Oral Surgery

Lecture: 4 **Dr. Saif Saadedeen**

"Neurophysiology"

The neuron or the nerve cell is the structural unit of the nervous system; it is able to transmit messages between the central nervous system and all parts of the body. There are two types of neuron: the sensory neuron and the motor neuron.

Sensory neurons that are capable of transmitting the sensation of pain consist of three major parts which are the cell body, the axon and the dendrites. The **dendrites**, which ends in the free nerve endings, is the most distal segment of sensory neuron which responds to stimulation produced in the tissue 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 axon has been measured as 100 -200 cm). It is composed of neural cytoplasm and covered by a thin sheath which is the nerve membrane, in some nerves a lipid-rich layer of myelin covers this membrane and the membrane is known as myelinated nerve fiber. Current theories stated that nerve excitability and conduction are both attributed to the changes developing within the nerve membrane.

The nerve membrane consists of two layers of lipid molecules (bilipid layer of phospholipids) and associated proteins and carbohydrates. Since the nerve membrane has selective permeability, therefore significant differences exist for ions between the intracellular and the extracellular concentrations

The **cell body** in the sensory neurons located at a distance from the axon (axon: the main pathway of impulse transmission in the nerve). The cell body is, therefore, not involved in the process of impulse transmission. The primary function of the cell body is to provide metabolic support to the entire neuron.

The function of a nerve is to carry messages from one part of the body to another. These massages, in the form of electrical action potentials, are called impulses. Action potentials are transient depolarization of the membrane that result from a brief increase in the permeability of the membrane to sodium and usually from a delayed increase in the permeability to potassium. Impulses are initiated by chemical, thermal, mechanical or electrical stimuli.

“Mechanism of action of local anesthesia”

The concept behind the action of LA is that it prevents both the **generation** and the **conduction** of a never impulse thereby they act like a roadblock between the source of impulse (as a surgical incision in soft tissue) and the brain.

Many theories have been suggested to explain the mechanism of action of LA, in general, the nerve membrane is the site at which LA agents exert their pharmacological actions, the most popular theories are:

1. Membrane expansion theory: this theory states that anesthetic molecules diffuse through the nerve membrane producing a general disturbance of the bulk membrane structure, expanding some critical regions in the nerve membrane and decrease the permeability to sodium ions (decreased diameter of sodium channels), thus inhibiting both conduction and nerve excitation.

2. Specific receptor theory: this is most favored theory today, it proposes that local anesthetics act by binding to specific receptors in the sodium channel (protein channel) in the nerve membrane, the action of the drug is direct, not mediated by some change in the general properties of the cell membrane. Once the local anesthetic has gained access to these receptors permeability to Na ions is decreased or eliminated and nerve conduction is interrupted.

“Factors Affecting The Local Anesthetic Action”

1. PH value

It is well known that the pH of a local anesthetic solution (as well as the pH of the tissue into which it is injected) greatly influences its action. Acidification of tissue decreases local anesthetic effectiveness. Inadequate anesthesia results when local anesthetics are injected into inflamed or infected areas. The inflammatory process produces acidic products: The pH of normal tissue is 7.4; the pH of an inflamed area is 5 to 6.

Local anesthetics containing epinephrine or other vasopressors are acidified by the manufacturer to inhibit oxidation of the vasopressor. The pH of solutions without epinephrine is about 6.5; epinephrine-containing solutions have a pH of about 3.5. Clinically, this lower pH is more likely to produce a burning sensation on injection, as well as a slightly slower onset of anesthesia.

Elevating the pH (alkalinization) of a local anesthetic solution speeds its onset of action, increases its clinical effectiveness and makes its injection more comfortable.

Despite potentially wide pH variation in extracellular fluids, the pH at the interior of a nerve remains stable. Normal functioning of a nerve, therefore, is affected very little by changes in the extracellular environment. However, the ability of a local anesthetic to block nerve impulses is profoundly altered by changes in extracellular pH.

