < pKa

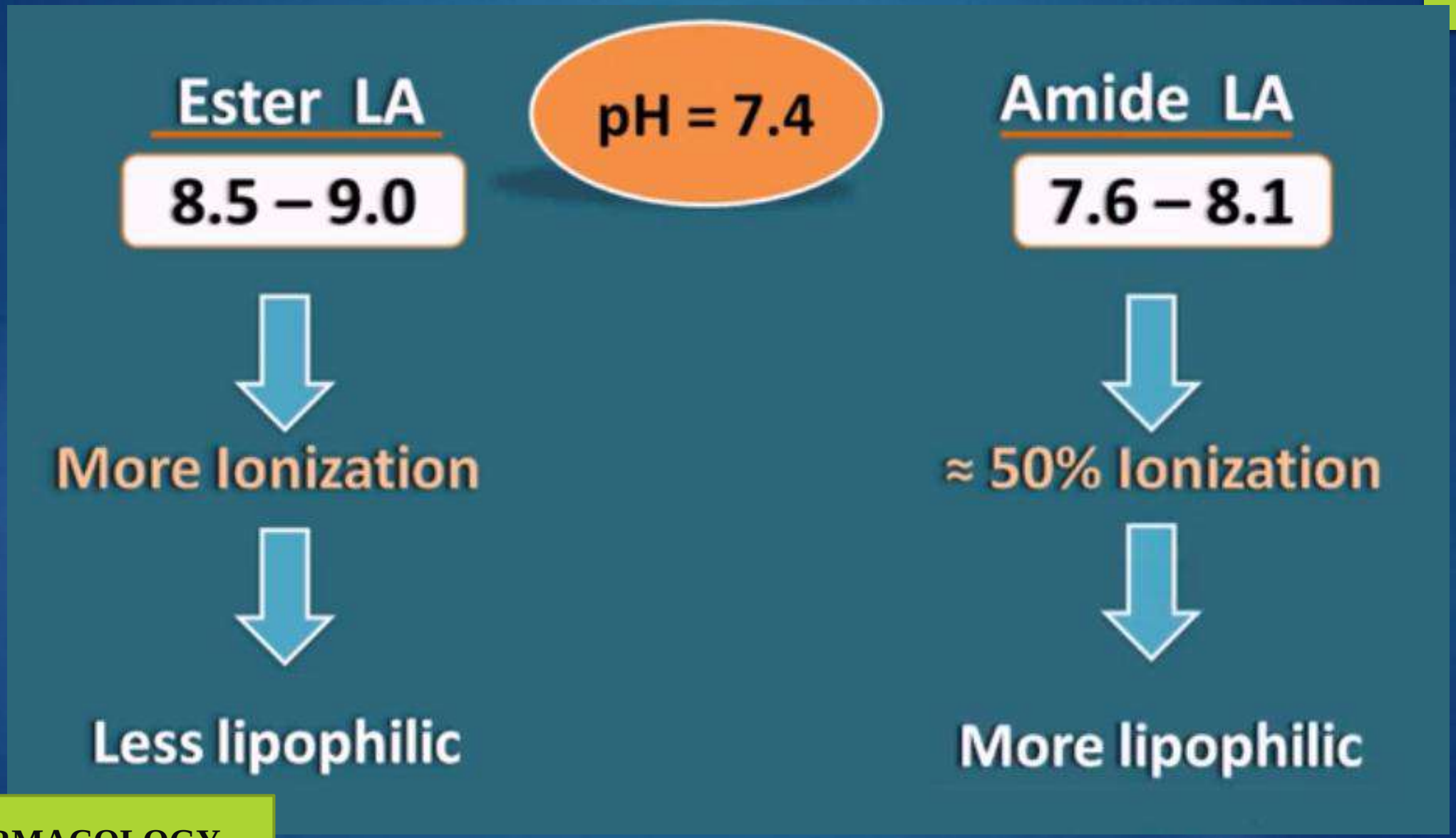
[base] < [salt]

More Ionization

1. Acid dissociation constant (PKa)



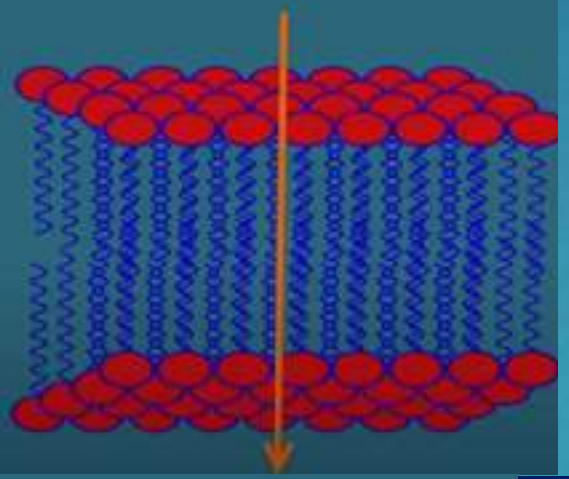
1. Acid dissociation constant (PKa)



2.Onset of action

2. Onset of action:

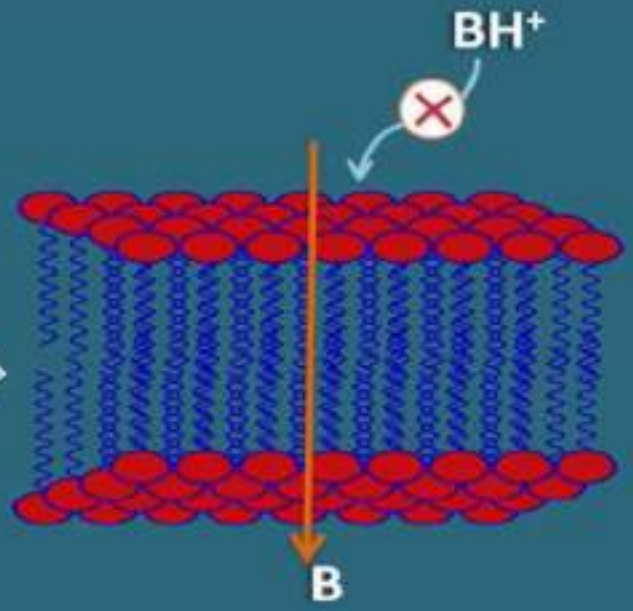
Drug transport across the membrane



2. Onset of action:

Drug transport across the membrane

Depends on pKa



Ester and Amide LA :

