Review Article

LOCAL ANAESTHESIA: A REVIEW

DR. SHASHWAT AGRAWAL*[1], DR. ASHUTOSH AGRAWAL[2], DR. NIDHI AGRAWAL[3], DR. SONAL AGRAWAL[4]

[1]**PG RESIDENT, DEPT. OF ANESTHESIA, NIMS HOSPITAL, JAIPUR, RAJASTHAN**

[2]**SENIOR RSIDENT, DEPT OF ANESTHESIA, LATE SHRI LAKHIRAM AGRAWAL MEMORIAL GOVERNMENT MEDICAL COLLEGE, RAIGARH (C.G.)**

[3]**SENIOR RSIDENT, DEPT OF ORTHODONTICS, NEW HORIZON DENTAL COLLEGE, BILASPUR, CHHATTISGARH**

[4] **SENIOR RSIDENT, SIR JJ GROUP OF HOSPITALS, GRANT GOVERNMENT MEDICAL COLLEGE, MUMBAI, MAHARASHTRA**

*Corresponding author: Dr.SHASHWAT AGRAWAL; Email id. –shashwatagrawal77@gmail.com

ABSTRACT

Pain is an overall term that depicts any sort of unpleasant or awkward sensation in the body. Pain management is most important challenge in medical and dental field. Local anesthetics are backbone of pain control techniques. These represent safest and most effective method for managing pain associated with medical and dental treatment. Local anesthetics are the drugs which provides loss of sensation in circumscribed area without loss of consciousness. These drugs are in opposite to general anethesia. In order to induce anesthesia in a given area, a weak solution of anesthetic drug is applied topically to mucous membrane or injected hypodermically. It interferes with excitation process of the nerve membrane. Local anesthetics are reversible in nature with very less side effects and toxicity. Local anesthetics are widely used by the clinicians. Understanding the pharmacology and mechanism behind the action of these local anesthetics is of importance for more efficient clinical outcome. Potency, speed of onset, duration of action and safety of a specific drug in a given clinical situation depends upon properties of the local anesthetic. Other factors influencing anesthetic properties are dosage and vasoconstrictors concentration present in the solution. Local anesthetics travel many tissue barriers to reach their site of action. Local Anesthetics are toxic on many tissues but clinically apparent nerve damage is very rare. Local anesthetics induced toxicity after peripheral nerve block has a good prognosis overall. This article focuses on basic structure, function, mechanism of action and different techniques of local anesthesia.

Keywords: Local Anesthetics, Anesthesia, Pain Control, Nerve Blocks

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INTRODUCTION

According to Stanley F. Malamed (1980)[1]Local anesthesia is characterized as a deficiency of sensation in restraint of the conduction interaction in peripheral nerves. Local anesthesiaor bar of nerve impulses to nullify sensation might be created by numerous tertiary amine bases, certain alcohols, and an assortment of different medications and toxins.Currently available useful agents are amino-esters or amino-amides. These drugs, when applied in sufficient concentration at the site of action, prevent conduction of electrical impulses by the membranes of nerve and muscle. Loss of sensation can be produced without inducing a loss of consciousness.

HISTORICAL BACKGROUND

Cocaine was first local anesthetic agent isolated by Nieman from coca tree. In 1884, Karl Koller demonstrated the anesthetic action. First successful and broadly utilized synthetic local anesthetic was procaine produced by Einhorn in 1905 from benzoic acid and diethyl amino ethanol. Biberfield identified procaine anesthetic properties Braun implied procaine for clinical practice. In 1948, Lofgren introduced Lidocaine. The discovery of its anesthetic properties was followed in 1949 by clinical use by T. Gordh. Thereafter, series of potent anesthetic agents soon followed with a wide spectrum of clinical properties.[2]

DESIRABLE PROPERTIES OF LOCAL ANESTHESIASELLA-NASION LINE

Local anesthesia has following desirable properties

- ✓ Non-irritating to tissue to which it is applied
- ✓ Not cause any permanent alteration of nerve structure.
- ✓ Low systemic toxicity.
- ✓ Short time of onset.
- ✓ It should be effective regardless of whether it is injected into the tissue or applied locally to mucous membrane.
- ✓ The duration of action should be long enough to permit the completion of procedure (yet not so long as to require an extended recovery)
- ✓ In addition to these qualities, BENNET lists other desirable properties of ideal L.A
- ✓ It should have the potency sufficient to give complete anesthesia without the use of harmful concentration solutions.
- ✓ It should be free from producing allergic reactions.
- ✓ It should be stable in solution and relatively biotransformation in the body.
- ✓ It should be either sterile or be capable of being sterilized by heat without deterioration.

