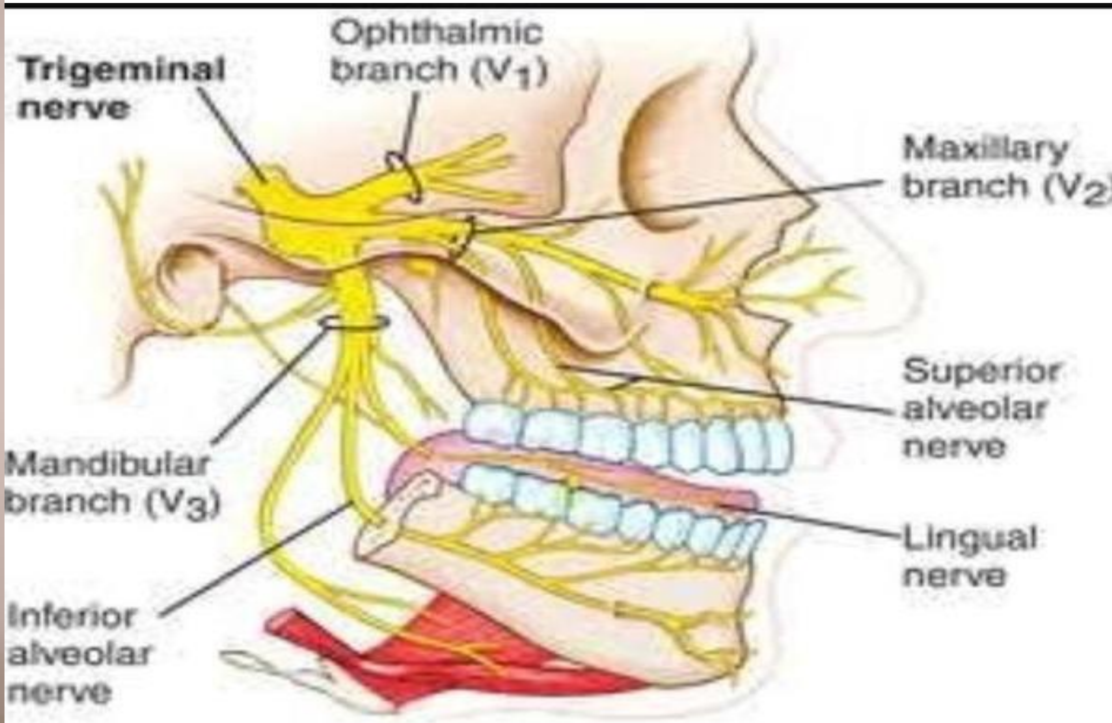
LOCAL ANAESTHESIA IN DENTISTRY



PROF.ABBAS AY TAHER

Introduction to local anesthesia :

Many dental procedures, such as tooth extraction, are both **painful** and **prolonged** and should be performed without pain by using a local anesthesia.

Local anesthesia has been defined as “**loss of sensation in a circumscribed area of the body caused by inhibition of the conduction process in the peripheral nerves”.**

An important feature of local anesthesia is that it produces this loss of sensation without inducing loss of consciousness. In this one major area, local anesthesia differs dramatically from general anesthesia.

Many methods are used to induce local anesthesia:

1. mechanical trauma (compression of tissues)
2. low temperature
3. anoxia
4. chemical irritants
5. neurolytic agents such as alcohol and phenol
6. chemical agents such as local anesthetics

However, only those methods or substances that induce a **transient** and **completely reversible state of anesthesia** have application in clinical practice.

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Brief history:

Local anesthesia as it is known today began when a German chemist **(Albert Niemann-1860)** successfully isolated the active principle of coca leaf, he named it **cocaine.** In 1898 professor Heinrich Braun introduced **procaine** as the first derivative of cocaine which is known as the first synthetic local anesthetic drug.

In 1940 the first modern local anesthetic agent was introduced and known as Lidocaine (trade name Xylocaine). It relieves pain during the dental surgeries and produces the desired anesthetic effect for several hours.

Terminology:

Pain: According to the World Health Organization (WHO) pain is defined as an *“unpleasant sensation that occurs from imminent tissue damage”*. From a physiological perspective, pain is a warning system. During dental treatment, patients will experience pain as something unpleasant.

Analgesia: loss of pain.

Anesthesia: loss of sensation (loss of pain and touch sensation).

☐ In dentistry, local anesthesia is used to permit the performance of surgery or other painful procedures with no pain.

Paresthesia: altered sensation (tingling), such as when a local anesthesia is starting to work or its effect is wearing off. Or when a damaged nerve is regenerated.

The effect of local anesthetics on nerve fiber has been shown to be dependent on:

1- The duration of exposure of local anesthesia

2- Concentration

3- Volume of the solution

Relative analgesia: is a sedation technique in which the patient remains conscious but mental relaxation is induced by inhalation of a mixture of nitrous oxide and oxygen.

Pain threshold:-a point at which the patient feels discomfort when exposed to painful stimuli.

The patient who feels minimal discomfort from painful stimuli is having high pain threshold

Many factors influence this response such as fear, apprehension and fatigue, all of which lower the pain threshold.

Premedication:-is the use of drugs to reduce a patient's apprehension prior to operative treatment.

Sedation techniques: involve the administration of a sedative to reduce anxiety in the conscious patient, usually, given by I.V route.

Properties (requirements) desirable for ideal local anesthetic:

1. It should not be irritating to the tissue to which it is applied.
2. It should not cause any permanent alteration of nerve structure.
3. Its systemic toxicity should be low.
4. The time of onset of anesthesia should be as short as possible.
5. The duration of action must be long enough to permit completion of the procedure.
6. It should be relatively free from producing allergic reactions.
7. It should be stable in solution and should readily undergo biotransformation in the body.
8. It should be sterile.

Of paramount importance is systemic toxicity, because all injectable and most topical local anesthetics are eventually absorbed from their site of **administration into the cardiovascular system**.

Several of the more potent injectable local anesthetics **(e.g., procaine, mepivacaine) prove to be relatively ineffective when applied topically to mucous membranes**.

To be effective as topical anesthetics, these **drugs must be applied in concentrations that prove to be locally irritating to tissues while increasing the risk of systemic toxicity.**

Lidocaine, on the other hand, is effective anesthetic when administered by injection or topical application in clinically acceptable concentrations.

Clinical duration of action does vary considerably among drugs and also among different preparations of the same drug, as well as by the type of injection administered (e.g., nerve block vs. supraperiosteal).
The duration of anesthesia necessary to complete a procedure is a major consideration in the selection of a local anesthetic.

Uses or indications of local anesthesia:

Local anesthesia is indicated in almost all the dental procedures, it is used to render the teeth, the supporting tissues and associated structures insensitive to painful stimuli.

A. Diagnostic:

Administration of LA can be a useful way of finding the source of patient’s pain. An example of this is the pain of pulpitis which can be very difficult for both the patient and the dentist to isolate because of its tendency to be referred to other parts of the mouth or face, so LA can differentiate between maxillary and mandibular sources, and even between individual upper teeth provided they are not immediately adjacent.
Another example is a patient with myofacial pain who is convinced that an upper tooth is causing the problem, LA may help this patient and the surgeon in this situation to eliminate the tooth as the cause of pain and may thus avoid unnecessary treatment. **4**

B. Therapeutic:

LA can constitute part of a treatment for painful conditions, for example, the use of block technique to eliminate the pain of dry socket. Blocks of the inferior alveolar nerve, mental nerve or infraorbital nerve can also be used for the treatment of trigeminal neuralgia when pain breakthrough.

