University of Mosul College of Dentistry
Oral &Maxillofacial Surgery Dept. (Third Year)
LOCAL ANESTHESIA

Lect. 1

Anesthesia is the loss of feeling or sensation in a part of or all of the body. Anesthesia may occur as a result of injury to or disease of a nerve, but the term anesthesia is mostly applied to the technique of reducing or abolishing an individual's sensation of pain to enable surgery to be performed. This is affected by administering drugs (local anesthesia or general anesthesia).

Local anesthesia: is the loss of all sensation in a localized area in the body without loss of consciousness,

General anesthesia: is the loss of all sensation in all part of the body accompanied by loss of consciousness.

(reversible state of loss of consciousness with abolished reflex to painfull stimulus)

Sedation (general analgesia):loss of pain sensation in all part of the body without loss of consciousness

Pain

It is an unpleasant emotional experience usually initiated by a noxious stimulus and transmitted over a specialized neural network to the CNS where it is interpreted

Pain has dual nature:

1. *Pain perception:* It is a physio-anatomic process were impulse is generated as transmitted.
2. *Pain reaction:* It is a psycho-physiological process where individual feels and senses pain.

Paresthesia is an abnormal sensation such as tingling, tickling, pricking, numbness or burning of a person's skin or mucosa.

1. *Specific theory:* Specific **mediator of touch,heat,** cold and pain are present on skin and from here sensory nerve takes the impulse to the brain where it is interpreted.

2. ***Pattern theory:*** Pain is dependent upon specific pattern of nerve impulse produced by summation of sensory input within **spinal cord.**

3. ***Gate control theory:*** Peripheral nerves carry impulse from skin to CNS, larger nerves also help in exciting or inhibing the impulse.

Methods for inducing local anesthesia:

several methods available for inducing loss of sensation (local anesthesia)

1. Mechanical trauma (pressure)
2. Low temperature (freezing)
3. Anoxia (reduce oxygen tension)

In 1,2 and 3 nerve impulse transmission will inhibited

4. Chemical irritant…….euginol
5. Neurolytic agent ……alcohol (irreversible nerve damage)
6. Chemical agent (anesthetic agent)

For chemical agents that have local anesthetic activity to be used as anesthesia, should have following properties:

1. <u>Mandatory</u> properties:

a) Potent and reliable for producing local anesthesia

b) Reversible. It should not cause any permanent alteration of nerve structure and function. (alcohol cause anesthesia but not regarded as anesthetic agent).

c) Minimal toxicity(margin of safety MOS). Its systemic toxicity should be low as possible.

d) Rapid onset. The time of onset of anesthesia should be as short as possible.

e) Acceptable duration. The duration of action must be long enough to permit completion of the procedure yet not so long as to require an extended recovery. This depend on the type of procedure to be performed.

f) Non-irritating. It should not be irritating to the tissue to which it is applied. Thus, it should produce no inflammatory response.

g) Stable in solution to prevent precipitation of anesthetic agent.

h) Easily metabolized by plasma or liver with least toxic byproducts.

2. **Preferable properties**

a) Minimal incidence of allergisity (thus procaine has limited use)

b) Adequate shelf life for the solution (from manufacturing till uses)

c) Surface anesthesia:It must be effective regardless of whether it is injected into the tissue or is applied locally to mucous membranes.

d) Easy to sterile

Indications of local anesthesia

1. Diagnosis of original site of pain(in case of undiagnosed pain site)
2. Reduce or eliminate pain during dental treatment

Contraindication of local anesthesia

Systemic

1. Uncooperative patient like child or mentally retarded patient that is difficult to be controlled
2. Uncontrolled hemorrhagic patient in which bleeding may induced by injection specially in case of deep injection site in the muscle like in block injection.
3. Allergic patient to local anesthesia agents or one of its constituents.

