

University of Manitoba
School of Dental Hygiene
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PAIN MANAGEMENT WORKSHOP



UNIVERSITY
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Rady Faculty of
Health Sciences

Presenter Disclosure

- **Faculty Member:** Diane Girardin, dip. DH, RDH
- **Relationships with commercial interests:**
 - None to report

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Disclosure of Commercial Support

- This program has not received any financial support from industry.
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Rationale for Pain Control in Dental Hygiene Care

- Pain Control
 - Initial Therapy
 - Tooth Sensitivity
- Control of Bleeding

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When to Use Local Anesthetics in Dental Hygiene Practice

- Client exhibits low pain threshold
- Treatment to be performed is extensive enough to warrant anesthesia i.e. sextant or quadrant scaling
- Periodontal therapy including root planing and/or soft tissue curettage
- Localized areas of more extensive periodontal involvement



When to Use Local Anesthetics in Dental Hygiene Practice

- Periodontal therapy generates abundant bleeding making visibility difficult
- Any instance where procedure, as assessed by the clinician, will produce **PAIN!**

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Reasons for Hesitation to Use Pain Control

- Time
- Inconvenience
- Lack of Knowledge

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Increasing Usage

- Awareness
- Increase Knowledge of Pain Management
- Appropriate Training

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Desirable Properties of Local Anesthetics

- Sterile agents
- Stable in solution but will readily undergo biotransformation in the body
- Non-irritating to the tissues
- Will not cause permanent damage to the nerve structure
- Low systemic toxicity

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Desirable Properties of Local Anesthetics

- Low potential for producing allergic reactions
- Adequate potency without use of harmful concentrations
- Rapid onset of anesthesia
- Long enough duration to permit completion of procedure

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Important Facts for the Administration of Local Anesthesia

- Thorough knowledge of the client's medical history **essential**
- Knowledge of complications that could occur
- Thorough knowledge of anesthetic agents and vasoconstrictors to enable appropriate selection of agents

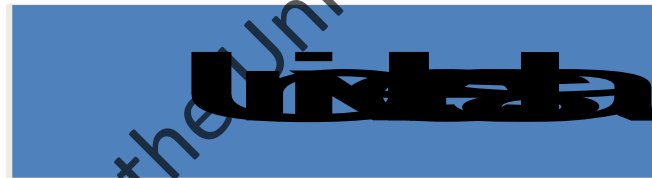
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Important Facts for the Administration of Local Anesthesia

- Certain agents are contraindicated for certain medical histories
 - i.e., uncontrolled hyperthyroidism (**no vasoconstrictors**)
- Elderly clients are on multiple meds therefore must have a thorough understanding of drug interactions