C. Preoperative:

The provision of pain-free operation is by far the most common use of LA providing an effective and safe method for almost all outpatient dentoalveolar surgical procedures. It can in conjunction with sedation techniques allows more difficult procedures to be carried out without the additional risks of general anesthesia and this is particularly of value in patients with significant cardiovascular or airway diseases.

D. Adjunct to General Anesthesia:

LA is also given to patients undergoing oral and maxillofacial surgery under general anesthesia this serves the following purposes:

1. It reduces the depth of general anesthesia needed.
2. It reduces the arrhythmia during surgery when significant stimulation is taking place, for example when a tooth is being elevated.
3. It provides local homeostasis to the operative site.
4. Provides immediate postoperative analgesia.

E. Postoperative

After surgery with either local or general anesthesia the continuous effect of the anesthesia is the most beneficial way of reducing patient’s discomfort, it helps to reduce or even eliminate the need for a stronger systemic analgesic.

Contraindications of local anesthesia

These can be divided into two groups:

1- Absolute contraindications:

A. History of allergy to local anesthetic agent

Local anesthetic agents belong to the same chemical group should not be used. However, LA agents in the different chemical group can be used. For example, if the patient gives a history of allergy to an amide local anesthetic agent, an ester LA agent should be used.

B. History of allergy to other constituents of the local anesthetic solution

When the patient has a history of allergy to any of the constituents of the LA solution, it should be avoided, a different type of solution should be used.

2- Relative contraindications:

A. Uncooperative patient such as medically retarded patient.
B. Presence of acute inflammation or suppurative infection at the site of injection, to avoid the spread of infection (with the passages of the needle) from the abscess area to the deeper tissue.
C. Patient with a significant medical disease such as cardiovascular disease, hepatic dysfunction, renal dysfunction, clinical hyperthyroidism, etc.
D. Major surgical procedures (needs to be done under general anesthesia).

Advantages of local anesthesia

1. Less expensive than general anesthesia (GA).
2. No special preparation of the patient is needed as in GA.
3. No complicated apparatus is needed.
4. Less complication rate than GA.
5. The patient remains awake with no need for aftercare.
6. Can be used if GA is contraindicated (the patient is unfit for GA).
7. Anesthetist and other additional trained personnel are not required.
8. The technique is not difficult to master.

Neurophysiology :

Desirable Properties of Local Anesthetics

Local anesthesia has been defined as **loss of sensation in a circumscribed area of the body** caused by depression of excitation in nerve endings or inhibition of the conduction process in peripheral nerves. An important feature of local anesthesia is that it produces this loss of sensation without inducing **loss of consciousness**. In this one major area, local anesthesia differs dramatically from general anesthesia.

Most local anesthetics meet the **first two criteria**:
They are (relatively) **nonirritating to tissues** and **their effects are completely reversible**.
Of paramount importance is **systemic toxicity**, because all injectable and most topical local anesthetics are eventually **absorbed from their site of administration into the cardiovascular system**.

The potential toxicity of a drug is an **important factor in its consideration for use as a local anesthetic**.
Toxicity differs greatly among the local anesthetics currently in use. Although it is a **desirable characteristic**, not all local anesthetics **in clinical use today** meet the criterion of being effective, regardless of whether the drug is injected or applied topically.

Several of the more potent injectable local anesthetics (e.g., **procaine,mepivacaine**) prove to be relatively ineffective when applied **topically to mucous membranes**. To be effective as topical anesthetics, these drugs must be applied in concentrations that prove to be locally irritating to tissues, while increasing the risk of systemic toxicity. Dyclonine, a potent topicalanesthetic, is not administered by injection because of its **tissue-irritating properties**.

Lidocaine and tetracaine, on the other hand, are effective anesthetics when administered by **injection or topical application** in clinically acceptable concentrations.
The last factors—rapid onset of action and adequate duration of clinical action—are met satisfactorily by most of the clinically effective local anesthetics in use today.

Clinical duration of action differs considerably among drugs and also among different preparations of the same drug, as well as by the type of injection administered (e.g., **nerve block** vs. **supraperiosteal**). The duration of anesthesia necessary to complete a procedure is a major consideration in the selection of a local anesthetic. In addition to these **qualities, Bennett** lists other desirable **properties of an ideal local anesthetic:**

1. It should have potency sufficient to give complete anesthesia without the use of harmful concentrated solutions.
2. It should be relatively free from producing allergic reactions.
3. It should be stable in solution and should readily undergo biotransformation in the body.
4. It should be sterile or capable of being sterilized by heat without deterioration.

No local anesthetic in use today satisfies all these criteria; however, all anesthetics do meet most of them. **Research con**tinues in an effort to produce newer drugs that possess a maximum of desirable factors and a minimum of negative ones

Fundamentals of Impulse Generation and Transmission

The discovery in the late 1800s of a group of chemicals with the ability to prevent pain without inducing loss of consciousness was a major step in the advancement of the medical and dental professions.

For the first time, **medical and dental procedures could be performed easily and in the absence of pain in conscious patients**, a fact that is taken for granted by contemporary medical and dental professionals and their patients.

The concept behind the actions of local anesthetics is **simple:** they **prevent both the generation** and the conduction of a nerve impulse. In effect, local anesthetics set up a **chemical roadblock** between the source of the impulse (e.g., **the scalpel incision in soft tissues**) and the **brain**.

The **aborted impulse**, prevented from reaching the brain, cannot be interpreted by the patient as painز

How, in fact, **do local anesthetics**, the most used drugs in dentistry, function to **abolish or prevent pain?**

Pain Processes:

Brain systems involved in processing pain-related information. There are four major processes: transduction, transmission, modulation, and perception.

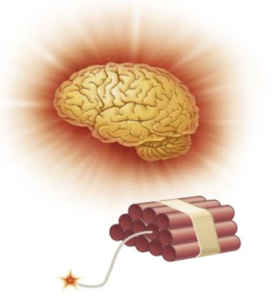
1-Transduction refers to the processes by which tissue-damaging stimuli activate nerve endings. A-Mechanical (pressure, pinch), B-Heat, and C-Chemical.

2-Transmission refers to the relay functions by which the message is carried from the site of tissue injury to the brain regions underlying perception.

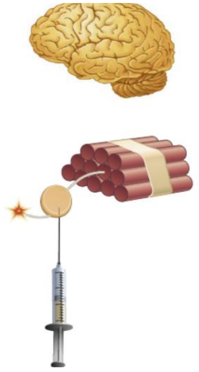
3-Modulation is a recently discovered neural process that acts specifically to reduce activity in the transmission system.

4-Perception is the subjective awareness produced by sensory signals; it involves the integration of many sensory messages into a coherent and meaningful whole.