Relative

1. Advanced liver disease as metabolism of most of local anesthesia occur in the liver.
2. Thyrotoxic goiter because adrenaline used as vasoconstrictor in local anesthesia may initiate thyrotoxic crisis in this patient which is dangerous emergency in dental clinic.
3. Uncontrolled cardiovascular disease. Also adrenaline may stimulate cardiac arrhythmia or produce cardiac arrest.

Local

1. Patient received radiotherapy in whom local anesthesia may reduce blood supply and inducing osteoradionecrosis.
2. Acute infection at injection site as local anesthesia fail and infection may spread by this injection.
3. Vascular abnormality at operation site like hemangioma because control of hemorrhage is difficult and injection at that site may stimulates bleeding.

Advantages of local anesthesia

1. Simple technique comparing to general anesthesia
2. Minimal equipment needed for this technique
3. Transportable (no need for special operating room)
4. Minimal contraindication comparing to general anesthesia
5. Hemorrhage could be controlled by vasoconstrictor that is added to solution
6. No airway impairment
7. Minimal postoperative care needed. (Patient can leave dental clinic immediately after operation)
8. No need for anesthetist as dentist administer the anesthesia
9. Duration could be controlled (short or long procedure could be controlled to certain limit)
10. Co-operative patient simplify the work.

Disadvantages of local anesthesia

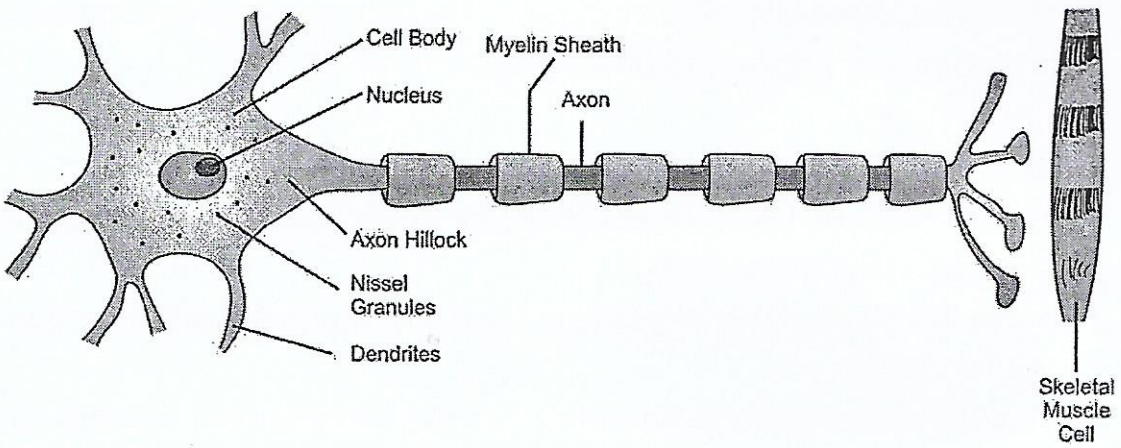
1. Difficult to achieve co-operation in child and mentally retarded patient
2. Mechanical obstruction by large tongue or limited mouth opening
3. Failure due to infection, anatomical variation or incomplete anesthesia
4. Prolonged paresthesia post operatively may produce patient discomfort.
5. Spread of acute infection at injection site

Mode of Action Of local Anesthesia

Lacal anesthesia act by its Prevention of generation and or conduction of nerve impulse thus its act as chemical roadblock between source of impulse and brain.

NERVE PHYSIOLOGY

1. Nerve cell body 2. Dendrite
3. Axon.



Nerve fibres are of two kinds:

1. Non-myelinated nerve fibres: In these fibres the axis cylinder is covered by a membrane called neurolemma. The speed of nerve impulse conduction in these fibers is less.

2. Myelinated nerve fibers : In these fibers the axis cylinder is covered by a thick sheath called myelin sheath which is in turn covered by neurolemma.

type of pain produced by specific fiber depend on the type of nerve fiber. Sharp pain mainly related to myelinated a delta fiber while dull pain (ache) mainly associated with type c fiber.