2. Lipid solubility

Increased lipid solubility of local anesthetics permits the anesthetic to penetrate the nerve membrane more easily. Local anesthetics with greater lipid solubility produce more effective conduction blockage at lower concentrations than less lipid soluble solutions

3. Protein binding

The degree of protein binding of the anesthetic molecule is responsible for the duration of local anesthetic activity. Local anesthetic possessing a great degree of protein binding appear to attach more securely to the protein sites and to possess a longer duration of clinical activity.

Longer-acting local anesthetics (e.g. bupivacaine) are more firmly bound in the nerve membrane (increased protein binding) than are shorter-acting drugs (e.g. lidocaine) and therefore are released more slowly from receptor sites in the sodium channels.

4. Vasodilator activity

The local anesthetic solution with greater vasodilator activity will increase blood flow to the area; this will lead to rapid removal of anesthetic molecules from the injection site, decreasing anesthetic potency and duration.

5. **Vascularity of the injection site**
   the duration is increased in areas of decreased vascularity.

6. **Presence or absence of vasoactive substance**
   the addition of a vasopressor into local anesthetic solution decrease the tissue perfusion thus increasing the duration of action.

- As the local anesthetic is removed from the nerve, the function of the nerve returns rapidly at first, but then it gradually slows. Compared with the onset of the nerve block, which is rapid.

Electrophysiology of the nerve conduction: (للأطلاع)
Following is a description of electrical events that occur within a nerve during the conduction of an impulse.

Resting state (phase 1)

A nerve possesses a resting potential. 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.

Depolarization (phase2)

When a stimulus of significant intensity is applied to the nerve it will excite the nerve and the following events occur in sequence:

1. Slow depolarization: this is the initial phase wherein the electrical potential inside the nerve become slightly less negative.

2. Rapid depolarization: the electrical potential which is becoming less negative, reaches a critical level, it results in an extremely rapid phase of depolarization.

3. Reversal of electrical potential: with the phase of rapid depolarization there is a reversal of electrical potential across the nerve membrane, the interior of the nerve is now electrically positive and the exterior is negative.

The depolarization occurs due to an increase in permeability of the membrane to Na ions. The rapid influx of Na ions to the interior of the nerve will cause depolarization of the nerve membrane from the resting level to its firing threshold.

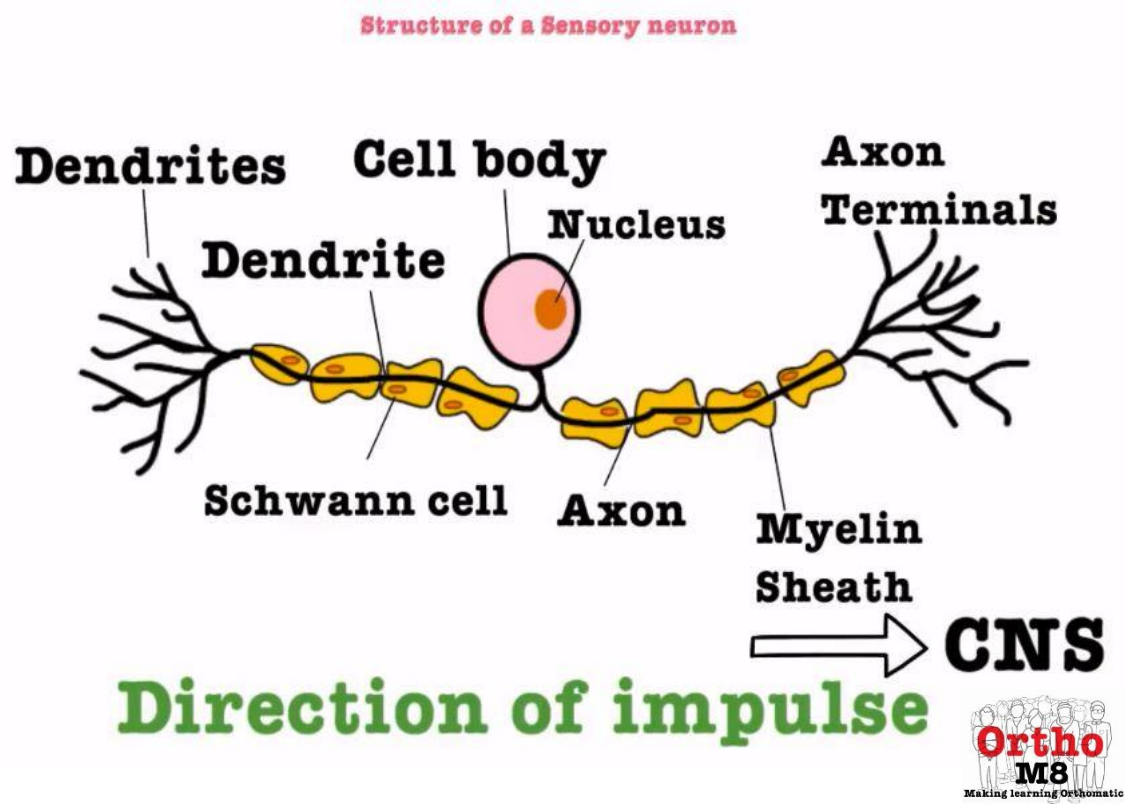
The firing threshold is actually the magnitude of the decrease in the negative membrane potential that is required to initiate an action potential (impulse). When firing threshold is reached, the permeability of the membrane to Na increases and at the end of depolarization the electrical potential of the nerve is reversed; an electrical potential of +40 mV exists. This process takes 0.3 milliseconds.