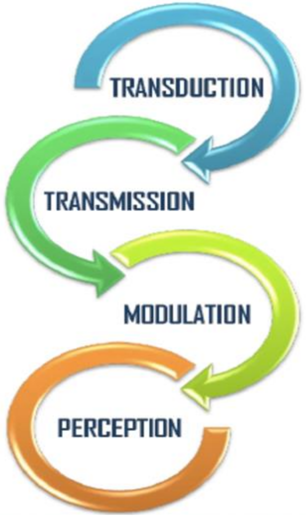
Perception is a complex function of several processes, including attention expectation, and interpretation.



The fuse is lit and the flame reaches the dynamite; an explosion occurs, and the patient experiences pain.

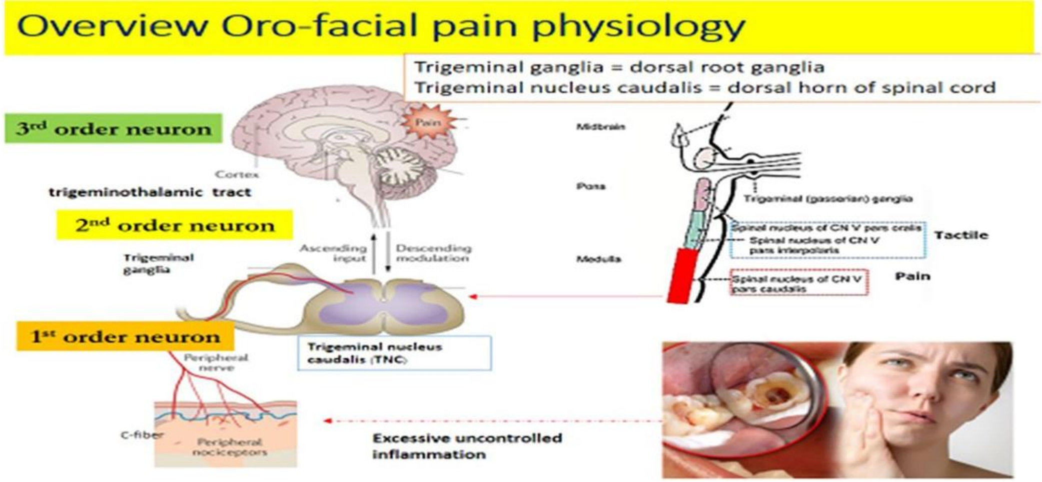


Local anesthetic is placed at some point between the pain stimulus and the brain (dynamite). The nerve impulse travels up to the point of local anesthetic application and then “dies,” never reaching the brain, and pain does not occur.



The pain pathway

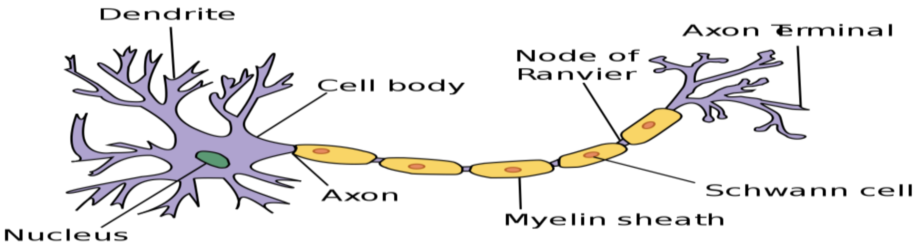
The pain pathway is mostly transmitted via **myelinated Aδ (sharp or stabbing pain) and unmyelinated C nerve fibers** (slow, dull, aching, or burning pain) of the trigeminal nerve, which supplies sensation to the teeth and gums via many divisions and branches



Overview Oro-facial pain physiology

Trigeminal ganglia = dorsal root ganglia
Trigeminal nucleus caudalis = dorsal horn of spinal cord

The Neuron:



The neuron, or nerve cell, is the structural unit of the nervous system. It is able to transmit messages between the central nervous system (CNS) and all parts of the body. There are two basic types of neuron: sensory (afferent) and motor (efferent). The basic structure of these two neuronal types differs significantly .Sensory neurons capable of transmitting the sensation of pain consist of three major portions.

The *peripheral process* (also known as the *dendritic zone*), composed of an arborization of free nerve endings, is the most distal segment of the sensory neuron. These free nerve endings respond to stimulation produced in the tissues in which they lie, provoking an impulse that is transmitted centrally along the axon. The *axon* is a thin cable-like structure that may be quite long (the giant squid axon has been measured at 100 to 200 cm). At its mesial central) end is an arborization similar to that seen in the peripheral process.

However, in this case the arborization forms synapses with various nuclei in the CNS to distribute incoming (sensory) impulses to their appropriate sites within the CNS for interpretation.

The *cell body* is the third part of the neuron. In the sensory neuron described here, the cell body is located at a distance from the axon, the main pathway of impulse transmission in this nerve.

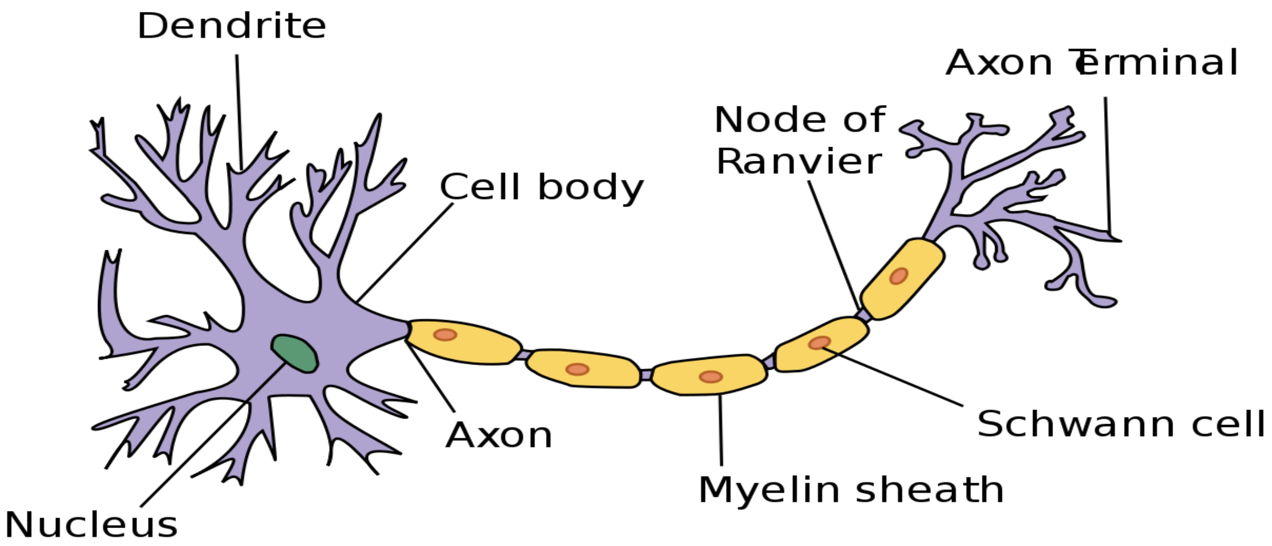
The cell body of the sensory nerve therefore is not involved in the process of impulse transmission, its primary function being to provide vital metabolic support for the entire .