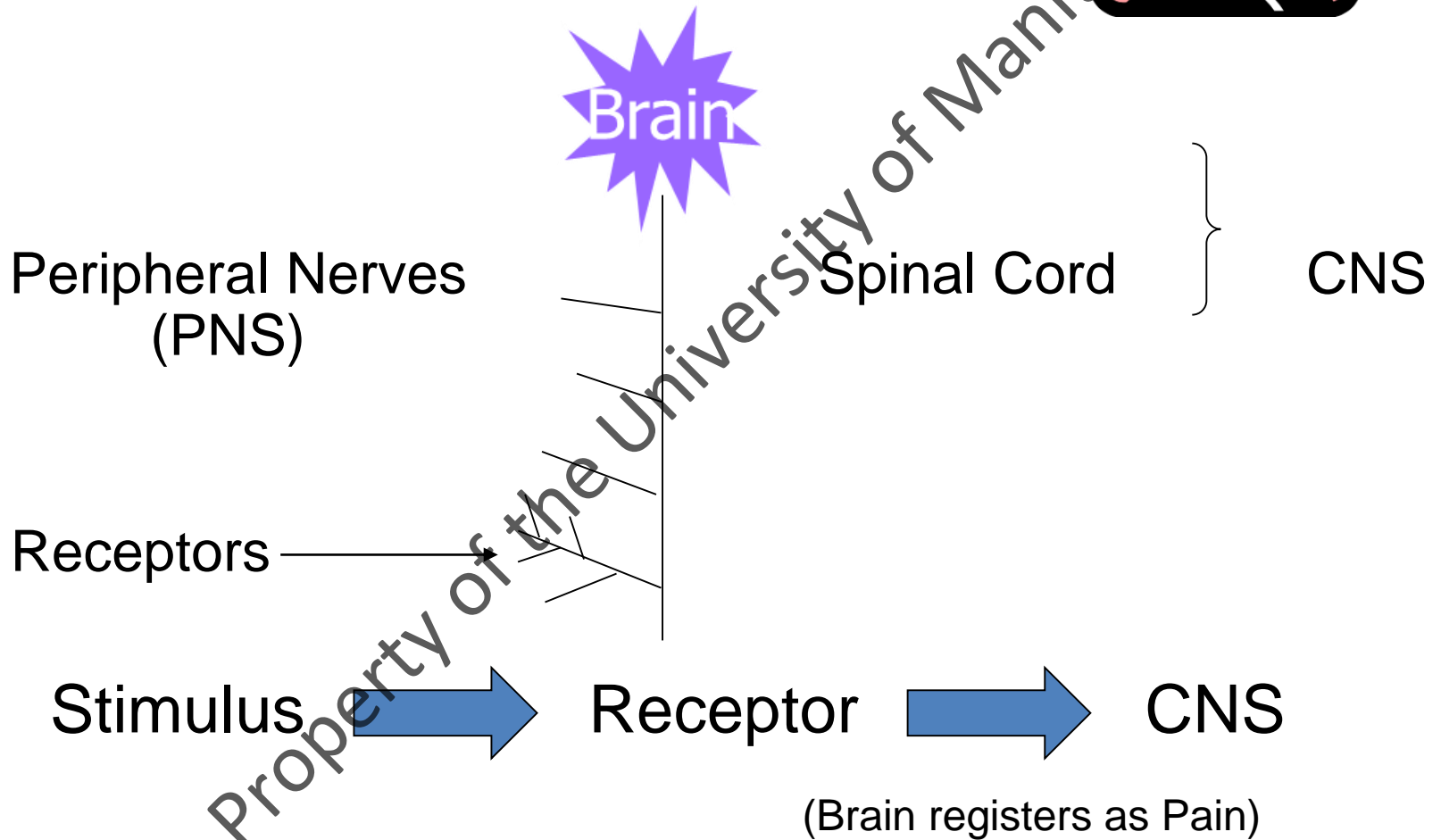




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Pain Perception



Pain Reaction



PERSON'S PERCEPTIONS

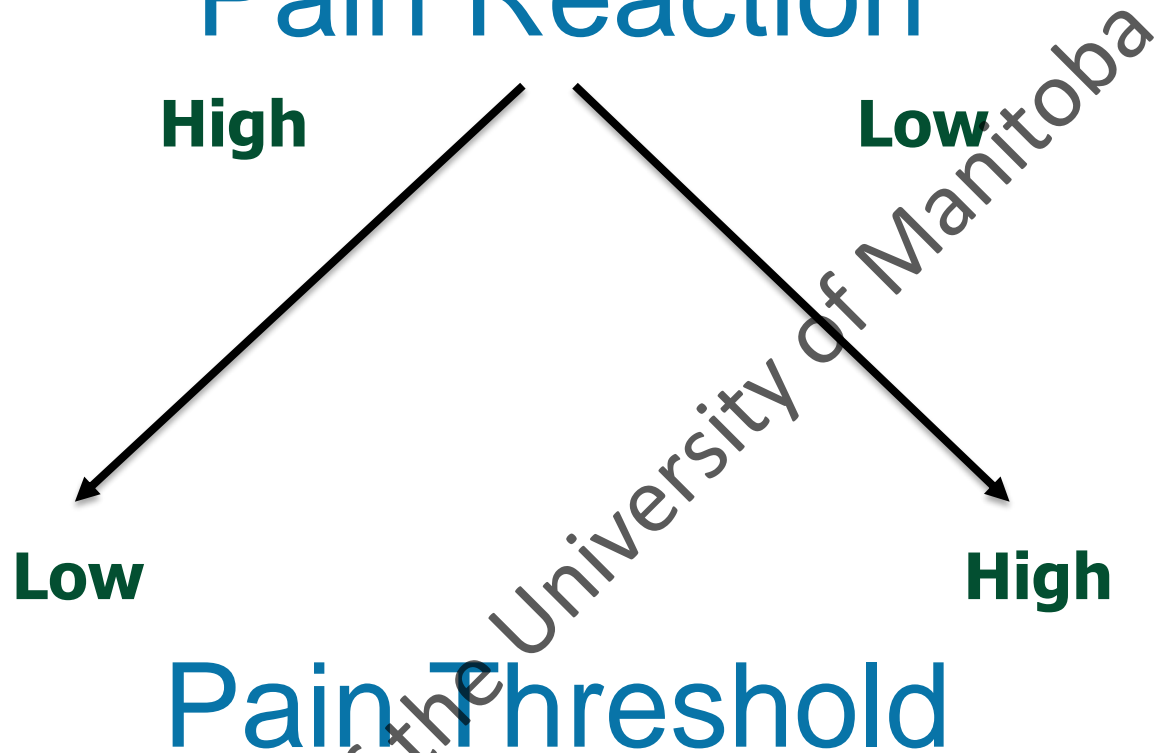


PERSON'S REACTIONS TO THE
PAIN

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Pain Reaction

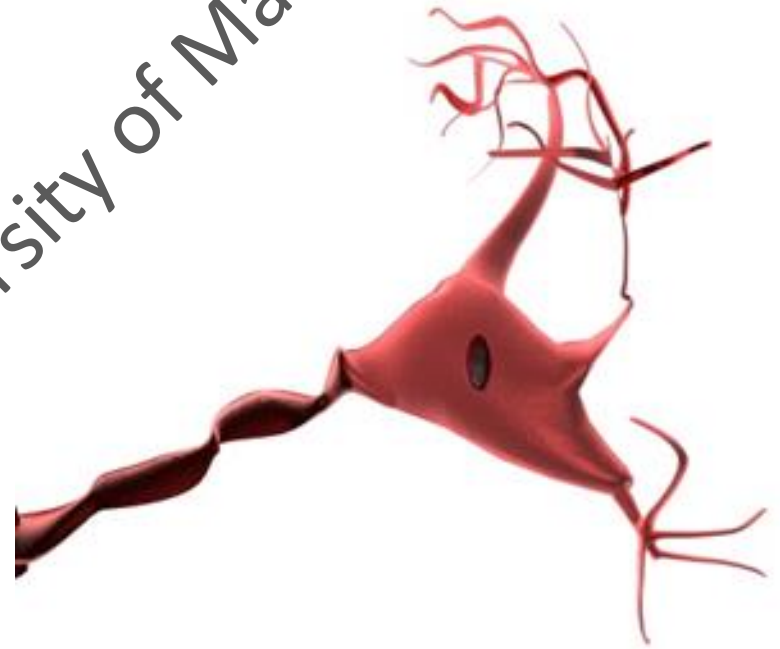


NOTE: pain reaction and the pain threshold are **inversely** related



Mechanism of Nerve Impulse

- Polarization
- Depolarization
- Repolarization
- Refractory Period



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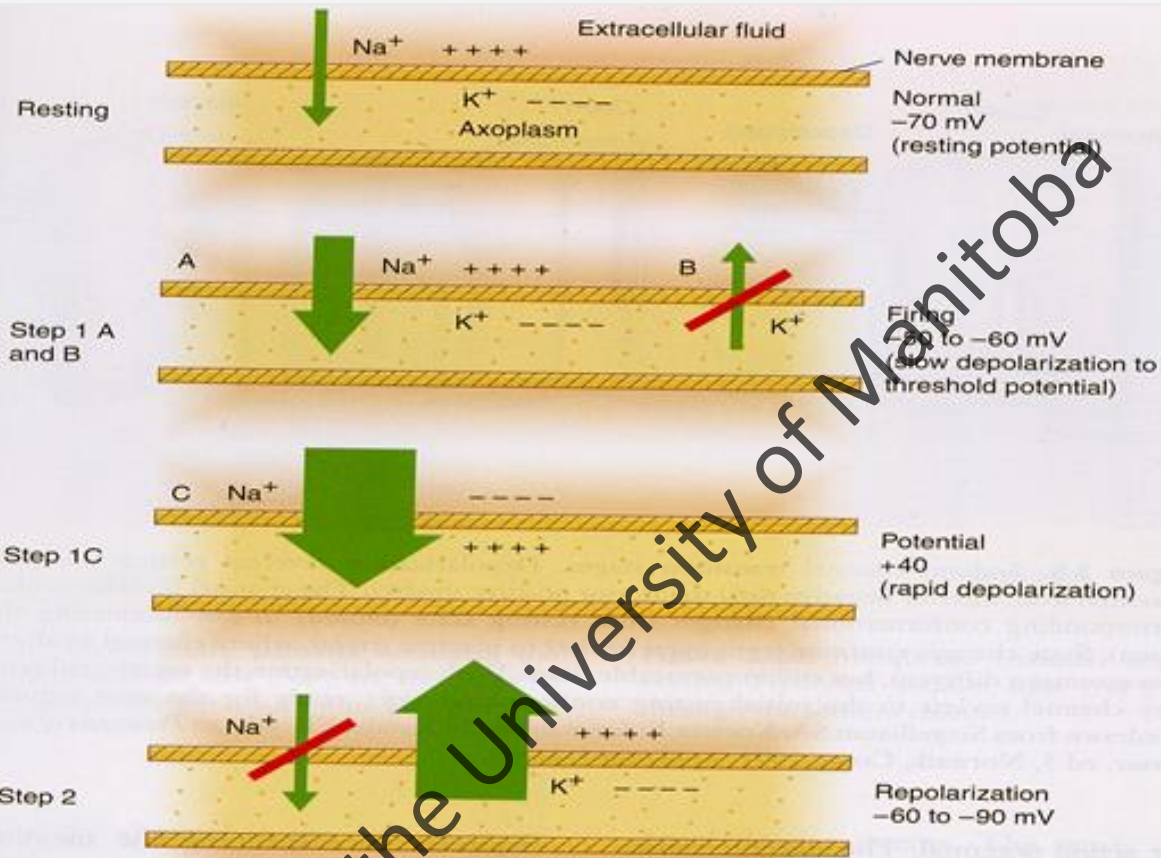


Figure 1-7. Top, Resting potential. Step 1, A and B, Slow depolarization to threshold. Step 1, C, Rapid depolarization. Step 2, Repolarization.