CLASSIFICATION (Rule of "i's")

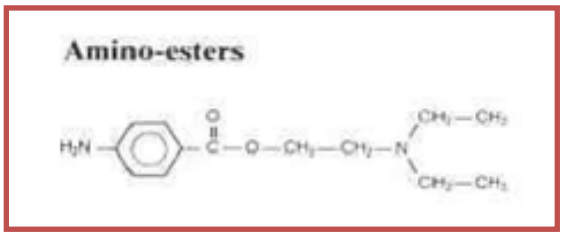
Esters	Am"i"des
Cocaine	Bupivacaine
Chloroprocaine	Lidocaine
Procaine	Ropivacaine
Tetracaine	Etidocaine
	Mepivacaine

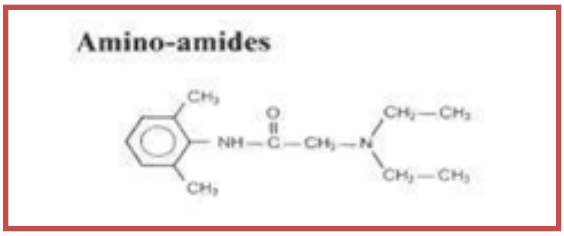
Differences of Esters and Amides

All local anesthetics are weak bases. Chemical structure of local anesthetics has an amine group on one end connect to an aromatic ring on the

other and an amine group on the right side. The amine end is hydrophilic (soluble in water), and the aromatic end is lipophilic (soluble in lipids). Two classes of local anesthetics are amino amides and amino esters. Amides get metabolized in liver. Esters are metabolized in plasma.

Structures of Amides and Esters-





The amine end is hydrophilic in nature. It's likewise liable for the answer for staying on one or the other side of the nerve membrane. The aromatic end is lipophilic (solvent in lipids). Anesthetic particles enter through the nerve membrane lipid bilayer.

COMPOSITION

Agent	Action
Local anesthetic drug (xylocaine, lignocaine)	blockade of nerve conduction
Vasoconstrictor (adrenaline 1:80,000)	Increase depth and duration of anesthesia
Sodium metabisulphite	reducing agent (antioxidant)
Methylparaben, caprylhydrocuprieno toxin	bacteriostatic agent
Thymol	fungicide
Vehicle (distilled water &NaCl)	volume and isotonicity of solution

MECHANISM

Local anaesthetics block the transmission of the action potential by inhibition of voltage-gated sodium ion channels. Under normal or resting circumstances, the neural membrane is characterized by a negative potential of roughly –90 mV (the potential inside the nerve fibre is negative relative to the extracellular fluid). This negative potential is created by active outward transport of sodium and inward transport of potassium ions, combined with a membrane that is relatively permeable to potassium and relatively impermeable to sodium ions. With excitation of the nerve, there is an increase in the membrane permeability to sodium ions, causing a decrease in the transmembrane potential. If a critical potential is reached (i.e., threshold potential), there is a rapid and self-sustaining influx of sodium ions resulting in depolarization, after which the resting membrane potential is re-established. From an electrophysiological standpoint, local anaesthetics block conduction of neural transmission by decreasing the rate of depolarization in response to excitation, preventing achievement of the threshold potential. They do not alter the resting transmembrane potential, and they have little effect on the threshold potential.[3,4]Duration of action of local anaesthetics is determined by rate of elimination of agent from site injected and factors such as lipid solubility, dose given, blood flow at site, addition of vasoconstrictors (does not reliably prolong all agents). Local anaesthetics are diluted as *parts of active drug per 100 parts of solution (grams percent)*.