Nerve cells that conduct impulses from the CNS toward the periphery are termed ***motor neurons*** and are structurally different from the **sensory neurons** just described **in that their cell body is interposed** between the axon and **dendrites.** In motor neurons the cell body not only is an integral component of the impulse transmission system but also provides metabolic support for the cell. Near its termination, the axon branches, with each branch ending as a bulbous axon terminal (or bouton). Axon terminals synapse with muscle cells (

The Axon:

The single nerve fiber, the axon, is a long cylinder of neural cytoplasm (axoplasm) encased in a thin sheath, the nerve membrane, or axolemma. Neurons have a cell body and a nucleus, as do all other cells; however, neurons differ from other cells in that they have an axonal process from which the cell body may be at a considerable distance.

The axoplasm, a gelatinous substance, is separated from extracellular fluids by a continuous nerve membrane. In some nerves, this membrane is itself covered by an insulating lipid-rich layer of myelin.

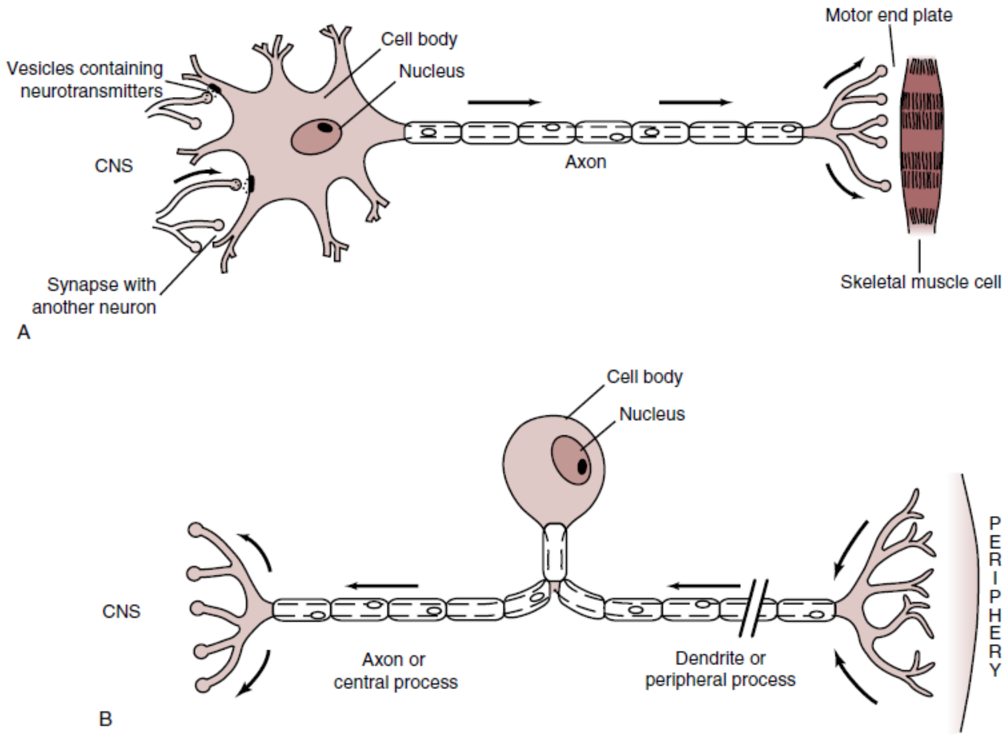


Both sensory nerve excitability and conduction are attributable to changes that develop within the nerve membrane.

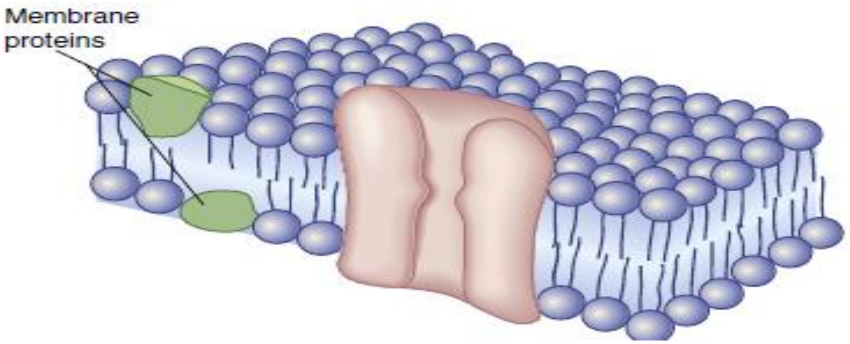
The cell body and the axoplasm are not essential for nerve conduction. They are important however, for the metabolic support of the nerve membrane is probably derived from the axoplasm.

The nerve (cell) membrane itself is approximately **70 to 80 Å thick.** (An angstrom is 1/10,000 of a micrometer.) presents a currently acceptable configuration. block

All biological membranes are organized to block membranes are organized to the diffusion of water-soluble molecules, to be selectively permeable to certain molecules via specialized pores or channels, and to transduce information through protein receptors responsive to chemical or physical stimulation by neurotransmitters or hormones (chemical) or light, vibration, or pressure (physical). The membrane is described as a flexible nonstretchable structure consisting of two layers of lipid molecules (bilipid layer of phospholipids) and associated proteins, lipids, and carbohydrates. The lipids are oriented with their hydrophilic (polar) ends facing the outer surface and their hydrophobic (nonpolar) ends projecting to the middle of the membrane.



A, Multipolar motor neuron.
B, Unipolar sensory neur



Classification of Peripheral Nerves According to Fiber Size and Physiologic Properties

Fiber Class	Subclass	Myelin	Diameter (µm)	Conduction Velocity (m/s)	Location	Function
A	Alpha	+	6–22	30–120	Afferent to and efferent from muscles and joints	Motor, proprioception
	Beta	+	6–22	30–120	Afferent to and efferent from muscles and joints	Motor, proprioception
	Gamma	+	3–6	15–35	Efferent to muscle spindles	Muscle tone
	Delta	+	1–4	5–25	Afferent sensory nerves	Pain, temperature, touch
B		+	<3	3–15	Preganglionic sympathetic	Various autonomic functions
C	sympatheticC	–	0.3–1.3	0.7–1.3	Postganglionic sympathetic	Various autonomic functions
	dorsal root gammaC	–	0.4–1.2	0.1–2.0	Afferent sensory nerves	Various autonomic functions; pain, temperature, touch

From Berde CB, Strichartz GR: Local anesthetics. In Miller RD, editor: *Anesthesia*, ed 5, Philadelphia, 2000, Churchill Livingstone, pp 491–521.