Ester LA		Amide LA
Tetracaine – 8.5	Fast	Mepivacaine – 7.6
Cocaine – 8.7		Lidocaine – 7.9
Procaine – 8.9		Prilocaine – 7.9
Chlorprocaine – 9.0		Ropivacaine – 8.1
	Slow	Bupivacaine – 8.1

When $pK_{a=}$ pH, there is equal proportion of ionized and unionized form of an agent are present in equal amounts.

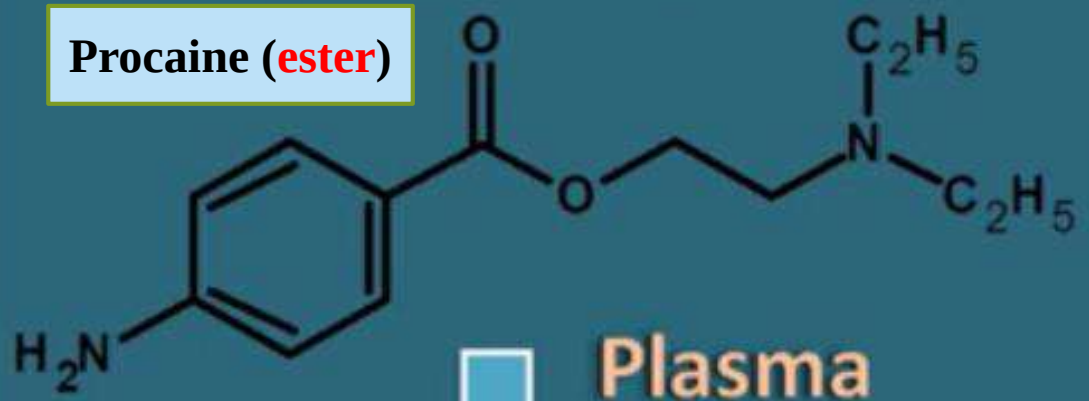
- The **lower the pK_a** , the more the unionized form, the greater the lipid solubility → **Higher the Onset**
- The **higher the pK_a** , the more the ionized form and the slower the lipid solubility → **Lower the Onset**

Note // Rate of diffusion across the nerve sheath and nerve membrane

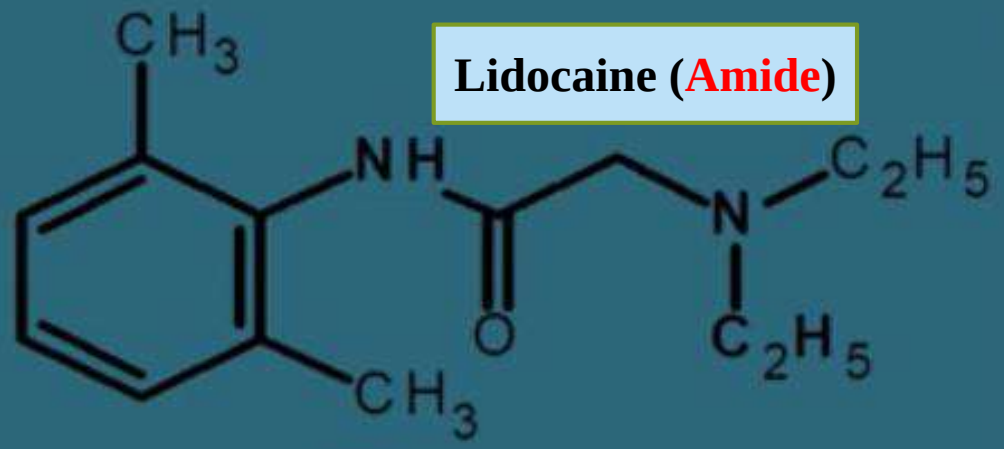
- As rate of diffusion across the nerve sheath and nerve membrane is related to the proportion of non-ionised drug,
- local anaesthetics with **low p*Ka*** have a **fast onset of action**,
- and local anaesthetics with **a high p*Ka*** have a **slow onset of action**.
- For example, lidocaine (p*K*a=7.8) has a fast onset in comparison with bupivacaine (p*K*a=8.1), because at pH 7.4 a greater proportion of lidocaine exists in the non-ionised form.
- Inflamed and infected tissue is more acidaemic, and therefore more local anaesthetic exists in the ionised form, reducing the amount of un-ionised drug available to cross the nerve and provide analgesia.
- The pH of tissue can be affected by adjuvants, for example, some clinicians add bicarbonate to speed the onset of epidural anaesthesia

3. Chemical stability :

Procaine (ester)



Lidocaine (Amide)