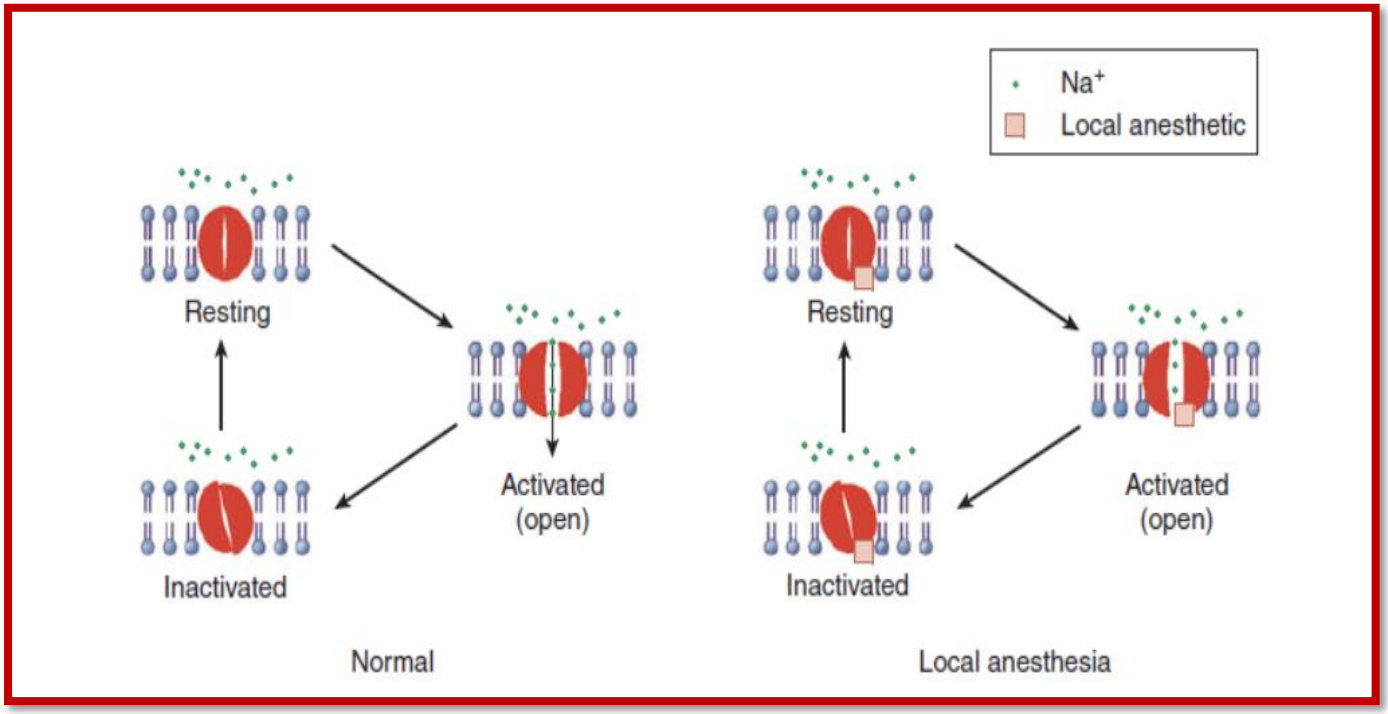


Fig.-1. Voltage-gated sodium (Na) channels exist in (at least) three states—resting, activated (open), and inactivated. Note that local anaesthetics bind and inhibit the voltage-gated Na channel from a site that is not directly accessible from outside the cell, interfering with the large transient Na influx associated with membrane depolarization. (Source: Morgan & Mikhail's Clinical Anaesthesiology 5th Ed.)[3]

FACTORS AFFECTING THE REACTION[4]

Lipid solubility

All local anesthetics have feeble bases. The essential idea of local anesthetics make them lipid dissolvable, expanding quicker nerve infiltration, blocks sodium channels, and builds the beginning of activity. Local anesthetics have two structures, ionized and non-ionized. The non-ionized structure can cross the nerve films and square the sodium channels. In this way, the more non ionized introduced, the quicker the beginning activity.

pH influence

pH impact for local anesthetics for the most part goes from 7.6 to 8.9. Abatement in pH shifts balance toward the ionized structure, postponing the beginning activity. Lower the pH, arrangement turns out to be more acidic and gives more slow beginning of activity.

Vasodilation

Vasoconstrictor is a substance which slows down the absorption of anesthetic agent into the bloodstream prolonging the action of the drug. Epinephrine (adrenaline) is naturally occurring vasoconstrictor.

PHARMACOKINETICS

Local anaesthetic drugs are deposited at the target site, hence their pharmacokinetic profile in blood are important determinants of elimination and toxicity.[5]

Absorption

Local anaesthetics are readily absorbed from mucous membrane, leading to immediate onset of action, whereas absorption from intact skin is poorer. Rate of absorption depends upon on the blood flow to the area of injection. Systemic Absorption of Local Anesthetics depends upon:

- ✓ Site of injection -Absorption is related to vascularity of the site. Absorption order: intravenous (intraarterial) > tracheal > intercostal >paracervical> epidural >brachial plexus > sciatic > subcutaneous.
- ✓ Addition of vasoconstrictor – vasoconstrictors decreases the blood flow to the area, decreasing the absorption and prolonging duration of action.
- ✓ Pharmacologic profile of the local anesthetic – More lipid soluble local anaesthetics are slowly absorbed compared to less lipid soluble agents.

