LOCAL ANESTHESIA IN DENTISTRY NONA SMOLAREK, MD, PHD



METHODS OF PAIN CONTROL

Solocal anesthetic agents
Solocal anesthetic agents
Solocal anesthetic agents
Solocal anesthesia
Solocal anesthesia

LOCAL ANESTHESIA

50 most frequently used in dentistry

- so local anesthesia <u>temporarily</u> blocks the normal generation and conduction action of the nerve impulses
- *so local anesthesia is obtained by <u>injecting</u> the anesthetic agent near the nerve in the area intended for dental treatment*
- *produce loss of sensation to pain in a specific region without the loss of consciousness*

DEFINITION OF LOCAL ANESTHETICS

The term is used to describe compounds, which in a suitable form and appropriate dose applied to the skin or mucous membranes (surface analgesia), injected locally into the tissue near the peripheral nerve or for the nerve, interrupts impulse conduction in this nerve, preventing receiving and conduction of centripetal impulses in a limited area.

DEFINITION OF LOCAL ANESTHETICS

so as a result of occuring a local anesthesia processes the incoming information do not reach the pain centers of the brain cortex, receiving conscious experience of pain

so effect is reversible, anesthesia persists differently long, depending on the properties of the compound, rate of absorption, metabolism and excretion

DEFINITION OF LOCAL ANESTHETICS

 used even in small concentrations, can cause damage to the nerve fibers causing them irreversible changes
 they differ substantially from analgesics, whose mechanism of action is associated with influence on the specific structures of the central nervous system
 ensure the perform of procedures with full consciousness of patient, which allows to keep permanent contact with him EFFECTS OF LOCAL ANESTHETICS

ກລnalgesia ການeakness of tension of striated muscles (relaxatio)

new weakness of vegetative reflexes (hyporeflexio)

DESIRABLE PROPERTIES OF ANESTHETICS

- not solubility in water and body fluids
- not good penetration into the tissue
- *so the stability of the structure despite of the sterilization processes*
- napid, reversible and sufficient long action
- no large therapeutic spread
- ninimal toxicity, no general adverse effects
- substance and its metabolites should not produce allergy
- nonirritating to the tissues in the area of the injection
- so action limited to pain fibers

MECHANISM OF ACTION

stabilization of cell membranes

so inhibition of depolarization and generation of action potential

mpreventing the conduction of active state in nerve fibers

MECHANISM OF ACTION

inhibition of penetration of sodium ions into the cell, the main process of conditioning depolarization
 inhibiting the release of calcium ions from storage locations in the cell

Inflamed tissue has a lower pH (5.0-5.5), thus responds poorly to anesthesia carried out by substances that are weak bases.

ORDERING OF INHIBITION OF NERVE FIBERS

ຣາ sympathetic fibers ຣາ fibers conducting sensation of cold ຣາ fibers conducting pain sensation ຣາ fibers conducting touch ຣາ fibers conducting deep feeling

ORDERING OF INHIBITION OF NERVE FIBERS

so the sensitivity of individual types of cells and nerve fibers is different

so is due to the thickness of the fibers - the most sensitive are the thinnest fibers

so in mixed sensory fibers are inhibited in the following order:

✓ fibers conducting pain sensation
 ✓ fibers conducting temperature
 ✓ fibers conducting touch

so topical
so infiltration
so block anesthesia
so periodontal ligament

so topical anesthesia provides a temporary numbing effect on nerve endings that are located on the surface of the oral mucosa so supplied as:
> ointments

≻ liquids> sprays

<u>so infiltration</u> is achieved by injecting the solution directly into the tissue at the site of the dental procedure

Somost frequently used in <u>maxillary</u> teeth
Soused as a secondary injection to block gingival tissues surrounding the <u>mandibular</u> teeth

Block_anesthesia

 so the solution is injected near a major nerve, and the entire area served by that nerve is numbed
 so required for most mandibular teeth

Inferior alveolar nerve block

- *so injecting the anesthetic solution near the branch of the inferior alveolar nerve close to the mandibular foramen*
- so type of injection for half of the lower jaw, including the teeth, tongue, and lip

Incisive nerve block

injection given at the site of the mental foramenused for mandibular anterior teeth or premolars

Periodontal ligament

alternative infiltration anesthesia method anesthetic solution is injected directly into the periodontal ligament and surrounding tissues

LOCAL ANESTHETIC CAUTIONS

so injection into a blood vessel so infected area solocalized toxic reaction systemic toxic reaction *so temporary numbness* so paresthesia

DURATION OF ACTION

Length of time from induction until the reversal process is complete. 50 short-acting: > local anesthetic agent lasts less than 30 minutes

ntermediate-acting:

Iocal anesthetic agent lasts about 60 minutes

so long-acting:

Iocal anesthetic agent lasts longer than 90 minutes

LOCAL ANESTHETICS MOLECULES

all LA's are amphipathic
molecules have lipophilic and hydrophilic characteristics
classes are amides and esters
amides: metabolised in liver
esters: metabolised in plasma
esters are more likely to cause allergic reaction

so very few ester local anesthetics in use today

LOCAL ANESTHETICS MOLECULES

so chemical structure of local anesthetics have an amine group on one end connect to an aromatic ring on the other



LOCAL ANESTHETICS MOLECULES

- The amine end is <u>hydrophilic</u> (soluble in water), anesthetic molecule dissolve in water in which it is delivered from the dentist's syringe. It's also responsible for the solution to remain on either side of the nerve membrane.
- **So The aromatic end is <u>lipophilic</u> (soluble in lipids). Because nerve cell is made of lipid bilayer it is possible for anesthetic molecule to penetrate through the nerve membrane.**
- **So** The trick the anesthetic molecule must play is getting from one side of the membrane to the other.

FACTORS AFFECTING LOCAL ANESTHETICS

- Increasing the lipid solubility leads to faster nerve penetration, block sodium channels, and speed up the onset of action.
- **50** The more tightly local anesthetics bind to the protein, the longer the duration of onset action.
- *LA's 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.*
- Decrease in pH shifts equilibrium toward the ionized form, delaying the onset action.

FACTORS AFFECTING LOCAL ANESTHETICS

so vasocontriction prolongs the duration of an anesthetic agent by decreasing the blood flow in the immediate area of the injection

Secreases bleeding in the area during surgical procedures
 Secreases bleeding in the area during surgical procedures

so most frequently epinephrine, also levonordefrin (αmethylnorepinephrine), felypressin (non-catecholamine)

CONTRAINDICATIONS FOR THE USE OF VASOCONSTRICTORS

so unstable angina

recent myocardial infarction
recent coronary artery bypass surgery
untreated or uncontrolled severe hypertension
untreated or uncontrolled congestive heart failure
hyperthyreosis
epilepsy

TOXIC EFFECTS OF LOCAL ANESTHETIC

so is proportional to the quantity of agent introduced into the body so the result of an overdose *socumulation of the drug modisturbances of excretion* sensitiveness so toxicity increases in geometric progression

| Comparison of forms of local anesthetics overdose | | | |
|---------------------------------------------------|-----------------------------------------------------------------|----------------------------|----------------------------------------------|
| Cause of overdose | The beginning and the severity of symptoms | The duration of symptoms | The main methods of preventing |
| Intravascular injection – <u>often</u> | seconds!!! the most intensified | 2 - 3 minutes | aspiration and slow injection |
| Too high a dose - <u>the most often</u> | 5 – 30 min the gradual onset with increasing intensity | 5 - 30 minutes | the administration of minimum doses |
| Slow biotransformation - <u>rarel</u> y | 1 – 3 hours the gradual slow onset | potentially long | appropriate assessment of the patient |
| Slow elimination - <u>the least often</u> | several hours | potentially the longest | appropriate assessment of the patient |

TOXIC EFFECTS OF LOCAL ANESTHETIC - SYMPTOMS

so anxiety, somnolence
so vertigo, tremor of muscles
so enhanced muscle tension
so vomiting
so breathing and circulation disturbances, including the cardiac arrest and breathing

TOXIC EFFECTS OF LOCAL ANESTHETIC

so central nervous system
so autonomic ganglia
so neuromuscular junction
so cardiovascular system
so system of smooth and striated muscle

TOXIC EFFECTS TO THE CENTRAL NERVOUS SYSTEM

so initially stimulating effect

manxiety

so tremor progressing to convulsions

So after a period of strong stimulation, occurs CNS depression, sometimes leading to death due to INHIBITION OF RESPIRATORY CENTER!

TOXIC EFFECTS TO THE CARDIOVASCULAR SYSTEM

reduce the conduction in the electrical conduction system of the heart
reduce myocardial irritability
reduce the contractile force of cardiac muscle

LOCAL ANESTHETICS CAN CAUSE ANAPHYLACTIC SHOCK!