Polarization

Polarization is caused by relative impermeability of cell membrane to sodium



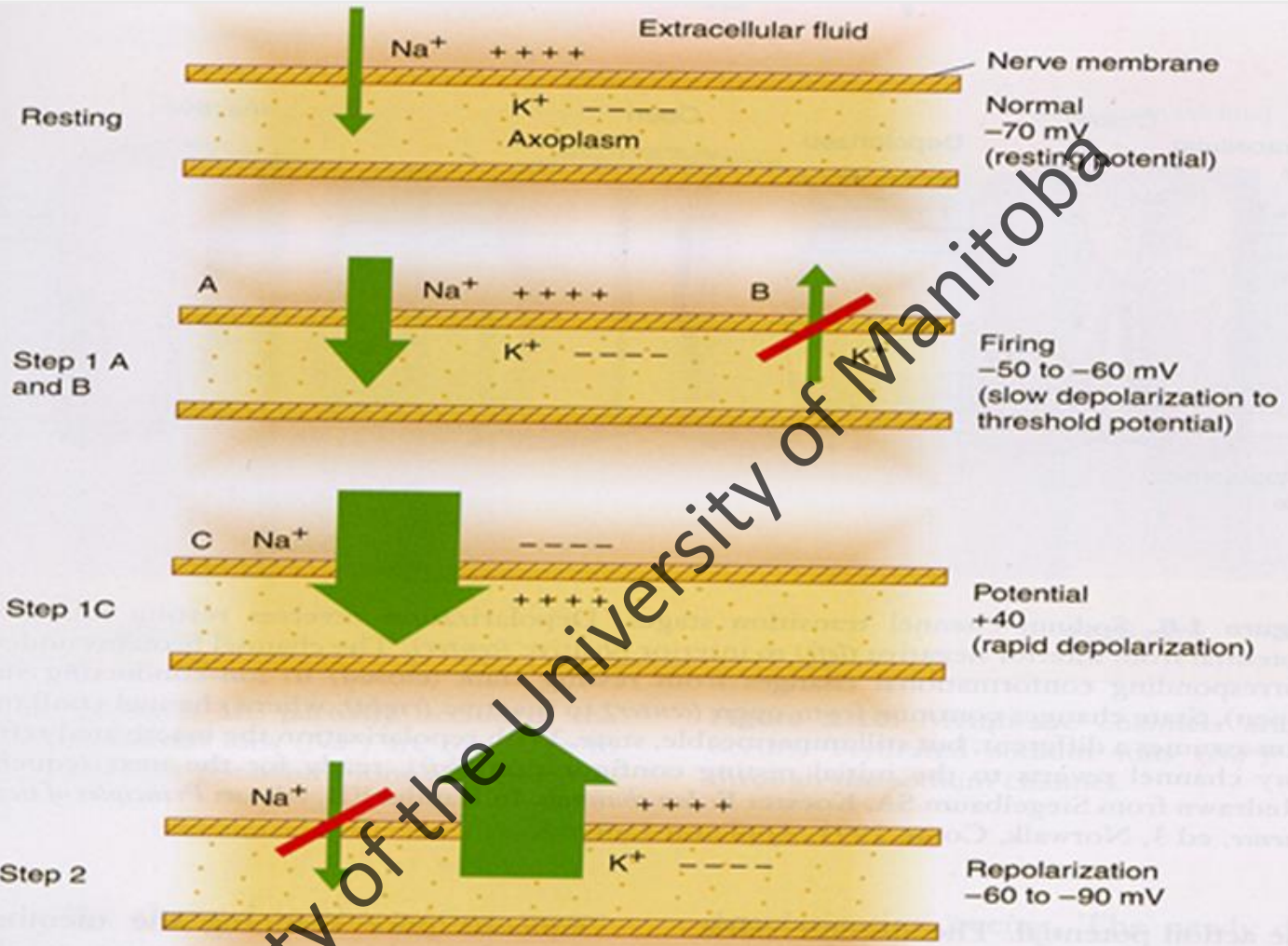


Figure 1-7. Top, Resting potential. Step 1, A and B, Slow depolarization to threshold. Step 1, C, Rapid depolarization. Step 2, Repolarization.

Depolarization



Depolarization

1. Stimuli alter permeability of cell membrane
 2. Sodium allowed into axoplasm
 3. Potassium goes out
 4. Positive charge in axon
 5. Negative charge outside membrane
 6. Threshold Potential
 7. Firing Potential
- } Point of Change

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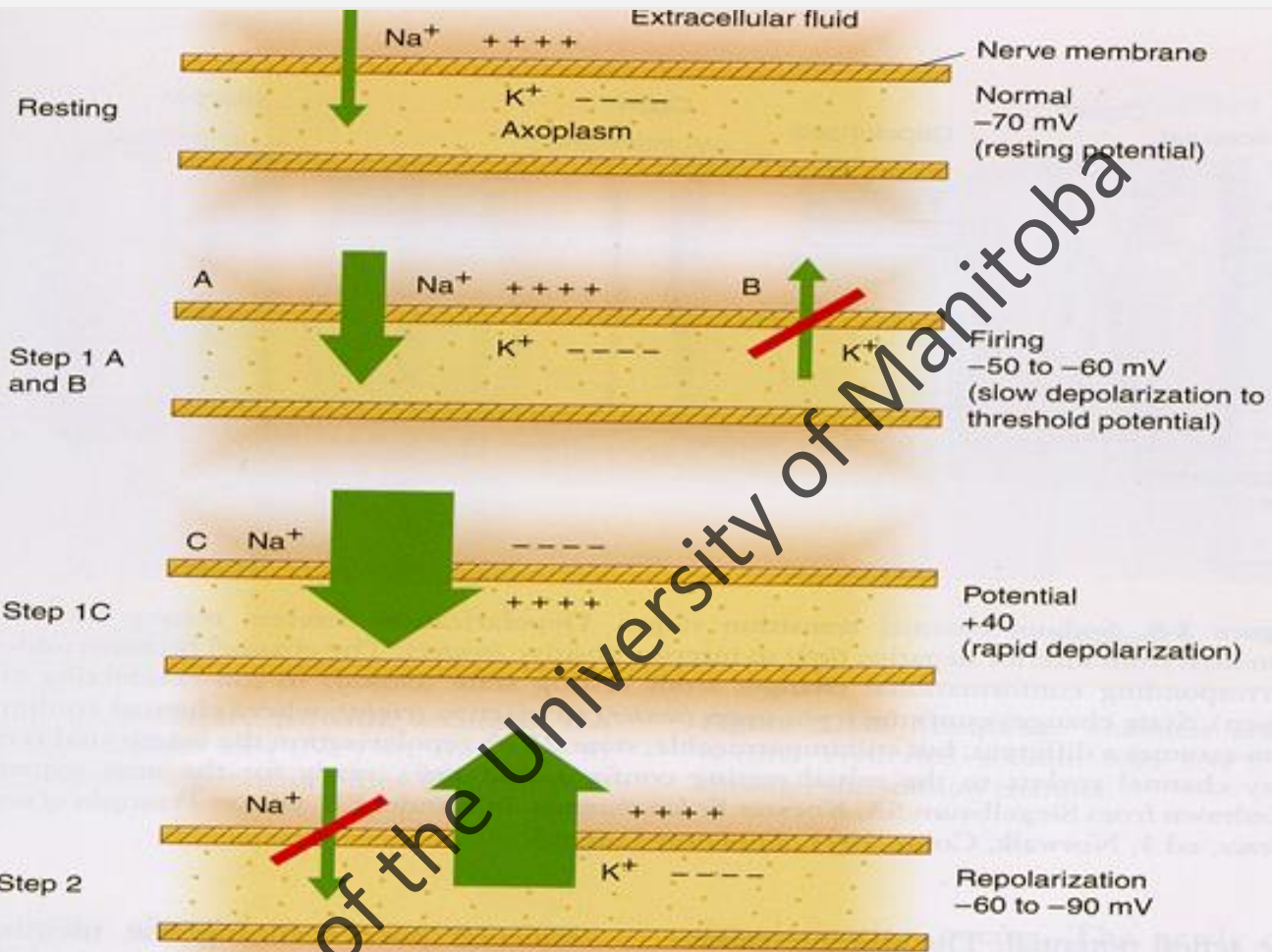