Plasma esterases

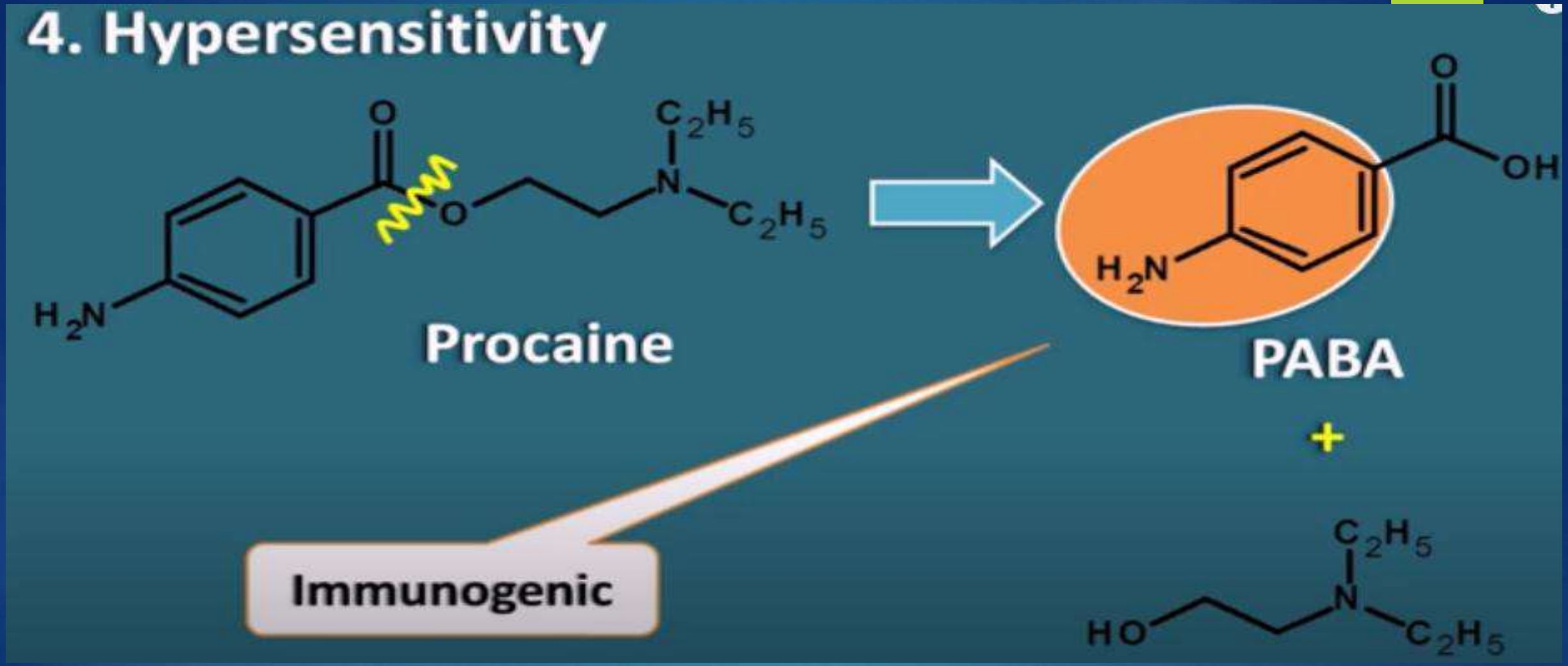
Less stable

Stable in Heat

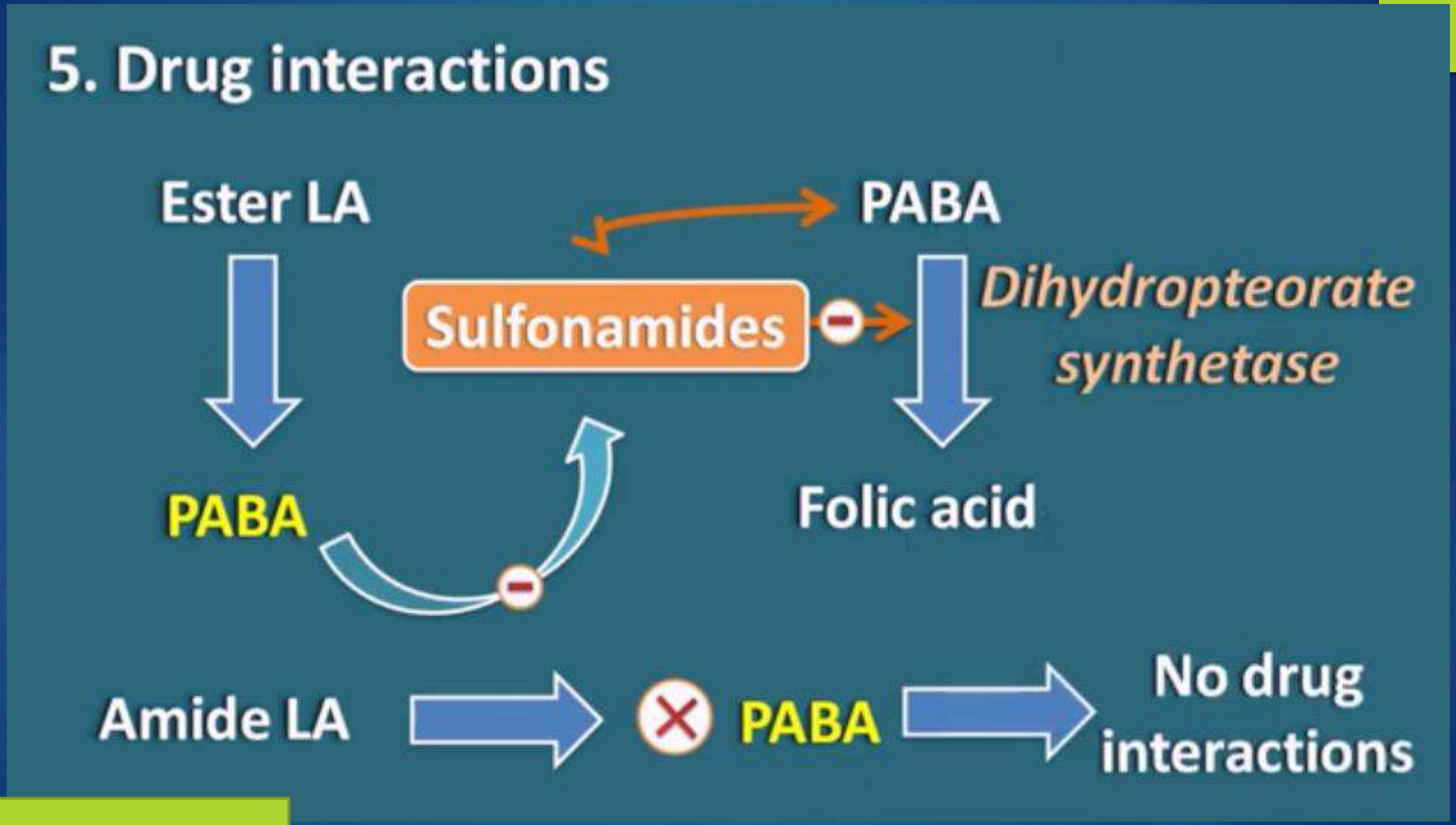
Stable in light

LIVER

More stable



Metabolism Procaine and benzocaine are metabolized to p- aminobenzoic acid (PABA), which has been associated with allergic reactions