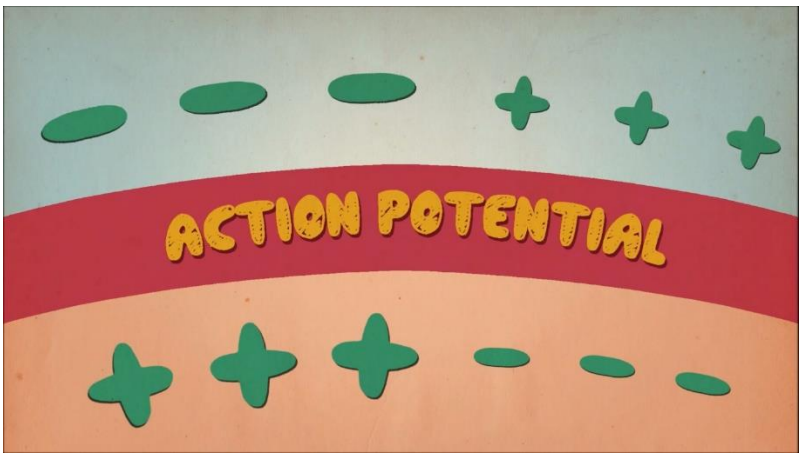
Re-polarization (phase 3)

The electrical potential gradually becomes more negative inside the nerve relative to the outside until the original resting potential of -70 mV is restored, this is achieved by increasing permeability to k ions; K ions move to the outside. This process takes 0.7 milliseconds. The movement of Na ions to the inside of the nerve during depolarization and the movement of K ions out of the nerve during re-polarization are a passive process (no energy is required); each ion moves along its concentration gradient (higher → lower).

When the nerve return to its resting state a slight excess of Na ions exist within the nerve cell and a slight excess of K ions exist extracellularly. A period of metabolic activity then begins in which active transfer of sodium ions out of the cell occurs via the **sodium pump**. An expenditure of energy is necessary to move sodium ions out of the nerve cell against their concentration gradient; this energy comes from the oxidative metabolism of adenosine triphosphate (ATP). The same pumping mechanism is thought to be responsible for the active transport of potassium ions into the cell against their concentration gradient. The process of repolarization requires 0.7 milliseconds.

Accordingly, the action potential could be defined as a transient membrane depolarization that results from a brief increase in the permeability of the membrane to sodium and also from a delayed increase in the permeability to potassium. Once an impulse has been initiated it moves along the surface of the axon to the CNS.





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