Figure 1-7. Top, Resting potential. Step 1, A and B, Slow depolarization to threshold. Step 1, C, Rapid depolarization. Step 2, Repolarization.

Repolarization



Repolarization

1. Membrane again impermeable to sodium
2. Potassium pumped back into axon
3. Sodium pumped OUT by **sodium pump**
4. Normal membrane potential restored (-70mV)

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Refractory Period

1. **Absolute Refractory Period:** nerve is unable to respond to another stimulus.
2. **Relative Refractory Period:** follows the ARP but can only be initiated by a stronger than normal impulse.

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How Local Anesthetic Works

- Primary effect occurs during depolarization
 - Nerve membrane depolarizes
 - Anesthetic attaches to a specific receptor at the nerve membrane (located near the sodium channel)
 - No action potential develops

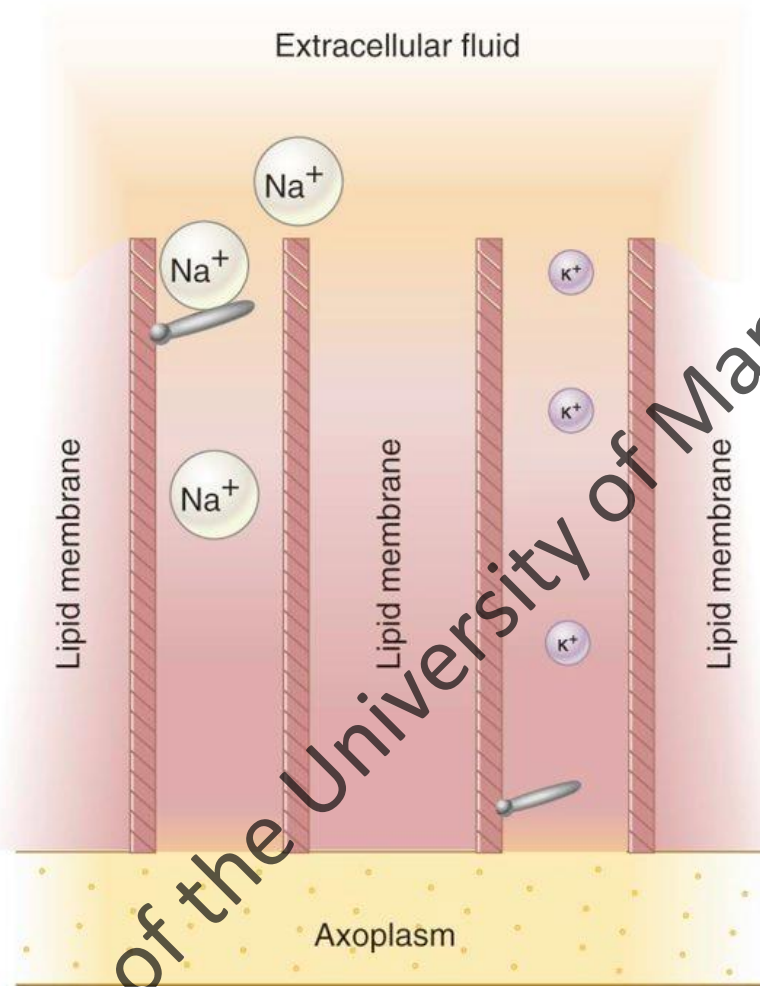
NOTE: In myelinated nerves where impulse travels between Nodes of Ranvier, anesthetic must cover 2-3 nodes (8-10mm of nerve)



Mechanism of Action

- Primary effect of local anesthetic is a decrease in the permeability of the nerve membrane to sodium ions
- Sequence of Action
 - Displacement of calcium ions from nerve receptor site
 - Binding of local anesthetic molecule to this receptor site
 - Blockade of the sodium channel
 - Decrease in sodium conductance
 - Depression of rate of electrical depolarization
 - Failure to reach Threshold Potential level
 - Lack of development of propagated action potential
 - Conduction blocked





Mechanism of Action

Primary effect of local anesthetic is a decrease in the permeability of the nerve membrane to sodium ions



Mechanism of Action

- An impulse that arrives at a blocked nerve segment is **stopped** because it is **unable to release the energy needed for continued propagation**.
- The nerve block produced by local anesthetics is called a “**nondepolarizing nerve block**”

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General Chemical Forms

- Most injectable local anesthetics are **tertiary amines**

Typical Local Anesthetic Structure

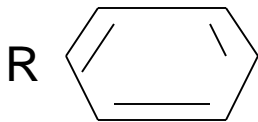
Benzoic Acid

Lipophilic Part

Intermediate Chain

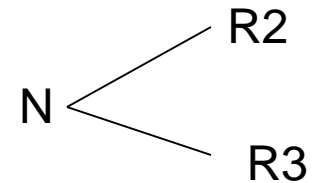
Hydrophilic Part

Ester

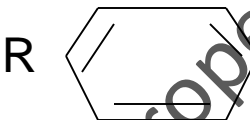


COOR 1

Ester

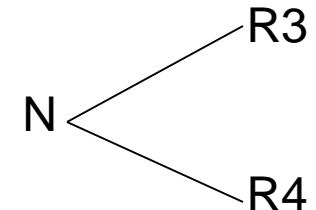


Amide



NHCO R2

Amide



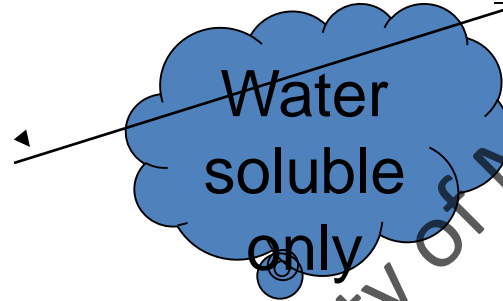
Chemical Form

- **Lipophylic** part is the largest part of the molecule and is derived from **benzoic acid** or aniline
- **Hydrophilic** part is an amino acid derivative of **ethyl alcohol** or **acetic acid**
- Local anesthetics without a hydrophilic part are not suitable for injection but are good as topical anesthetics
- **Intermediate hydrocarbon** chain determines whether it is an ester or amide linkage
- Local anesthetics are classified either as **esters** or **amides**