5. Drug interactions
Ester LA
PABA
Sulfonamides
Dihydropteorate synthetase
PABA
Folic acid
Amide LA
PABA
No drug interactions

6. Tissue toxicity

Ester LA → Less systemic toxicity

Amide LA → More systemic toxicity → CNS

- Tinnitus
- Dizziness
- Blurred vision
- Restlessness
- Agitation
- Convulsions

Differences

- **Ester LA.**
- More Pka
- Less lipophilic
- Slow onset
- Less stable
- More allergic
- DI with sulfas
- Less toxic

- **Amide LA.**
- less Pka
- more lipophilic
- Fast onset
- more stable
- Less allergic
- No DI with sulfas
- more toxic

Summary

- Rapid Onset:
  - Low pK_a value– more unionized – Amides
  - Higher Partition coefficient – more lipid soluble
  - High PH
- Long duration of action:
  - High protein binding.
  - Low vasodilatation property.

Pharmacokinetic of LA

- **Pharmacokinetic of LA**
- Absorption: - Surface anesthetics from mucus membrane and abraded areas
- - Depends on Blood flow to the area, total dose and specific drug characteristics
- - Procaine has poor penetration in mucus membrane
- - Procaine is negligibly bound to plasma protein but amides are bound to alpha 1 acid glycoprotein
- **Distribution:**

- Widely distributed in the body: (lipophilic)

- Enters brain, heart, liver and kidney

- Followed by muscle and other viscera

-Amide are widely distributed & sequestered in fat.

-Ester short plasma t1/2 ; No enough time for distribution

Pharmacokinetic of LA

- **METABOLISM**
- **Ester type LA** - Ester in plasma butrylcholinesterase
- Hydrolysis by cholinesterase in plasma to PABA derivatives (4-Aminobenzoic acid (also known as para-aminobenzoic acid or PABA because the number 4 carbon in the benzene ring is also known as the para position) - pseudo cholinesterase or butrylcholinesterase
- Generally, short acting and low systemic toxicity
- Prolonged effects seen with genetically determined deficiency or altered esterase (cholinesterase inhibitors)
- **Amide type LA /**
- Bound to alpha1 acid glycoprotein
- Hydrolyzed by liver microsomal enzymes (P450)
- Longer acting & more systemic toxicity than esters
- High first pass metabolism on oral ingestion

Pharmacological base

- Pharmacological basis of the drug is Unionised form=can penetrate the cell membrane(effective) for example it can be absorbed by the body.

- Drug ionised (polar) = cannot penetrate the cell membrane (ineffective)(not very effective) for example can not absorbed by the body.

- The local anesthatic is weak base

Factors Affect the Reaction of Local Anesthetics

Lipid solubility

- All local anesthetics have weak bases. Increasing the lipid solubility leads to faster nerve penetration, block sodium channels, and speed up the onset of action.
- The more tightly local anesthetics bind to the protein, the longer the duration of onset action.
- Local anesthetics have two forms, ionized and nonionized. The nonionized form can cross the nerve membranes and block the sodium channels.
- So, the more nonionized presented, the faster the onset action.

pH influence

- Usually at range 7.6 – 8.9
- Decrease in pH shifts equilibrium toward the ionized form, delaying the onset action.
- Lower pH, solution more acidic, gives slower onset of action

Factors affect the reaction of local anesthetics

Vasodilation

- Vasoconstrictor is a substance used to keep the anesthetic solution in place at a longer period and prolongs the action of the drug
- vasoconstrictor delays the absorption which slows down the absorption into the bloodstream
- Lower vasodilator activity of a local anesthetic leads to a slower absorption and longer duration of action
- Vasoconstrictor used the naturally hormone called epinephrine (adrenaline). Epinephrine decreases vasodilator.

Side effects of epinephrine

- Epinephrine circulates the heart, causes the heart beat stronger and faster, and makes people feel nervous.

Toxicity

- Toxicity is the peak circulation levels of local anesthetics
- Levels of local anesthetic concentration administered to patients are varied according to age, weight, and health.
- Maximum dose for an individual is usually between 70mg to 500mg
- The amount of dose also varied based on the type of solution used and the presence of vasoconstrictor

Example:

---For adult whose weight is 150lbs and up, maximum dose Articaine and lidocaine is about 500mg

---For children, the dosage reduced to about 1/3 to ½ depending on their weight.

The doses are not considered lethal.

Some common toxic effects:

--light headedness ---shivering or twitching --seizures

--hypotension (low blood pressure) --numbness

Local Anesthetics - Toxicity

Tissue toxicity - Rare

- Can occur if administered in high enough concentrations (greater than those used clinically)
- Usually related to preservatives added to solution

Systemic toxicity - Rare

- Related to blood level of drug secondary to absorption from site of injection.
- Range from lightheadedness, tinnitus to seizures and CNS/cardiovascular collapse

Local Anesthetics - Allergy

- True allergy is very rare
- Most reactions are from ester class - ester hydrolysis (normal metabolism) leads to formation of PABA - like compounds
- Patient reports of "allergy" are frequently due to previous intravascular injections

Factors of circulation levels

- Factors of circulation levels are the rates of absorption, distribution, and metabolism.
- Absorption depends on the speed of administration and levels of the doses.
- Distribution allows absorption to occur in three phases. First, the drug occurs at highly vascular tissues in the lungs and kidneys. Then it appears less in vascular muscle and fat. Then the drug is metabolized.
- Metabolism involves in the chemical structure based on two classes, amide and ester as discussed earlier.
- Decreasing the potential toxicity resulted in rapid metabolism.