IMPULSE GENERATION AND CONDUCTION IN A NERVE FIBRE (Generation of Action potential)

Resting State

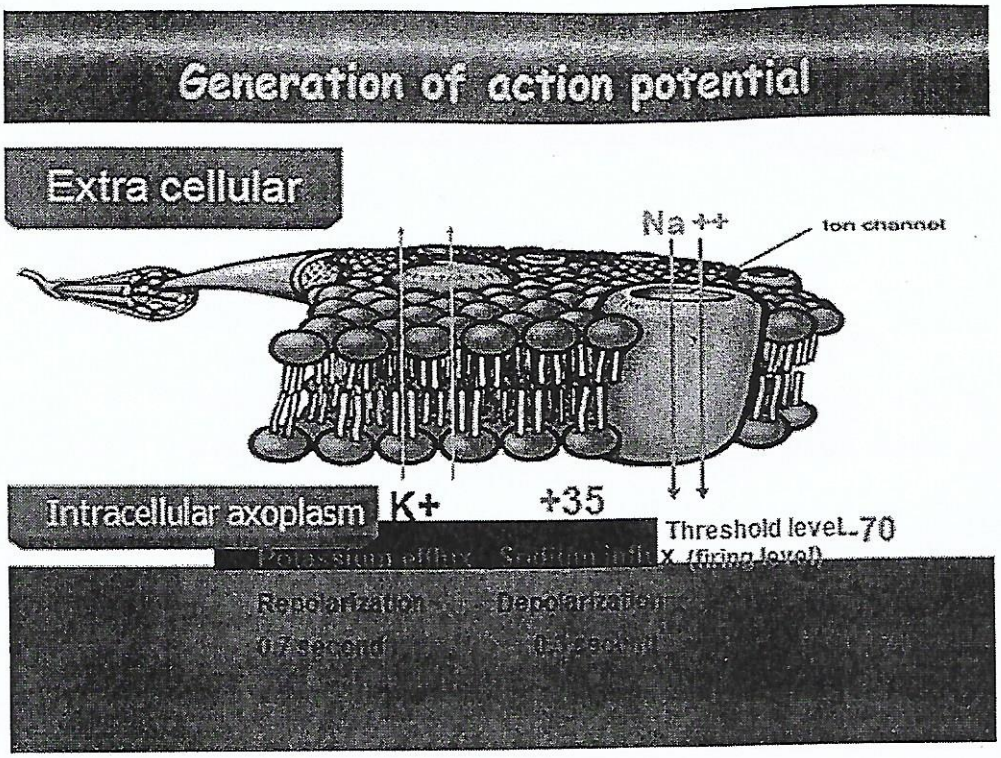
During resting state the membrane is impermeable to Na+ ions. This change of permeability causes a potential difference across the membrane of -70 mV to -90 mV relative to inside. (Resting potential)

Depolarised State

As any impulse reach the nerve membrane making membrane freely permeable to Na+ ions. This alters the membrane potential. Slow sodium influx makes the membrane potential positive relative to outside. when membrane potential reaches specific level (Threshold or firing level) the sodium influx starts to be faster. This process is active as it dependent on difference in electrostatic and concentration gradient.

Repolarised State

Repolarization occur immediately after termination of stimulus by slow potassium efflux to regain negative polarity .



The nerve impulse is transmitted through the nerve either by rapid saltatory jumping in myelinated fiber or by slow creep conduction in nonmyelinated fiber.

How local anesthesia work

Local anesthesia is caused by depression of excitation in nerve ending or an inhibition of the conduction process in the peripheral nerves.
Local anesthetics interfere with propagation of the action potential by blocking the increase in sodium permeability during depolarization.
Depolarization inhibited
Firing level not reached
Action potential inhibited

Theories of Local Anesthesia Action

1. *Acetylcholine theory:*

2. *Calcium displacement theory:*

3. *Surface charge theory:*

4. *Membrane expansion theory:* Local anesthesia solution must be lipid soluble and it easily diffuses into the nerve membrane, thus altering the nerve membrane permeability by disordering the membrane and sodium channel. It blocks the sodium channel and inhibiting depolarization. Only benzocaine is lipid soluble and may act according to this theory.