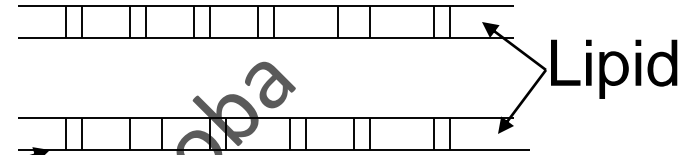


Local anesthetic

- Weak base
- Unionized
- Readily diffuses through lipids



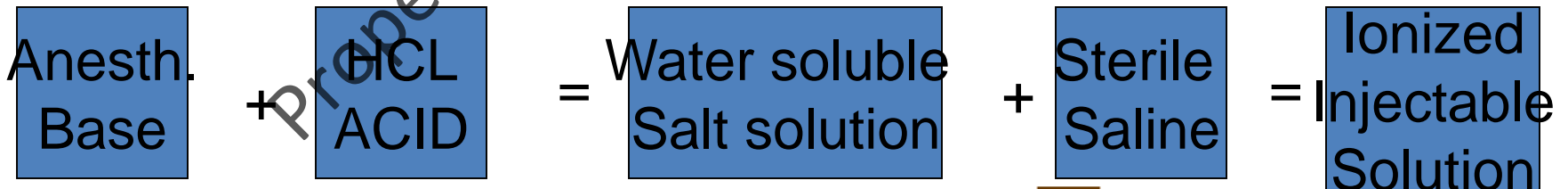
Nerve Sheath



Oral mucosal tissue (not lipid soluble)

BUT

Therefore, must alter the chemical structure of the anesthetic to get it across the tissues to the nerve!



pH

- Normal body pH = 7.0 – 7.4 (slightly basic)
- If BASE injected into a BASE -> little effect
- If ACID injected onto a BASE -> part of acid will neutralize into base until equilibrium is reached
- Therefore, anesthetics have acid (HCL) added to them to enable solution to MOVE through the tissues to the nerve
- When the nerve is reached only the ionized portion of the equilibrium yields the anesthetic property (activity)



pH

If anesthetic injected into “inflamed” tissues
(pH=5.0) → No reaction → No Movement!

Why???

1. Acid solution ↔ Acid tissues
2. More dilated blood vessels carry away anesthetic!



PKa (Dissociation Constant)

- PKa is the measure of a molecule's affinity for H⁺ ions
- When:
 - $\text{pH} = \text{PKa}$
 - $\frac{1}{2}$ the drug is in Cationic form (positively charged RNH⁺)
 - AND
 - $\frac{1}{2}$ the drug exists as a Free Base (uncharged RN)
- Proportion of each form depends on the pH of the solution or surrounding tissues



PKa (Dissociation Constant)

Uncharged RN	Helps diffuse agent into nerve cell membrane
Charged RNH⁺	Allows binding of agent to nerve cell membrane
If high pH	Lower H ⁺ = more RN's (better diffusion), but less RNH ⁺ (poor binding)
If low pH	Higher H ⁺ = less RN's (poor diffusion), but more RNH ⁺ (better binding)



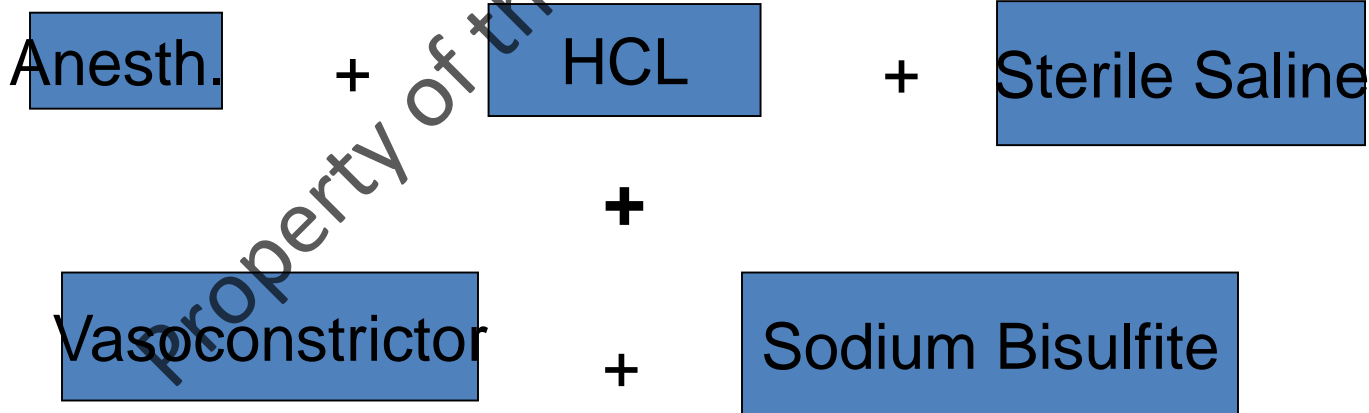
PKa (Dissociation Constant)

- Rate of onset of clinical action is related to the PKa of the local anesthetic and pH of the extracellular fluid
- They determine the ease of movement of local anesthetic to the nerve sheath
- Ideally, they should be equal!



Local Anesthetics with Vasoconstrictors

- Are acidified by manufacturers to retard oxidation of the vasoconstrictor
- This prolongs the drug's effectiveness
- Antioxidant agent: sodium bisulfite
- If pH is lowered, onset of action is slower