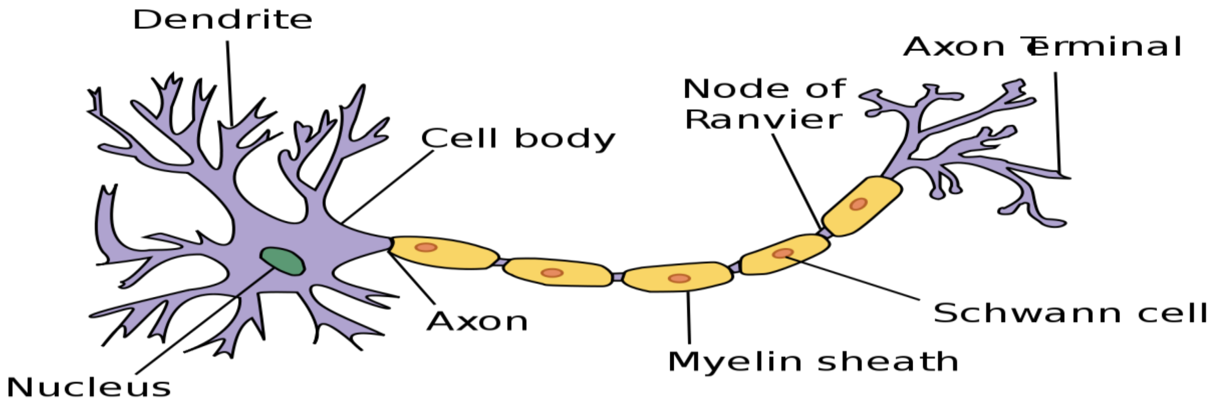
Transmission of Nerve Impulses

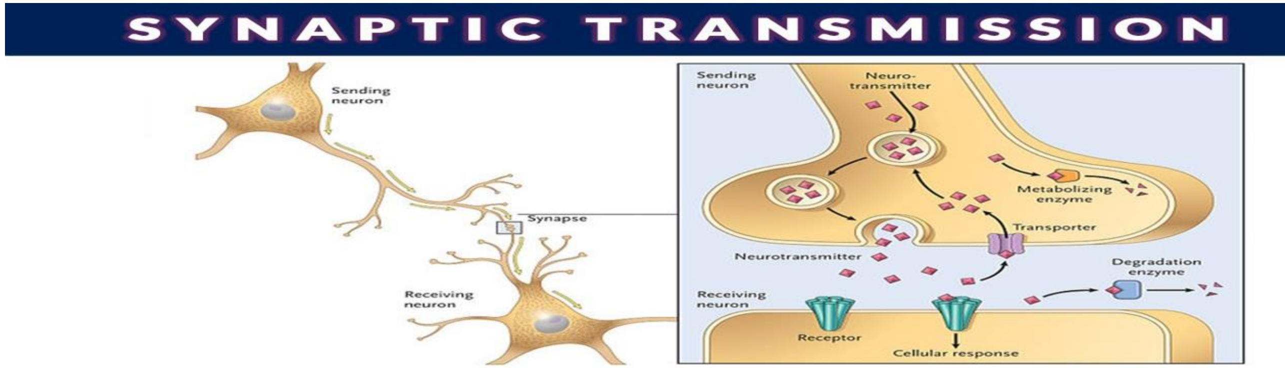
The transmission of a nerve impulse along a neuron from one end to the other occurs as a result of electrical changes across the membrane of the neuron. The membrane of an unstimulated neuron is polarized—that is, there is a difference in electrical charge between the outside and inside of the membrane. The inside is negative with respect to the outside .

Polarization is established by maintaining an excess of sodium ions (Na^+) on the outside and an **excess of potassium ions (K^+) on the inside**. A certain amount of Na^+ and K^+ is always leaking across the membrane through leakage channels, but Na^+/K^+ pumps in the membrane actively restore the ions to the appropriate side.

The main contribution to the **resting membrane potential** (a polarized nerve) is the difference in permeability of the resting membrane to **potassium ions versus sodium ions**. The resting membrane is much more **permeable to potassium ions** than to **sodium ions** **resulting in slightly more net potassium ion diffusion** (from the inside of the neuron to the outside) than sodium ion diffusion (from the outside of the neuron to the inside) causing the slight difference in **polarity right along** the membrane of the axon.

In addition to crossing the membrane through leakage channels, ions may cross through **gated channels.** Gated channels open in response to neurotransmitters, changes in membrane potential, or other stimuli





Electrophysiology of Nerve Conduction

A description of electrical events that occur within a nerve during the conduction of an impulse follows. Subsequent sections describe the precise mechanisms for each of these steps. A nerve possesses a resting potential (Fig. 1.7, step 1). This is a negative electrical potential of −70 mV that exists across the nerve membrane, produced by differing concentrations of ions on either side of the membrane .The interior of the nerve is negative relative to the exterior.

Step 1

A stimulus excites the nerve, leading to the following sequence of events:

1. An initial phase of slow depolarization. The electrical potential within the nerve becomes slightly less negative
2. When the falling electrical potential reaches a critical level, an extremely rapid phase of depolarization results.

This is termed *threshold potential*, or *firing threshold*.

Step 2

After these steps of depolarization, repolarization occurs .The electrical potential gradually becomes more negative inside the nerve cell relative to outside until the original resting potential of −70 mV is again achieved.

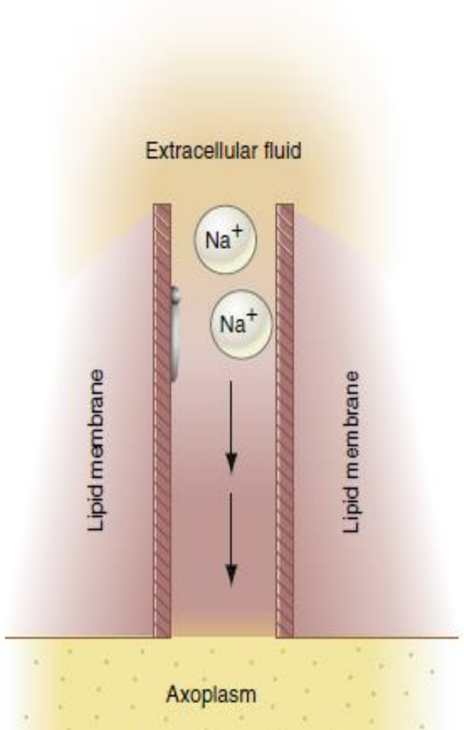
The entire process (steps 1 and 2) requires 1 millisecond:

depolarization **(step 1)** takes 0.3 milliseconds; repolarization**(step 2)** takes 0.7 milliseconds. Electrochemistry of Nerve Conduction .

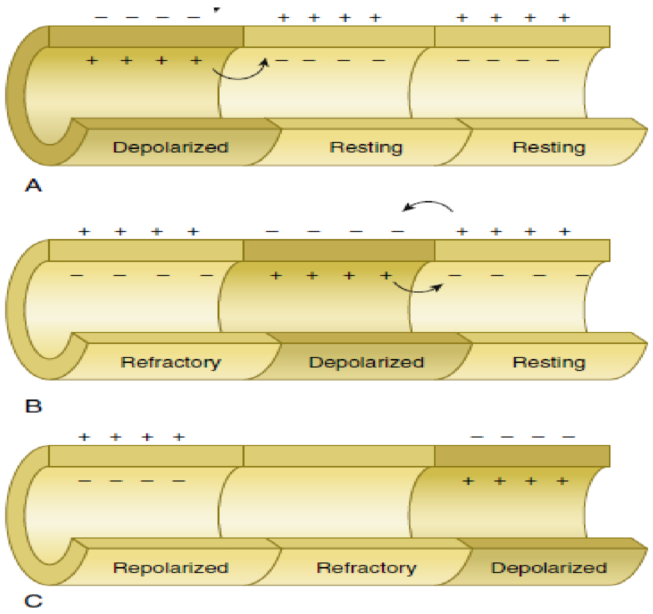
The preceding sequence of events depends on two important factors: the concentrations of electrolytes in the axoplasm **(interior of the nerve cell) and extracellular fluids** extracellular fluids, and the permeability of the nerve membrane to sodium and potassium ions