Metabolism and Excretion

Ester linked local anaesthetics are rapidly hydrolysed by plasma pseudocholinestrase and remaining by esterase in the liver. Amide linked local anaesthetics are degraded only in liver microsomes by dealkylation and hydrolysis. For all compounds very little nonmetabolized local anaesthetic is excreted by the kidney.[6]

Factors affecting circulation levels

Elements influencing dissemination levels are the paces of absorption, distribution, and digestion. Speed of organization and levels of the portions decides the absorption. Distribution happens in three stages. To start with, the medication happens in profoundly vascular tissues, that is, the lungs and kidneys. Later it shows up less in vascular tissues, muscle, and fat. Then, at that point, the medication gets processed. Metabolism associated with the compound design depends on two classes, amide, and ester. Diminishing the potential toxicity brought about fast digestion.

VASOCONSTRICTORS

Epinephrine expressed as a ratio of grams of drug to the total cc's of solution. Vasoconstrictors are added in 1:200,000 or 5µg/ml of epinephrine to local anaesthetic solution used for infiltration, peripheral block and epidural and spinal anaesthesia. Localized vasoconstriction produced limits systemic absorption and prologs the duration of action of local anaesthetic agent. Vasoconstrictors should not be used in the locations such as fingers, toes, nose, ear lobes and penis as these areas may lack collateral flow.

TOXICITY

Toxicity from local anaesthetic can happen when the greatest portion for an individual has been regulated over 500mg. Some normal toxic impacts are lightheadedness, shuddering or jerking, seizures, hypotension (low pulse) and deadness.

Tissue toxicity is uncommon in nature yet can happen whenever controlled in sufficiently high fixations (more noteworthy than those utilized clinically).

Tissue toxicity is normally identified with additives added to the arrangement. Fundamental toxicity is likewise uncommon and is identified with the blood level of the medication optional to absorption from the site of infusion. Fundamental toxicity goes from lightheadedness, tinnitus to seizures and CNS/cardiovascular breakdown.

ALLERGY

Genuine allergy from neighborhood sedatives is extremely uncommon. Most responses are from ester class, ester hydrolysis (ordinary digestion) which prompts arrangement of PABA - like mixtures. Patients with past intravascular infusions organization reports the event of allergy.

REGIONAL ANESTHESIA

Regional anaesthesia renders a specific area of the body, e.g. foot, arm, lower extremities, insensate to stimulus of surgery or other instrumentation. Regional anaesthesia is useful in many aspects, provides anesthesia for a surgical procedure, provides analgesia post-operatively or during labor and delivery and helps in diagnosis or therapy for patients with chronic pain syndromes.

Types

- Topical
- Intravenous block ("Bier" block)
- Peripheral (named) nerve, e.g. radial n.
- Plexus - brachial, lumbar
- Central neuraxial - epidural, spinal

Topical Anesthesia

Topical anaesthesiaare application of local anesthetic to mucous membrane (cornea, nasal/oral mucosa). These are used for awake oral, nasal intubation, superficial surgical procedure. The technique is easy to be used with minimal equipment requirement. Topical anaesthesia when provided in large doses can lead to toxicity.

Intravenous (Bier's) block

For biers block, tourniquet is applied to an extremity that has been exsanguinated in order to isolate from the central situation for injecting local anaesthetic solutions into the venous system.

Peripheral nerve block

In peripheral nerve block, local anesthetic agent is Injected near the course of a named nerve. This nerve block is used in surgical procedures in the distribution of the blocked nerve. It requires relatively small dose of local anesthetic to cover large area and is rapid in onset. The procedure is complex and may lead to neuropathy.

Plexus Blockade

In plexus blockade, local anesthetic agent is injected adjacent to a plexuss, e.g cervical, brachial or lumbar plexus. This can be given for careful sedation or post-employable absense of pain in the dissemination of the plexus. Barricade covers an enormous space of sedation with generally huge portion of specialist. Technique is in fact complex, has potential for poisonousness and may prompt neuropathy.

Central neuraxial blockade – "Spinal"

In this blockade, Injection of local anesthetic is done into CSF. The blockade is used for profound anesthesia of lower abdomen and extremities. The technique is easy (LP technique), high success rate, rapid onset. Various disadvantages are "high spinal", hypotension due to sympathetic block, post-dural puncture headache.

Central Neuraxial Blockade - "epidural"

The blockade involves injection of local anesthetic into the epidural space at any level of the spinal column. This blockade is used for anesthesia/analgesia of the thorax, abdomen, lower extremities. Controlled onset of blockade, long duration of anesthesia can be obtained when catheter is placed. The block provides post-operative analgesia. Technically the procedure is complex, toxicity may occur and "spinal headache".

CONCLUSION

Local anaesthetic techniques are easier to perform and avoids potential complications and limit soft tissue anaesthesia. Local anaesthetic doses must be controlled. Local anaesthesia remains the backbone of pain control in surgeries. It has implications both in general as well as dental surgeries. Research can be continued to seek new and better means of managing pain associated with many surgeries treatment.

Conflict of Interest

Authors declare that there is no conflict of interest.

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