Local anesthetics - Duration

- Determined by rate of elimination of agent from site injected • Factors include.

1 - protein binding.

2 - lipid solubility.

3 - dose given.

4 - blood flow at site.

5 - addition of vasoconstrictors (does not reliably prolong all agents).

Local anesthetics – Duration and pKa Definition

pKa Definition

The dissociation of amphipathic local anaesthetics is determined by their pKa and the pH of the tissue into which they are injected. The pKa is the pH at which the ionised and un-ionised forms are present in equal amounts. For bases, such as local anaesthetics, the higher the pKa, the greater the ionised fraction in solution.

pKa is the negative log of the acid dissociation constant or Ka value.

A lower pKa value indicates a stronger acid.
That is, the lower value indicates the acid more fully dissociates in water.
pKa, the symbol for the acid dissociation constant at logarithmic scale.

Conclusion

Anesthetic	pKa	Onset	Duration (with Epinephrine) in minutes	Max Dose (with Epinephrine)
Procaine	9.1	Slow	45 - 90	8mg/kg – 10mg/kg
Lidocaine	7.9	Rapid	120 - 240	4.5mg/kg – 7mg/kg
Bupivacaine	8.1	Slow	4 hours – 8 hours	2.5mg/kg – 3mg/kg
Prilocaine	7.9	Medium	90 - 360	5mg/kg – 7.5mg/kg
Articaine	7.8	Rapid	140 - 270	4.0mg/kg – 7mg/kg

Local anaesthetics and infection

- The relevant feature of infected tissue is that it tends to be a more acidic environment than usual.
- As the pH is reduced the fraction of unionised local anaesthetic is reduced and consequently the effect is delayed and reduced.
- Infected tissue may also have an increased blood supply and hence more anaesthetic may be removed from the area before it can affect the neuron

local anaesthetic across the placenta

- **The rate and degree of diffusion of local anaesthetic across the placenta depends on:**
  - protein binding,
  - p*K*a,
  - maternal and fetal pH.
- Bupivacaine is highly protein bound (95%) and has an umbilical vein/maternal arterial ratio of 0.3. This contrasts with lidocaine (70%) with a ratio of 0.5–0.7. In prolonged labour, acidosis in the fetus can result in accumulation of local anaesthetic in the fetus by ion trapping. However, because of rapid hydrolysis, ester local anaesthetics do not cross the placenta in significant amounts.

LOCAL ANESTHESIA TECHNIQUE

❑ **Regional anesthesia**

- Field block
- Nerve block
- Local infiltration

❑ **Local Anesthesia Injection Technique**

- Supraperiosteal injections
- Intraligamentary injections
- Intraosseous injections
- Intraseptal injections
- Subperiosteal injections

❑ **Maxillary injection techniques**

- PSAN
- ASAN
- MSAN
- Nasopalatine nerve block
- Greater Palatine nerve block
- Maxillary nerve block

❑ **Mandibular Injection Techniques**

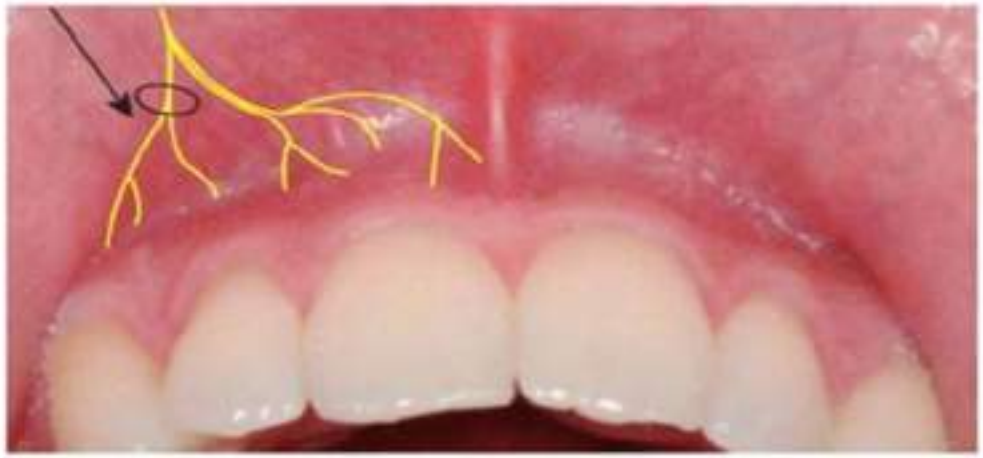
- IANB
- Mental nerve block
- Long buccal
- Gow–Gates nerve block
- Vazirani–Akinosi nerve block

REGIONAL ANAESTHESIA

- Regional anesthesia or "the nerve block" is a form of anesthesia in which only a part of the body is anesthetized ("made numb").