Electrochemistry of Nerve Conduction

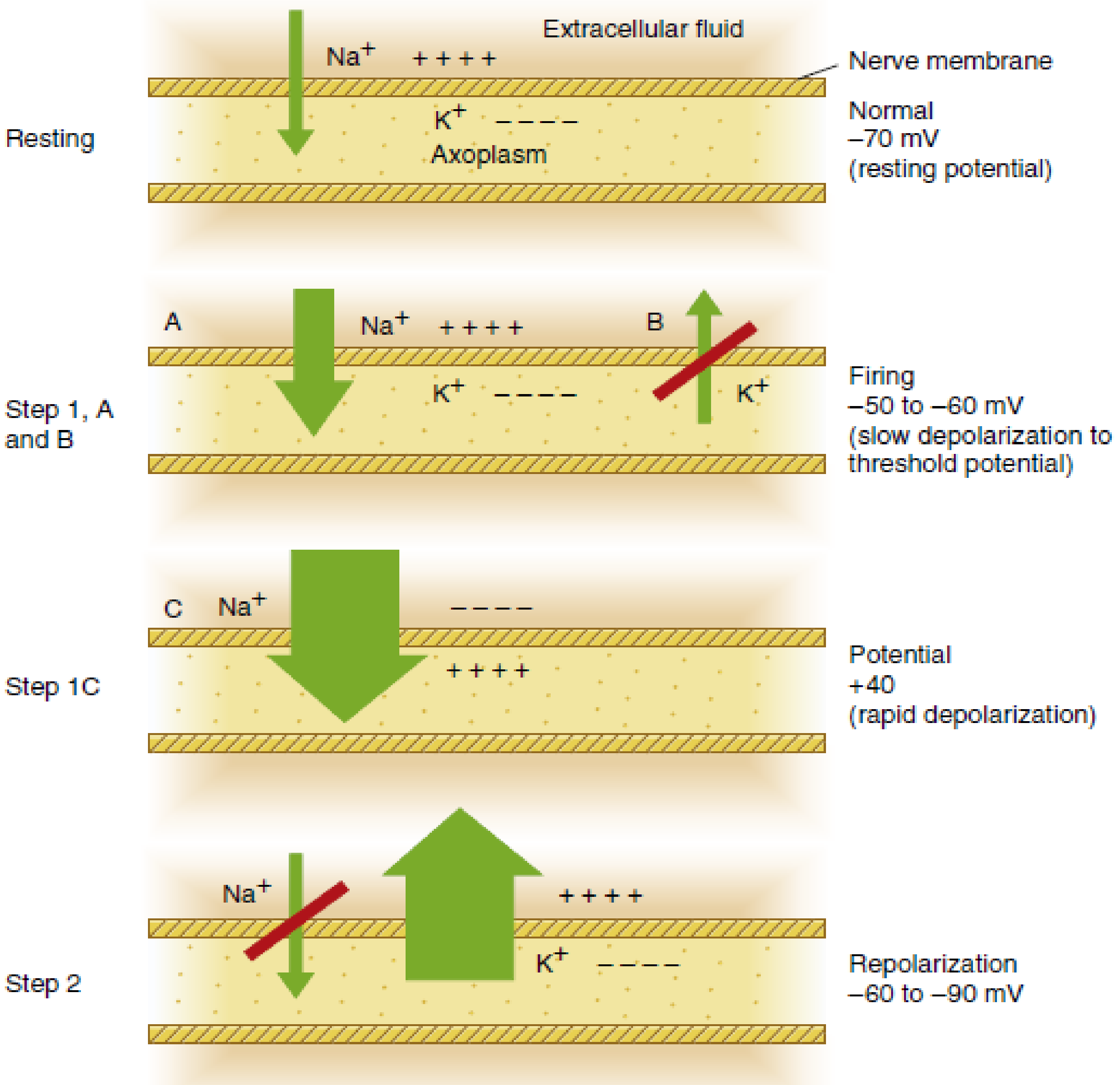
The preceding sequence of events depends on **two important factors:** the concentrations of electrolytes in the axoplasm (**interior of the nerve cell**) and extracellular fluids, and the permeability of the nerve membrane to sodium and potassium ions.



Membrane channels are open; depolarization occurs. Hydrated sodium ions (Na+) now pass unimpeded through the sodium channel.



Propagation. (A) Current flows between active (depolarized) and resting (polarized) membrane patches because depolarization reverses the membrane potential. (B) The previously resting membrane segment is now depolarized, setting up new current flows between it and the next membrane patch. The previously depolarized nerve segment (A) is on the road back to repolarization, leaving it refractory. The impulse can move forward only, as retrograde propagation is prevented by inexcitable (refractory) membrane. (C) The wave of depolarization has advanced by another segment, always trailed by a refractory membrane patch. The leftmost membrane segment, refractory in (A), has repolarized meanwhile and is once again ready to conduct a fresh impulse.