Specific receptor hypothesis: Local anesthesia solution attaches itself to specific receptors present at the internal surface of nerve membrane and generally near the Na+ channel. This blocks the sodium channel and inhibits depolarization. This theory is best accepted theory by chemical and electrophysiological studies.

Structure of local anesthesia

Anesthetic agent constitute from liopophlic aromatic part connected to hydrophilic terminals by intermediate connector. Type of the connector determine the group of the agent -either ester (linked by COO) or amide (linked by NHCO).

Thus chemically local anesthetic agent either ester or amide. There is new generation and other unclassified agent but these agent are less common in dental practice.

Dissociation constant PKa:

pH at which 50% of drug present in free base form and 50% in cationic form (water soluble)

Most local anesthetic Pka range (7-9)

How can local anesthesia cross nerve membrane(intracellular)?

Local anesthetic solution prepared as weak acid form at 4-5 pH (to prevent precipitation of free base in neutral solution), thus it present as cataionic ionized (h+)form that must converted to base form to be lipid soluble and cross cell membrane.

Local anesthetic agent after injection at any site and with function of plasma buffering will dissociate into free base form and cataionic form according to its pka and PH of the site. Free base form generated cross the cell membrane. After crossing the membrane another dissociation occur and cataionc form resulted will bind the receptor exerting anesthetic function.

Properties of local anesthesia

Onset (Induction Time)

Time from injection of local anesthesia to the sign of adequate surgical anesthesia achieved

Factors affect onset (induction time)

1. Concentration of the agent
2. pH of the solution and site of injection. The nearest the PH to Pka the fastest the onset and.
3. Pka.
4. Anatomical barrier. This depends mainly of the diameter of nerve sheath.(block technique associated with slower onset comparing to infilteration)
5. Lipid solubility. Lipid soluble agent crosses nerve sheath immediately after injection.

Duration

Time from early sign appeared to the complete loss of all effects of drug occur

This results from reduced concentration of drug with no binding with the receptors.

Concentration gradient of the agent depleted by dilution by interstitial fluid, action of capillary and lymph, absorption by other tissue and hydrolysis of ester. These factors result in reduced concentration extra-neuronal. Local anesthetic then move from intra to extra- neuron.

Recovery from local anesthesia is slower than induction or onset. This results from protein binding capacity of agent. This capacity is differ from agent to another (buivicaine and etidocaine have high protein binding capacity and longer duration).

Factors affecting duration:

1. Protein binding.
2. Vasoactivity of the agent (Vasoconstrictor or vasodilator)
3. Concentration
4. Vascularity of the site (highly vascular site resulting dilution of local anesthesia and systemic distribution, thus shorter duration).

Reinjection of local anesthesia

Sometime during procedure additional anesthesia needed. The concentration then will re- increased and local anesthesia regain exerting the function. Some time failure to achieve adequate anesthesia occurs. The main cause of failure of reinjection to achieves profound anesthesia is:

1. Edema
2. Localized hemorrhage
3. Clot formation

   1,2 and 3 either from injection or from the dental procedure. These factors will forms a barrier that isolate the nerve and prevent anesthesia from reaching the nerve.

4. Reduced pH at the site due to edema or acidity of injected solution (poor buffer capacity of plasma will thus inhibit the action of local anesthesia)
5. Hypernatremia. Increased concentration of sodium ion extraneuronaly reaching high level forcing the closed sodium channel to opened and allowing accumulated sodium to inter intraneuronaly

Progression of local anesthetic function

Loss of sensation by local anesthesia differ and occur gradually. These differences occurs due to difference in diameter of nerve fiber and weather its mylinated or not.

Progression of loss of sensation mainly occur in following pattern.

1. Dull pain
2. Thermal change
3. Sharp pain
4. Touch
5. Deep pressure
6. Proprioception
7. Motor function