Field block

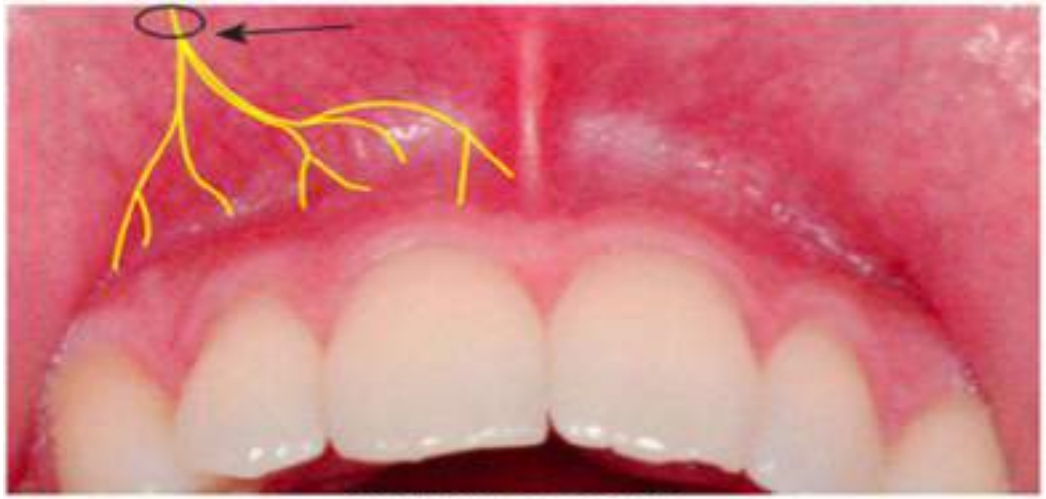
- In a field block, local anesthetic is infiltrated around the border of the surgical field, leaving the operative area undisturbed.
- Deposited in proximity to the larger nerve branches.
- Field block also may be considered when operating on the ear or lips. Eg. Gow-gates technique is a kind of field block.



FIELD BLOCK

❑Nerve block

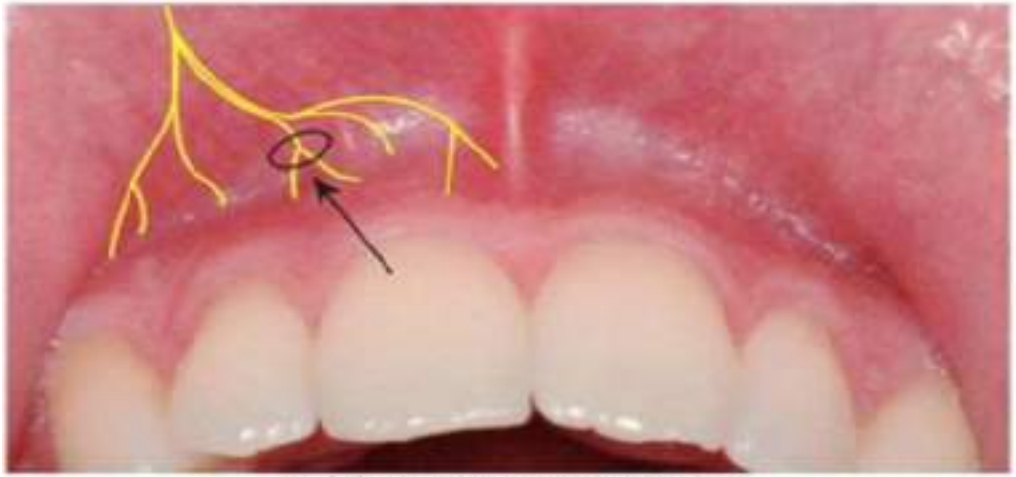
- In a nerve block, anesthetic solution is injected within close proximity to a main nerve trunk.
- Nerve blocks are used for pain treatment and management.
- Often a group of nerves, called a plexus or ganglion, that causes pain to a specific organ or body region can be blocked with the injection of medication into a specific area of the body. The injection of this nerve-numbing substance is called a nerve block. Eg. IANB
- Dose- 1.8- 2.0 mL



NERVE BLOCK

❑ Local infiltration

- Local infiltration is used when anesthesia is required in small areas (e.g., for repair of minor lacerations, skin biopsies).
- The anesthetic solution is infiltrated to the deep dermis, where the sensory plexus supplying the skin begins to branch.
- The amount of solution used depends on the area that needs to be infiltrated; however, extensive local infiltration is not recommended.
- Dose- 0.6- 1.0 mL

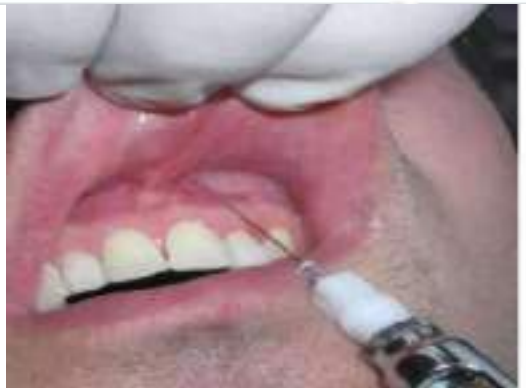


LOCAL INFILTRATION

Local Anaesthesia Technique

❑Supra Periosteal Injection

- Pulpal and Soft tissue anesthesia in maxillary anterior teeth.
- A short 25 or 27 gauge needle is recommended for this technique.
- Sometimes this injection technique is referred to as infiltration, but the solution is deposited near terminal branches of nerves so it is actually a type of field block.
- It is inserted at the height of the mucobuccal fold near the apex of the tooth to be treated. The bevel of the needle should be toward the bone



Supra Periosteal Injection

❑Intraligamentary injection/ PDL injection

- Dose- 0.2mL
- Depositing the LA solution within PDL through gingival sulcus.
- Provides 30-35 min of anesthesia.
- Indicated in patient with bleeding disorder& young handicapped patients .

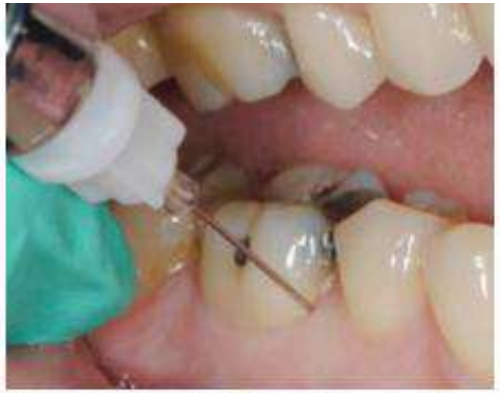


Figure 1. Intraoral photograph showing the position of the needle for the periodontal ligament anesthetic injection technique.