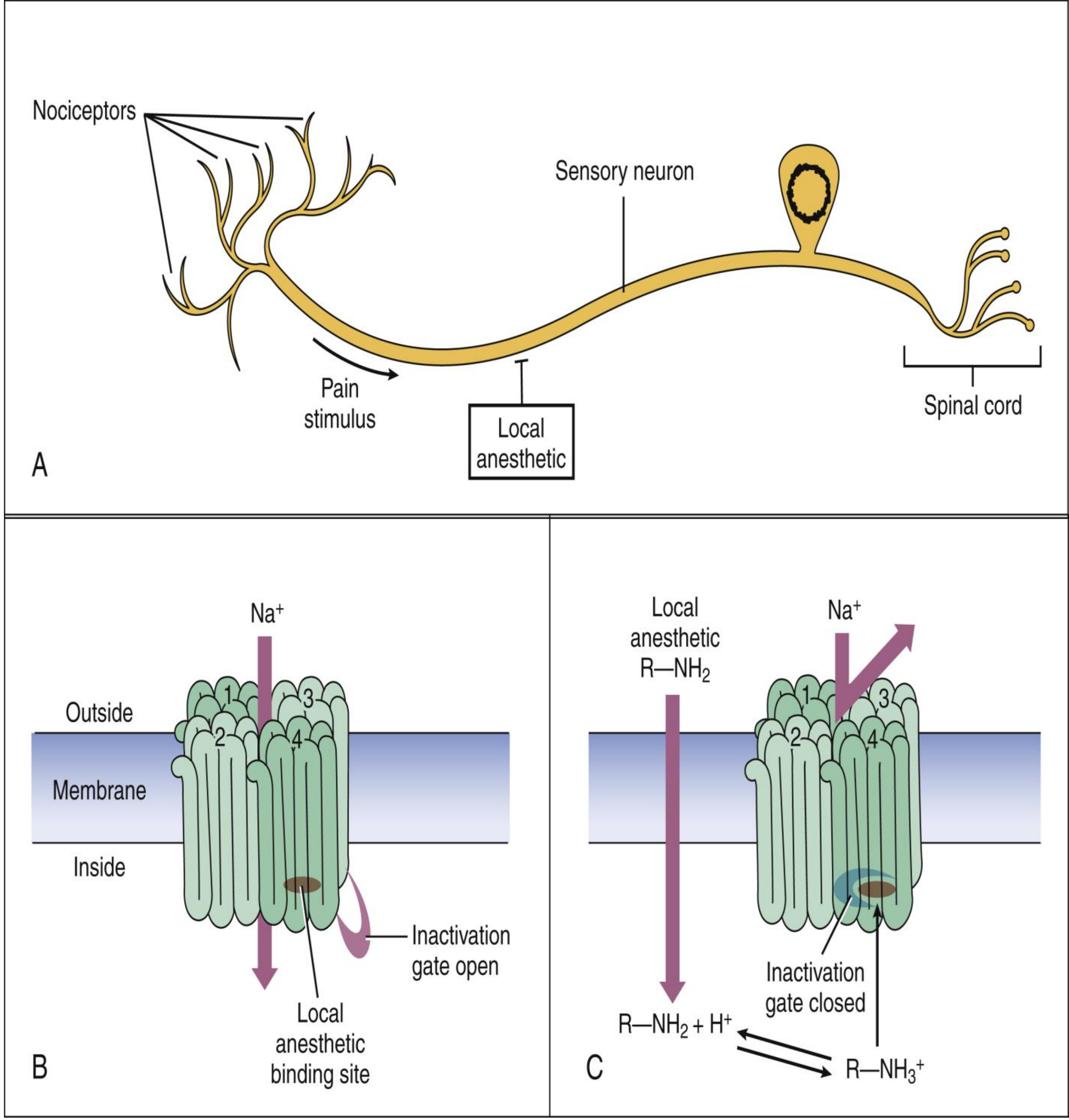


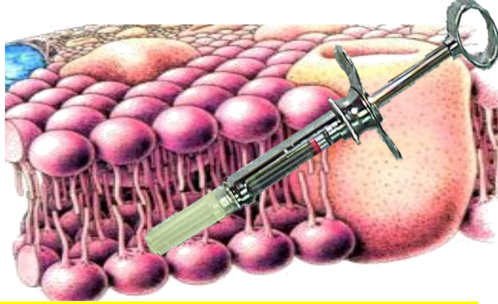
Resting potential, slow depolarization to threshold (step 1, A and B), rapid depolarization (step 1C), repolarization (step 2).

LA blocks the entrance of sodium ions into their channels in the nerve cellular membrane. The permeability to sodium is necessary to generate a new action potential to transmit nerve impulses to the brain . LAs are **classified** into **two large groups** depending on its chemical bond**: amides** or **ester;** the latter are almost disappearing in dentistry.

The Ester LAs are easily hydrolyzed in aqueous solution, while amide bond LAs are relatively resistant to hydrolysis.

The most important factors that affect onset and duration of action of LA are tissue pH, **drug pKa**, **diffusion time from the tip of the needle to nerve**, **nerve morphology**, **drug concentration**, and **drug solubility in lipids** .





The mechanism for **differential block**, the **block of pain perception** without motor block, is still unclear

Decreasing permeability to sodium ions

Decreases the rate of depolarization of the nerve membrane

Increases the threshold for excitability

Prevents propagation of the action potential

Local anesthetics may reduce permeability by competing with calcium for the membrane binding sites and by preventing the onset of nerve

Ionization factors

Local anesthetic agents are weak bases occurring equilibrated between

Fat-soluble (lipophilic) free base

Water-soluble (hydrophilic) hydrochloride salt

The proportion in each form is determined by

The acid dissociation constant (pKa) of the local anesthetic

The pH of the environment

The type of **nervous blockade produced by LA** is named **non-depolarizing nervous block**.

Local anesthetics **block sodium channels permeability**, they selectively .

Local anesthetic molecule. inhibit the maximum **permeability to sodium**, whose values usually are **five to six** times greater than the necessary minimum to conduct the impulse, when this value **fail**, and the **nerve block occurs**. In other words, local anesthesia is induced when the spread of **action potential** is **inhibited**, so a painful sensation cannot be transmitted from the site of origin to the brain. or, **LA** alter the mechanism to sodium ions to gain access to the **axoplasm nerve**.

The nerve membrane stays in a polarized state due to the impossibility of ionic movement responsible of action potential.

Because the **drug molecule must cross the lipid membrane to reach the cytoplasm**, the more lipid-soluble (**nonionized, uncharged**) form reaches effective intracellular concentrations more rapidly than **does the ionized form**.

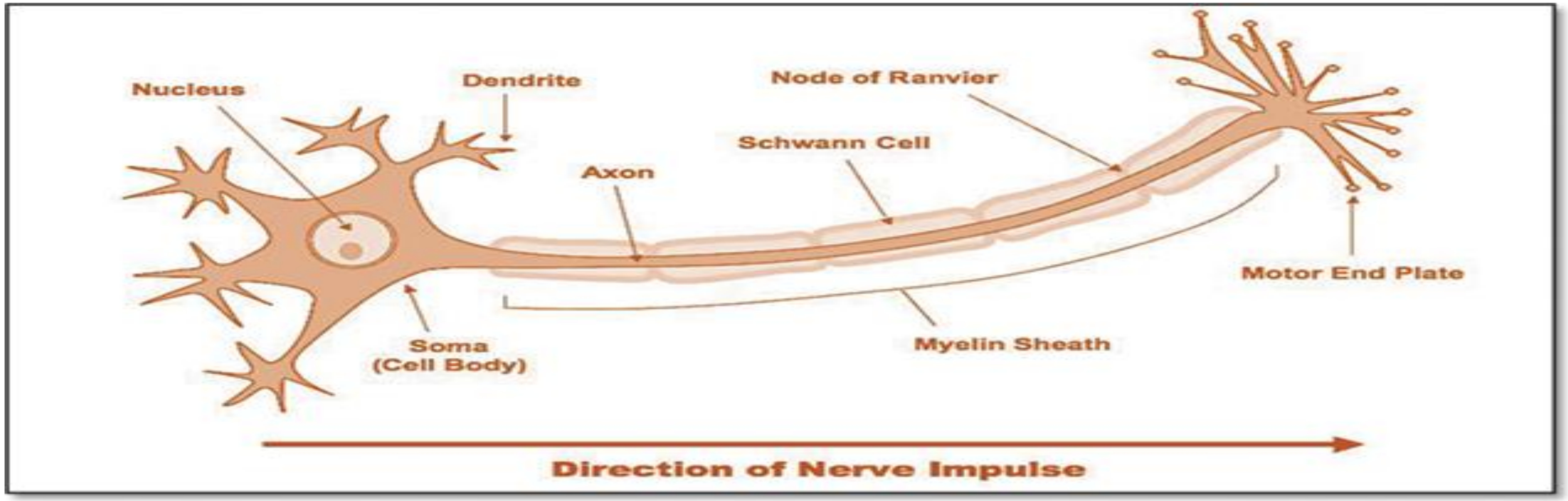
On the other hand, once inside the axon, the ionized (charged) form of the drug is the more effective blocking entity. Thus, both the nonionized and the ionized forms of the drug play important roles—the first in reaching the receptor site and the second in causing the effect. **The affinity of the receptor** site within the sodium channel for the local anesthetic is a function of the state of the channel, whether it is resting, open, or inactivated, and therefore follows the same rules of use dependence and voltage dependence that were described for the sodium channel-blocking antiarrhythmic drugs .