ANAPHYLACTIC SHOCK -PREVENTION

so is recommended to perform test of sensitization, especially in patients with suspected allergic interview

ANAPHYLACTIC SHOCK -PREVENTION

so conjunctival test:

✓ 1-2 drops of 1% solution into the conjunctival sac - the result after 5-10 min

nasal test:

 2-3 drops of 1% solution into the nasal cavity - acceleration pulse of 10/min after 15 min (positive test)

subcutaneous test:

0.1 ml of a 1% solution subcutaneously in the arm and the result is read after 20-30 min (redness and swelling - positive test)

TREATMENT OF POISONING LOCAL ANESTHETIC AGENTS

interval in the administration of the drug
horizontal position of the patient
unblocking and maintaining patency of the airway
potential application of oxygen
in the event of cardiac and respiratory arrest, lead resuscitation according to the general rules

TREATMENT OF POISONING LOCAL ANESTHETIC AGENTS

so hypersensitivity manifested local allergic reaction or asthma attack or anaphylactic shock

should be treated:

✓ oxygen

✓ intravenous infusion fluids

✓ benzodiazepines (diazepam) – very carefull

✓ suxamethonium (succinylcholine) – 0,1-0,5 mg iv

TREATMENT OF POISONING LOCAL ANESTHETIC AGENTS

should not be administered any centrally acting stimulants (analeptics)

so anaphylactic shock should be treated according to the rules proceedings in emergency situations

PHENTOL&MINE MESYL&TE (OR&VERSE*)

- *so alpha-adrenergic receptor antagonist*
- so local anaesthetic reversal agent for adults and children
- so designed to reverse the local vasoconstrictor properties used in many local anesthetics to prolong anesthesia
- so accelerate the reversal of the lingering soft-tissue numbness
- so administered via standard dental cartridge in dose 0,4 0,8 mg
- *it will be injected into the same site as the local anesthetic was previously deposited*
- neduces duration of anaesthesia by 50%

DEVELOPMENT OF GENERAL AND LOCAL ANESTHESIA

- took place in Western Europe from 1750 to 1850
 chemists and physicians collected sample of coca leaves for
 - experiments
- *so isolated active principle of coca leaf, synthesized to a drug for patients to feel more relief of pain when taking surgeries*
- *in 1860, German chemist Albert Niemann successfully isolated the active principle of coca leaf; he named it cocaine*

DEVELOPMENT OF GENERAL AND LOCAL ANESTHESIA

- Niemann discovered the effect of numbress of the tongues caused by alkaloid in 1860
- **based on Niemann's discovery, Russian physician Basil Von Anrep did experiments on animals, such as rats, dogs, and cats**
- *be injected small quantity of 1% solution to his tongue; tongue became insensitive*
- *be concluded cocaine is a good drug for surgical anesthetic*
- William Steward Halsted and Richard John Hall developed the inferior dental nerve block techniques for dentistry
- sensory nerve blockade was first described by Halsted in 1884

DEVELOPMENT OF GENERAL AND LOCAL ANESTHESIA

- *more physicians began to do research of cocaine in the clinic trials*
- *so the physician Sigmund Freud used the stimulant effect of cocaine to treat the morphine addiction in patients*
- n ophthalmologist Carl Koller realized the importance of the alkaloid's anesthetic effect on mucous membranes
- *in 1884, he used the first local anesthetic on a patient with glaucoma*
- Freud, Halsted, and Koller became addicted to the drug through selfexperimentation

SIDE EFFECTS OF COCAINE

Minor: so addiction so intoxication Severe:

so death

PROCAINE REPLACED COCAINE

Procaine replaced cocaine
so in 1898, Professor Heinrich Braun introduced procaine as the first derivative of cocaine, also known as the first synthetic local anesthetic drug
so trade name is Novocaine®.

PROCAINE PROBLEMS

- so took too long to set (i.e. to produce the desired anesthetic result)
 so wore off too quickly, not nearly as potent as cocaine
 so classified as an ester; esters have high potential to cause allergic reactions
- so caused high concentration of adrenaline, resulted in increasing heart rate, make people feel nervous
- so today, procaine is not even available for dental procedures

LIDOCAINE

n 1940, the first modern local anesthetic agent was lidocaine

so belongs to the amide class, cause little allergenic reaction; it's hypoallergenic

sets on quickly and produces a desired anesthesia effect for several hours

LIDOCAINE

- *so effect four times stronger and 2 times longer than procaine*
- so anesthesia occurs quickly (after 30 60 s) and lasts for 30 60 minutes
- so effective in all types of local anesthesia
- *the addition of vasoconstrictor causes significant prolongation of the action (approximately 50%)*
- so at one time should not exceed a dose of 200 mg (3 mg/kg bw) and with the addition of vasoconstrictor a dose of 500 mg (7mg/kg bw)

EXPRESSING & % SOLUTION IN MG/ML

So Example: 2% lidocaine 2% = 20 mg/mlSo 1 cartridge has 1.8 ml of fluid $\frac{20 \text{ mg}}{1 \text{ ml}} = \frac{X}{1,8 \text{ ml}}$ $X = 1,8 \times 20 \text{ mg} = 36 \text{ mg of drug/cartridge}$