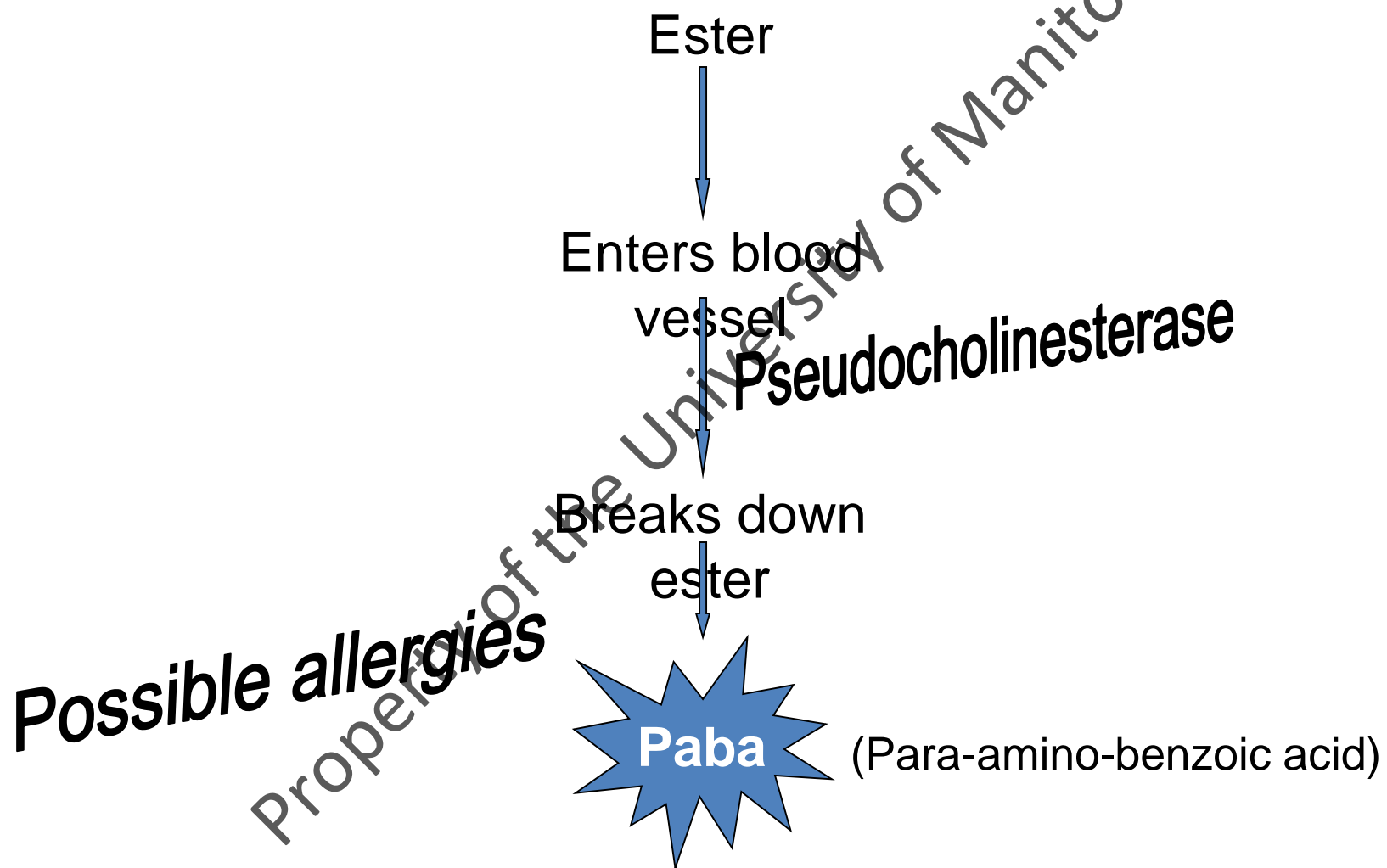
Esters VS Amides

- Anesthetics come in two categories:
 1. **Esters:** biotransformed by blood plasma
 2. **Amides:** biotransformed by liver

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Biotransformation of Esters



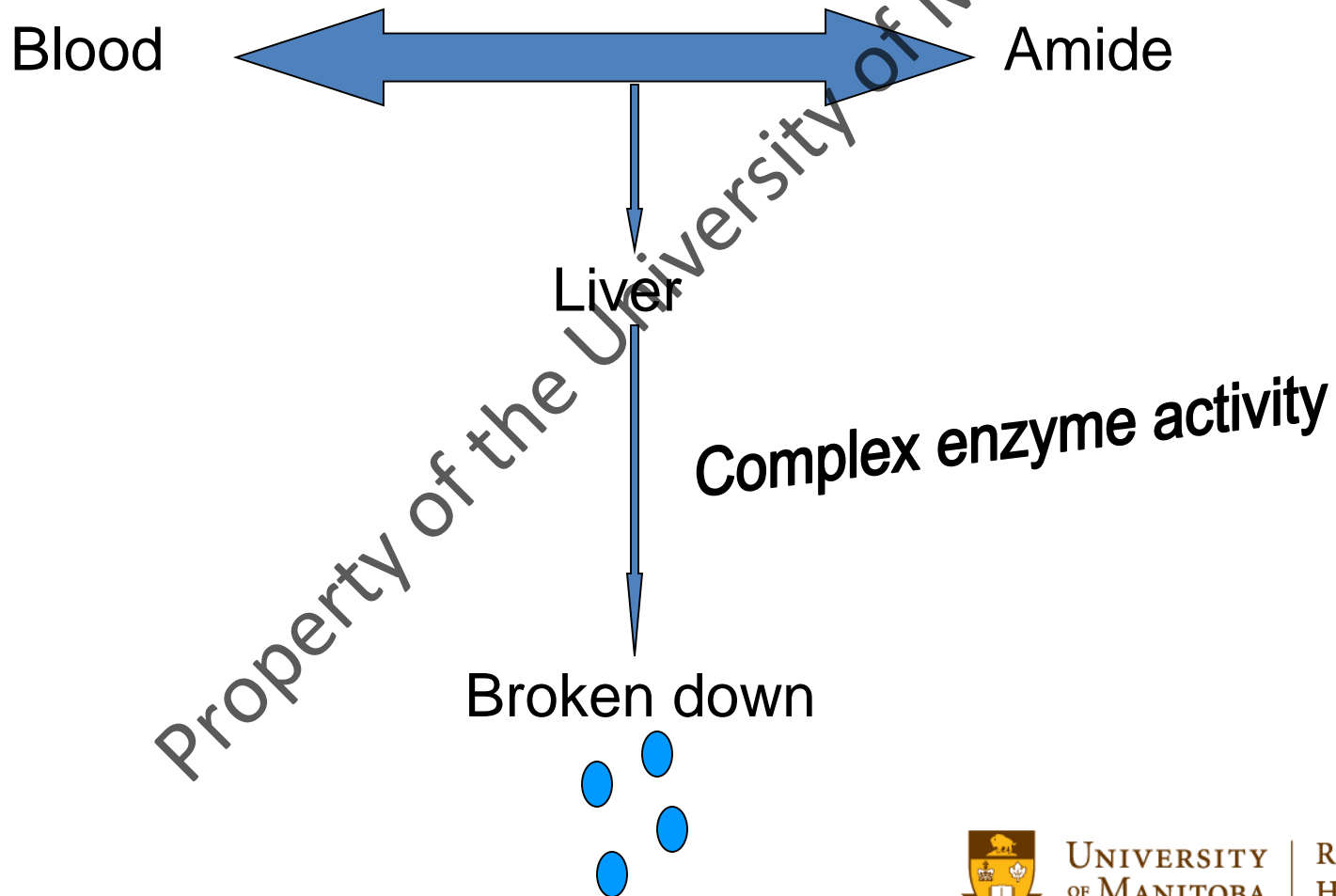
Atypical Plasma Cholinesterase

- Approximately 1 in 3,000 people has an atypical form of plasma pseudocholinesterase, which causes an inability to hydrolyze ester-type local anesthetics and other chemically related drugs e.g. succinylcholine (used during general anesthesia)