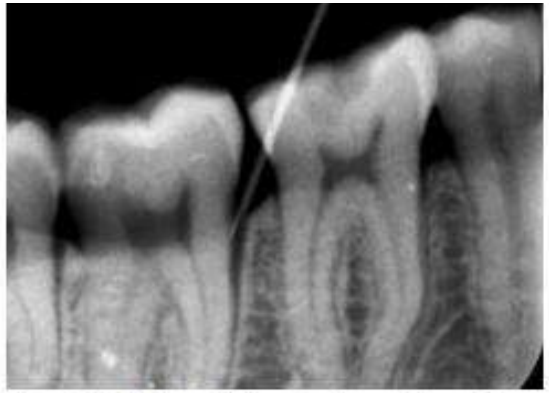
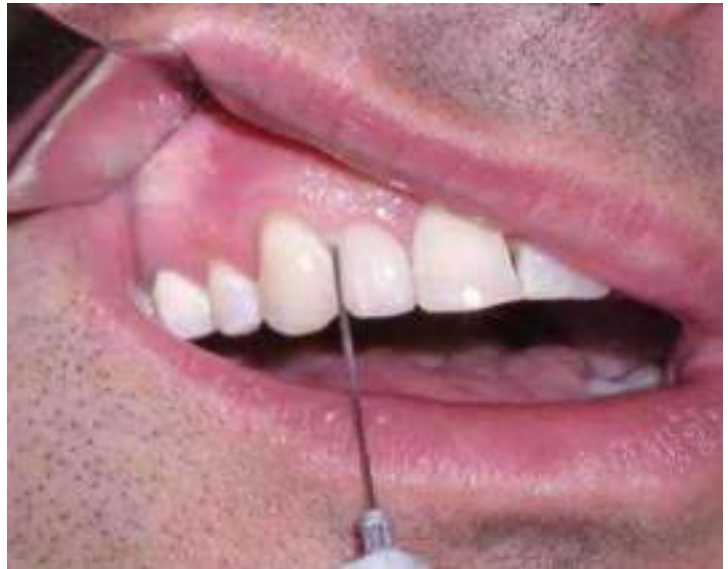


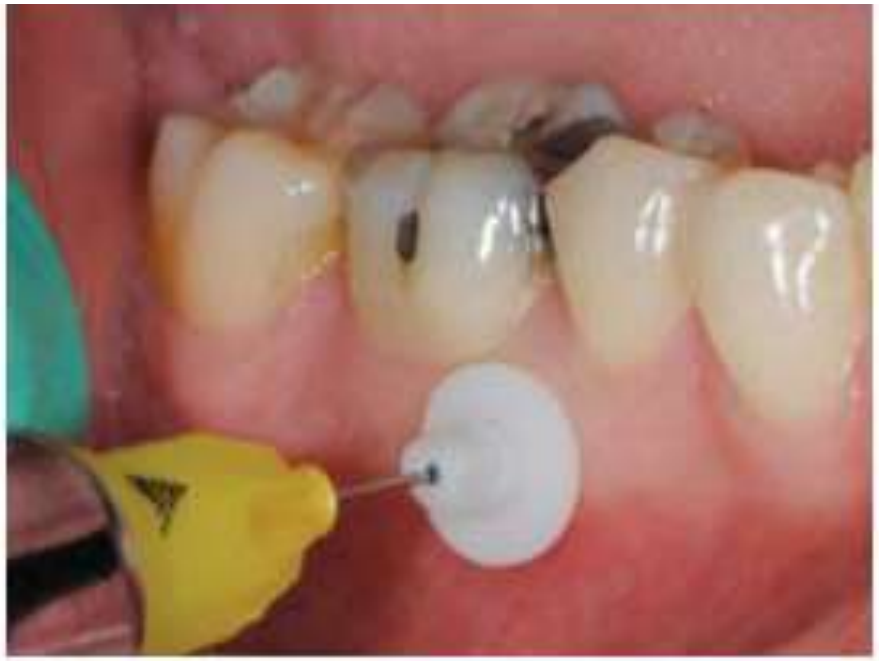
Figure 2. Radiograph showing the needle position for the periodontal ligament anesthetic injection technique.

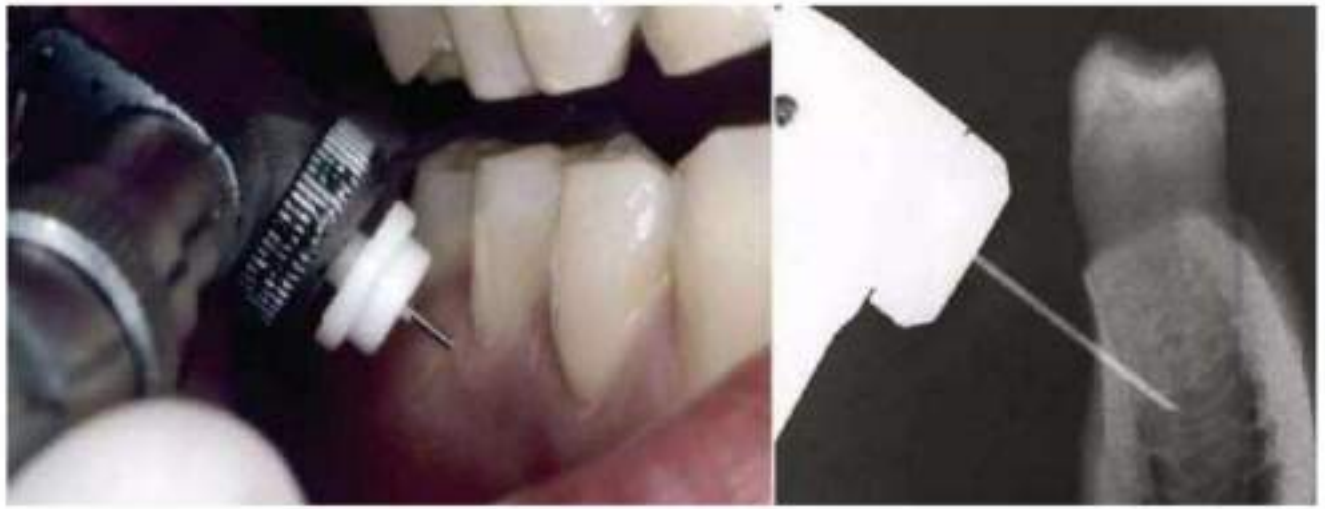
Figure 3. Illustration of the needle position for the periodontal ligament anesthetic injection technique.