PRILOCAINE

so less toxic than lidocaine

so anesthesia occurs very quickly (after 2 min) and lasts longer than after lidocaine (about 2 hours)

so for all types of anesthesia, and can be used in combination with vasoconstrictor (epinephrine, felypressin)

so a single dose should not exceed 8 mg/kg bw

MEPIVACAINE

- so chemical structure and pharmacological properties like lidocaine
- nesthesia occurs quickly (after 3-5 min) and lasts for 90 150 minutes
- to infiltration is used in a concentration of 0.5 1%, to nerve blockade 2 - 3%
- so a single dose should not exceed 7mg/kg bw
- so the addition of epinephrine only slightly longer duration of action, but reduces the side effects
- so used especially when there are contraindications to the use of vasoconstrictors

BUPIVACAINE

- synthetic long-acting amide local anesthetic agent
- *so also shows a analgesic activity is used to treat pain e.g. during childbirth, in the postoperative period*
- *so effects on sensory fibers is stronger than motor fibers*
- anesthesia occurs after 1–10 min and lasts for 3 8 hours onset and the time of action depends on the dose and the type of anesthesia
- so can be used in combination with vasoconstrictor (epinephrine)
- naximum single dose is 2 mg/kg

ARTICAINE

- 🔊 quickly and potent anesthetic
- so gives the deeper and stronger anesthesia than lidocaine at a dose reduced by half
- so used in combination with vasoconstrictor (epinephrine)
- so anesthesia occurs quickly (after 1-3 min) and lasts for 45 240 minutes it depends on concentration of epinephrine
- so a single dose should not exceed 7mg/kg bw (with epinephrine)
- so used in infiltration and block anesthesia

INHALATION SEDATION

So Nitrous oxide/oxygen $(\mathcal{N}_2 O/O_2)$ is a combination of these gases that the patient inhales to help eliminate fear and to help the patient relax

n History - dates back to 1844

> dr. Horace Wells first used it on his patients

So Effects

non addictive

- > easy onset, minimal side effects, and rapid recovery
- produces stage I anesthesia
- I dulls the perception of pain

CONTRAINDICATION OF USING N20/02

pregnancy: first trimester
 nasal obstruction: problems inhaling through the nose
 emphysema: increased O₂
 multiple sclerosis: breathing difficulties
 emotional stability: altered perception of reality

EXPOSURE TO NITROUS OXIDE

used only for patient treatment
never administered for recreational purposes
how to reduce N₂O hazards to dental personnel

- use a scavenger system
- use a patient mask that fits well
- > discourage patients from talking
- > vent gas outside the building
- > routinely inspect equipment and hoses for leaks
- ▶ use an N₂O monitoring badge system

PATIENT PREPARATION FOR INHALATION SEDATION

so review health history so obtain base-line vital signs *so describe the procedure of administering the gases* so describe the use of the mask and the importance of nasal breathing so describe the sensations that the patient will experience *preassure the patient*

ANTIANXIETY AGENTS

so for the relief of anxiety

sedatives solutions

so criteria for use:

patients are very nervous about a procedure
 procedures are long or difficult
 mentally challenged patients
 very young children requiring extensive treatment

SEDATIVES

Sone Commonly prescribed: ➤ alprazolam Clorazepate dipotassium > chlordiazepoxide HCl ➤ diazepam ➢ lorazepam ▷ oxazepam ▷ bromazepam

INTRAVENOUS SEDATION

Antianxiety drugs that are administered intravenously continuously throughout a procedure at a slower pace, providing a deeper stage I analgesia.

INTRAVENOUS SEDATION

80 Patient assessment

- a health history, physical examination, and signed consent are performed
- > baseline vital signs are taken and recorded
- oximetry and electrocardiogram are completed and recorded
 weight taken and recorded for dose determination

INTRAVENOUS SEDATION

so patient monitoring

no physiologic measurements taken and recorded every 15 minutes

- level of consciousness
- respiratory function
- > oximetry
- blood pressure
- heart rate
- cardiac rhythm

most often used drugs – midazolam, propofol, ketamine, opioids

GENERAL ANESTHESIA

A controlled state of unconsciousness in which there is a loss of protective reflexes, including the ability to maintain an airway independently and to respond appropriately to physical stimulation or verbal command.

This controlled state in loss of consciousness, produces stage III general anesthesia.

GENERAL ANESTHESIA

Pharmacologic make-up so combination of gases

- $\succ \mathcal{N}_2 O/O_2$
- halothane or enflurane mixtures

so intravenous agents such as thiopental sodium and methohexital sodium