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Biotransformation of Amides



Amides

- Significant liver dysfunction or heart failure are **relative contraindications** to the use of amide type agents
- Biotransformation products of LA are capable of producing significant clinical problems (those with “**congenital**” methemoglobinemia)
- Those receiving **prilocaine LA** or **benzocaine topical** in large quantities are at higher risk for developing methemoglobinemia



Contraindications

- **Absolute Contraindications:** under no circumstance should this drug be administered because of its highly probable toxic or lethal reactions
- **Relative Contraindications:** drug may be administered to the patient after carefully weighing the risk of using the drug to its potential benefit and an acceptable alternative is not available.
 - The smallest effective dose should be used
 - There is a slightly increased possibility of an adverse reaction



Excretion

- The kidneys are the primary excretory organs for both types of local anesthetic agents and their metabolites.
- Patients with significant renal impairment may be unable to remove the local anesthetic agent from the blood
 - This would result in elevated levels in the blood and increase potential for toxicity
 - This should be considered a **relative contraindication**



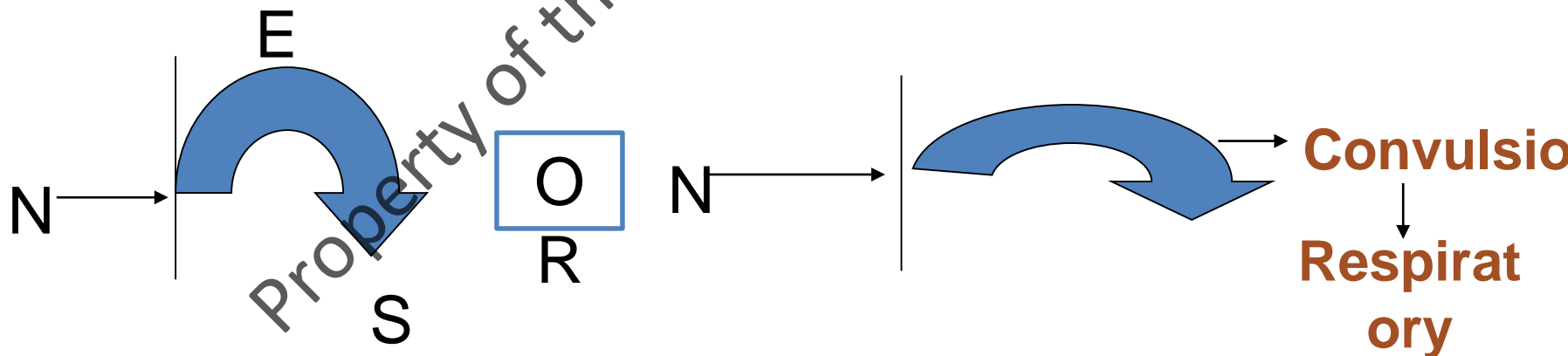
Central Nervous System (CNS)

- LA's readily cross the blood-brain barrier
- Action on the cells of the CNS = **depressed activity**
- Low non-toxic levels = no CNS actions
- **Toxic overdose levels = generalized tonic-clonic convulsive episode**
- Conversely at lower doses, local anesthetics have an anti-convulsing effect and have actually been used to decrease the duration of both grand mal and petit mal seizures



Central Nervous System (CNS)

- **Clinical onset of either excitatory signs or sedation following the administration of a local anesthetic should be a warning of a possible generalized convulsive episode which could ultimately result in respiratory arrest if severe enough



Convulsio
↓
Respirat
↓
ory
↓
Arrest



Cardiovascular System (CVS)

- LA's have a **direct** action on the heart (myocardium) and on the peripheral vasculature
- Action on the heart
 - Decreased electrical excitability
 - Decreased conduction rate
 - Decreased force of contraction

NOTE: Lidocaine and Tocainide have been used to treat arrhythmias



Action on Peripheral Vasculature

- All local anesthetics are vasodilating except cocaine which is a vasoconstrictor
- Vasodilating effect results in:
 - Increased blood flow
 - Increased rate of absorption
 - Decreased duration of anesthetic
 - Increased bleeding
 - Increased levels of anesthetic (that could lead to an overdose)
 - Primary effect of LA on blood pressure is **hypotension**

Use Vasoconstrictor!



Drug Interactions

- CNS Depressants (e.g. Narcotics, anti-anxiety agents, phenothiazines and barbiturates) if used with local anesthetics potentiate the cardiorespiratory action of the LA.
- Conjoint use of LA's and drugs that share a common metabolic pathway may produce adverse reactions

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Drug Interactions

- Drugs that induce production of hepatic microsomal enzymes (eg. Barbiturates) may alter the rate of metabolism of amide anesthetics (increase hepatic microsomal enzymes = > rate of metabolism of the LA)

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Malignant Hyperthermia

- Pharmacogenetic disorder
- Genetic variant in the individual alters the response to certain drugs
- Acute clinical manifestations: tachycardia, tachypnea, unstable B.P., cyanosis, respiratory and metabolic acidosis, fever (108 F or more!!!), muscle rigidity and death.
- Mortality ranges from 63% to 73%



Malignant Hyperthermia

- Many commonly employed general anesthetics can trigger malignant hyperthermia in certain individuals
- Amide type local anesthetics were thought to provoke malignant hyperthermia and were absolutely contraindicated for susceptible patients
- However, more current research contradicts this theory



Malignant Hyperthermia

- Amides are now considered as only **relative contraindications**
- If a patient discloses a history of malignant hyperthermia, the patient's physician should be consulted prior to treatment.
- Most patients with MH can be safely treated by close monitoring and use of “safe” drugs which now include the amide local anesthetics with or without epinephrine



Methemoglobinemia

- Methemoglobinemia is a condition in which a cyanosis-like state develops in the absence of cardiac or respiratory abnormalities
- When the condition is severe, the blood appears chocolate brown, & respiratory depression and syncope may occur.
- Death is rare, although a possibility.
- Methemoglobinemia is primarily caused through ***inborn errors of metabolism*** or they may be ***acquired*** through the administration of drugs or chemicals that increase formation of methemoglobin (***prilocaine and benzocaine***)



COMMONLY USED LOCAL ANESTHETIC AGENTS

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Classification of Local Anesthetics

Esters

Esters of Benzoic Acid

- Ethyl Amino Benzoate (Benzocaine)
- Tetracaine

Amides

- Articaine
- Bupivacaine
- Lidocaine
- Mepivacaine
- Prilocaine

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Concentration

- “Preparation of anesthetic agents according to their potency”
 - E.g., weakly potent agents are produced in higher concentrations in order to achieve desired effect
 - Lidocaine 2% VS Prilocaine 4%

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Potency

- “The amount of anesthetic required to produce the desired effect”