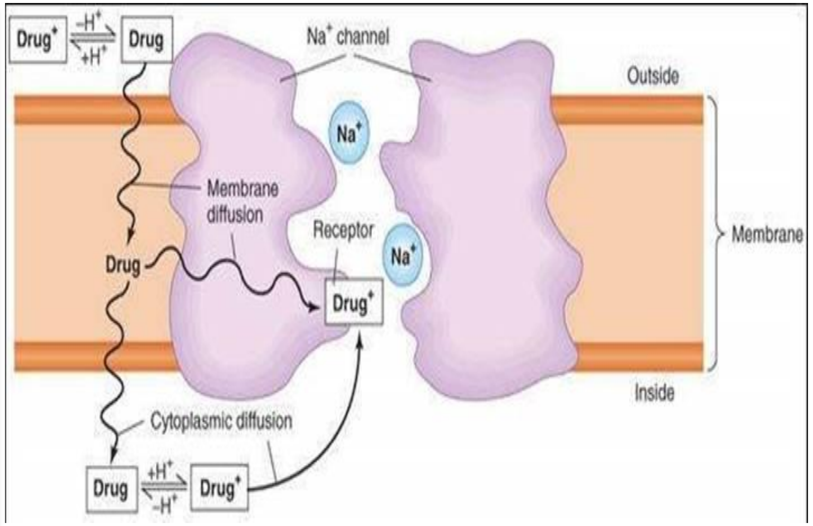
In particular, **if other factors are equal**, rapidly firing fibers are usually blocked before slowly firing fibers. **High concentrations of extracellular K$^+$** may enhance local anesthetic activity, whereas **elevated extracellular Ca^{2+} may antagonize it.** This portion is responsible for the **affinity of nerve cells**. The hydrophilic portion is an **amino ethanol** or **acetic acid derivative** and is responsible of water solubility and **diffusion across tissues**.

LAs are **amphipathic**; that is, they have **lipophilic** and **hydrophilic** characteristics at their opposite ends of their molecules .

Amides are the most common **molecules**; **procaine** is the prototype of this group and **benzocaine for topic application**.

The minimal concentration of LA to block the conduction of a nociceptive impulse is named **potency**. The therapeutic value of the drug in terms of **efficacy** and **tolerability** is called **toxicity.** The ability of the drug to reach tissues far from the site of administration of LA is called **diffusion**. Time between the action of LA and the metabolism of its compounds is named **time of action**





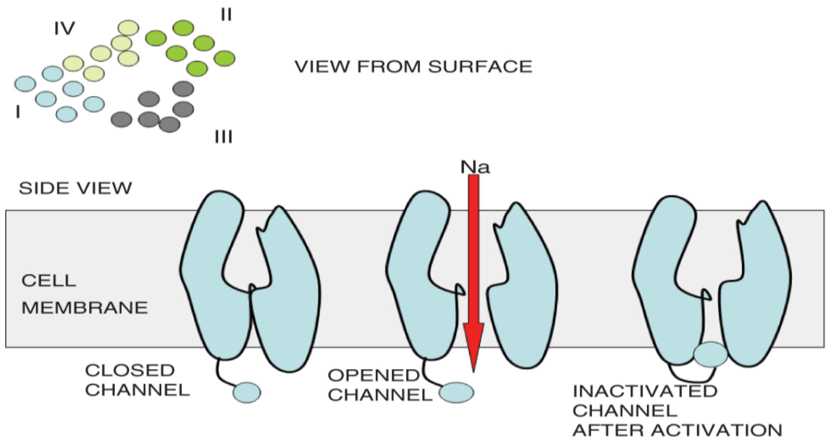
Mode of actions of LA:

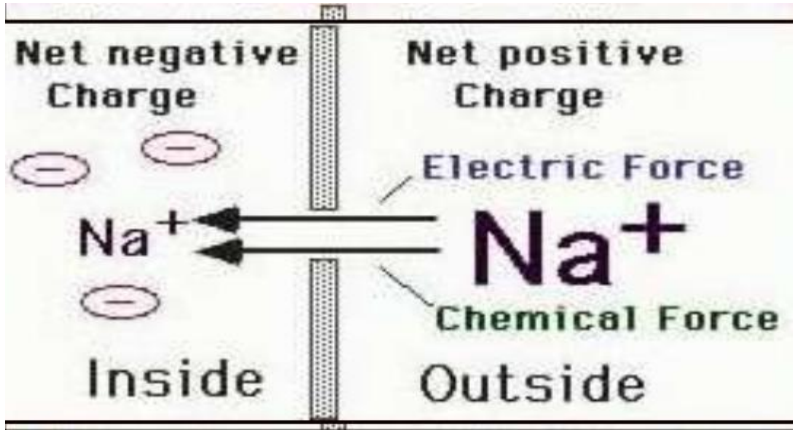
Mechanism of action of local anesthetics — **LAs reversibly inhibit nerve transmission by binding voltage-gated sodium channels (Na_v) in the nerve plasma membrane**. **Na_v** channels are integral membrane proteins, anchored in the plasma membrane .

Local anesthetics produce **anesthesia by inhibiting excitation of nerve endings or by blocking conduction in peripheral nerves**. This is achieved by anesthetics reversibly binding to and inactivating **sodium channels.**

Local anesthetics block voltage-dependent sodium channels and reduce the influx of sodium ions, thereby preventing **depolarization of the membrane** and **blocking conduction of the action potential**.

Local anesthetics gain access to their receptors from the **cytoplasm or the membrane**.





Schematic diagram depicting paths of local anesthetic (LA) to receptor sites. Extracellular anesthetic exists in equilibrium between charged and uncharged forms. The charged cation penetrates lipid membranes poorly; intracellular access is thus achieved by passage of the uncharged form. Intracellular re-equilibration results in formation of the more active charged species, which binds to the receptor at the inner vestibule of the sodium channel. Anesthetic may also gain access more directly by diffusing laterally within the membrane (hydrophobic pathway).

Active forms of local anesthetics

Commonly used amino amides include **lidocaine, mepivacaine, prilocaine, bupivacaine, etidocaine, and ropivacaine and levobupivacaine**. Commonly used amino esters include cocaine, procaine, tetracaine, chloroprocaine, and benzocaineCommonly used amino amides include **lidocaine, mepivacaine, prilocaine, bupivacaine, etidocaine, and ropivacaine and levobupivacaine**. Commonly used amino esters include cocaine, procaine, tetracaine, chloroprocaine, and benzocaine

Types of local anesthetics:

Esters:

These include **Cocaine, Procaine, Tetracaine, and Chloroprocaine, Amylocaine, Oxybuprocaine. Proparacaine,Tetracaine**

They are hydrolyzed in plasma by **pseudo-cholinesterase**.

One of the by-products of metabolism is para-aminobenzoic acid, the common cause of allergic reactions seen with these agents.

Amides:

These include **Lidocaine, Mepivicaine, Prilocaine, Bupivacaine, and Etidocaine. Cinchocaine,Articaine Dibucaine, Levobupivacaine, Ropivacaine,Sameridine,Tonicaine**

They are **metabolized** in the **liver** to **inactive agents**.

True allergic reactions are rare (**especially with lidocaine**)

Thank You!