❑ Intraosseous Injection

- Aim of intra-osseous anesthesia is to inject local anesthetic solution into cancellous bone adjacent to the apex of the tooth by piercing buccal gingiva and bone in relation to the tooth to be anesthetized.
- It can be used as a supplemental technique with mandibular nerve blocks to enhance deep pulpal anesthesia or as a primary technique so that patients do not experience numb lips or tongues postoperatively.

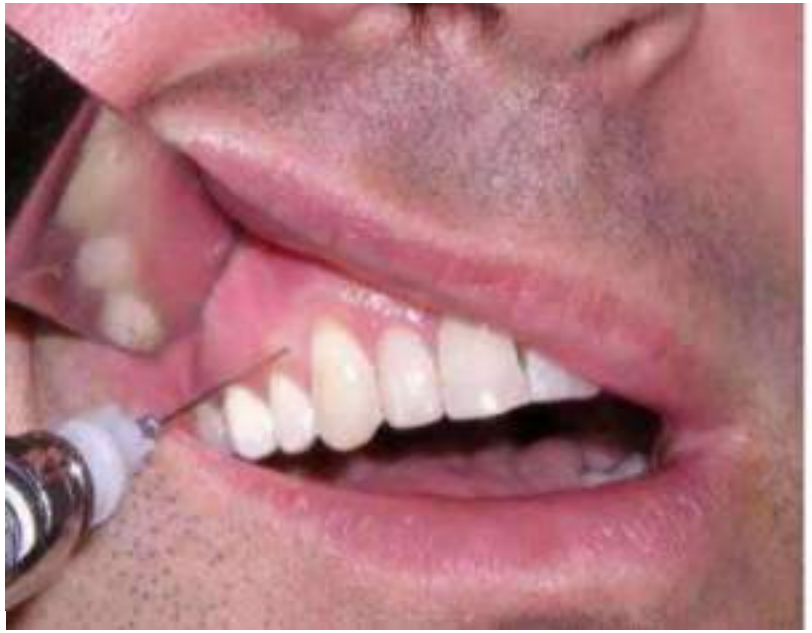




Intraosseous Injection

❑Intra Septal injections

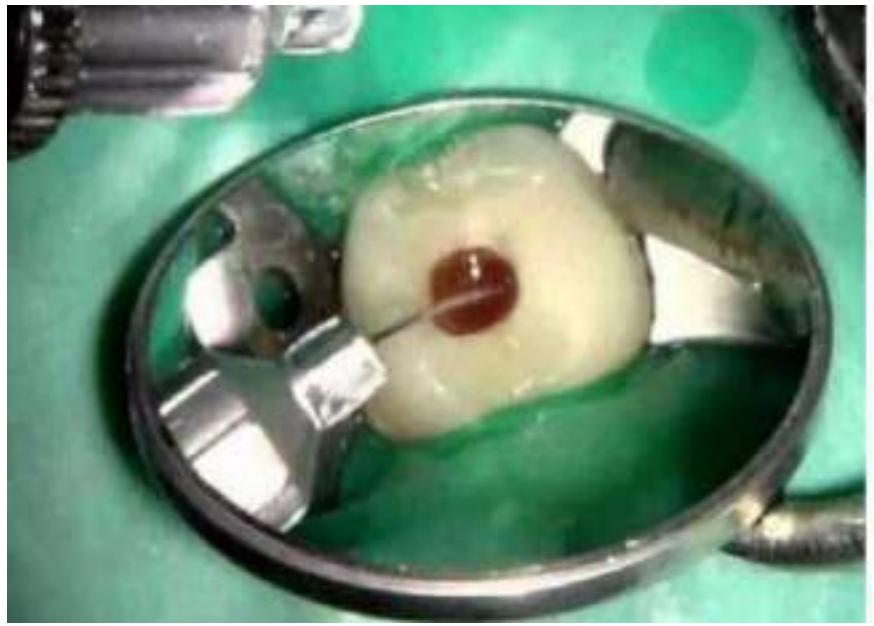
- Used for hemostasis, soft tissue anesthesia, and osseous anesthesia.
- Use a 27 gauge short needle and insert it into the papilla of the area to be anesthetized at an angle of 90º to the tissue.
- Slowly deposit 0.2 ml of solution.



Intraseptal Injection

❑Intra pulpal injection

- A 27-gauge short needle is inserted into the pulp chamber and wedged firmly into the root canal.
- A small volume (0.2-0.3mL) of local anesthetic is injected.
- While this technique may prove uncomfortable for the patient, it invariably works to provide effective pain control.
- In most cases, the duration is adequate to permit extirpation of the pulpal tissues.



Intrapulpal Injection

Factors that influence the choice of local anaesthetic technique

- 1) The area to be anaesthetized
- 2) Extent of the surgical procedures
- 3) Duration of the required anaesthesia

NEEDLE GAUGE

Gauge

- Refers to the diameter of the lumen of the needle; the smaller the number, the greater the diameter of the lumen.
- There is increased resistance to aspiration of blood through a thinner needle (eg,30-gauge) compared with a larger-diameter needle (eg,27- or 25-gauge).
- Needle deflection along the axis of the bevel and breakage must also be examined.
- The smaller the diameter of the needle, the more it deflects.

- Thirty-gauge needles deflect significantly, whereas 25-gauge needles essentially do not deflect at all. Likewise,25-gauge needles very rarely, if ever, break during an intraoral injection, and 99% of the needles that do break are 30-gauge needle

Thank you