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Maximum Recommended Doses

PRODUCT	%	MRD	EXAMPLE FOR 60KG/120 LB HEALTHY CLIENT	ABSOLUTE MAXIMUM DOSE
Lidocaine	2 % 1:50,000 epi	7 mg/kg for both	420 mg or 6 cartridges	500 mg or 6 cartridges
	2 % 1:100,000 epi		420 mg or 11 cart's	500 mg or 11 cart's
Articaine	4 % 1:100,000 epi	7 mg/kg for both	420 mg or 6 cart's	500 mg or 6 cart's
	4 % 1:200,000 epi		420 mg or 6 cart's	500 mg or 6 cart's
Mepivacaine	3 % Plain	6.6 mg/kg for both	396 mg or 7 cart's	400 mg or 7.5 cart's
	2 % 1:20,000 levenordefrin		396 mg or 11 cart's	400 mg or 11 cart's
Prilocaine	4 % Plain	8.00 mg/kg for both	480 mg or 6.5 cart's	600 mg or 8 cart's
	4% 1:200,000		480 mg or 6.5 cart's	600 mg or 8 cart's
Bupivacaine	.5 % 1:200,000 epi	2.0 mg/kg	90 mg is ABSOLUTE MAXIMUM DOSE	90 mg is ABSOLUTE MAXIMUM DOSE

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Local Anesthetic Calculations

- Formula

- 1.8 ml per cartridge X
Number of Cartridges X
Concentration of solution =
mg administered

- Example: 2 cartridges
of lidocaine HCL 2%

- $1.8 \times 2 \times 20 = 72 \text{ mg}$
- $1.8 \times 1 \times 20 = 36$
- MRD for Lidocaine: 420 mg
- $420/36 = 11$

- Concentrations

- 1% = 10 ml
- 2% = 20 ml
- 3% = 30 ml
- 4% = 40 ml



Properties of Topical Anesthetics

- Most commonly used topical anesthetics are lidocaine and benzocaine
- Their concentrations are considerably higher than those used for injection
- The higher concentrations facilitate diffusion of the drug through the mucous membranes



Properties of Topical Anesthetics

- Higher concentrations also lead to greater potential for toxicity
- Topical anesthetics do NOT contain vasoconstrictors & because agents are vasodilators, vascular absorption is rapid
- Many of the local anesthetics are ineffective as topicals (i.e., prilocaine, mepivacaine)



Benzocaine

- Mode of Action
 - Direct absorption by free nerve endings
- Site of Action
 - Mucous Membranes
- Products Available
 - Cetacaine (liquid & gel) 14%
 - Gingicaine (liquid & gel) 20%
 - Healthco Topical (gel only) 20%
 - Hurricaine (liquid, gel & spray) 20%
 - Topex (metered spray, liquid, gel) 20%
 - Topicala (gel, liquid, ointment) 18%



Lidocaine

- Lidocaine Base
 - Poorly soluble in water
 - Used as a 5% concentration
 - Indicated for use on ulcerated, abraded or lacerated tissues

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- Lidocaine Hydrochloride
 - Water soluble
 - Used as a 2% concentration
 - Penetrates tissues better than a base
 - Potentially greater toxicity than base form
 - Decreased potential for allergic reactions
 - Maximum recommended dose is 200 mg



Lidocaine

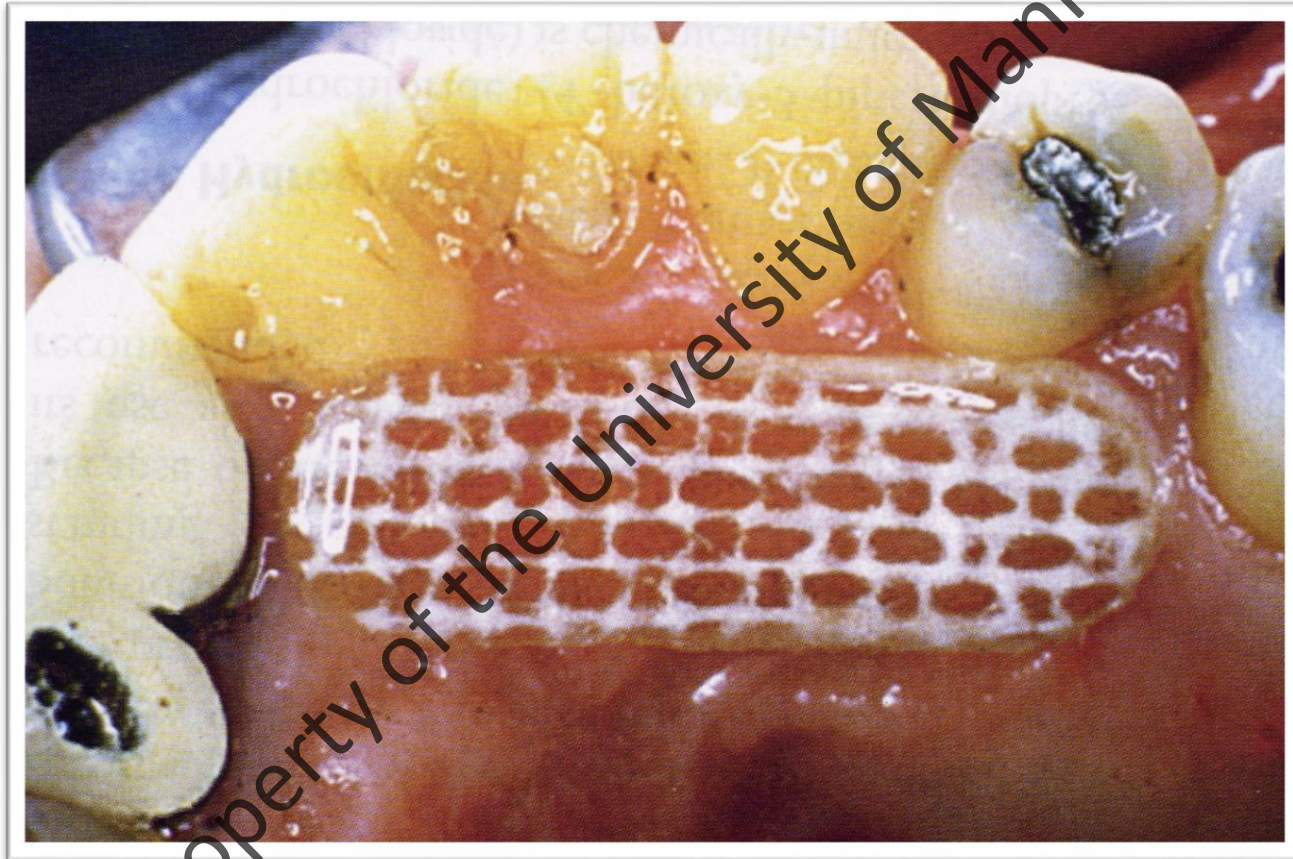
- Products Available

- Lidocaine (gel, liquid, ointment) 5%
- Xylocaine (liquid, ointment) 5%
- Xylocaine spray 10%
- Octocaine ointment 5%
- Dentipatch 5%

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Dentipatch



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Tetracaine

- Properties

- Ester
- 5-8 times more potent than cocaine
- Duration of action is 45 min. (metabolizes slowly)
- 2% concentration for topicals
- Rapidly absorbed by mucous membranes
- **EXTREME CAUTION:** Great potential for systemic toxicity

- Products Available

- Supracaine (0.7 mg/metered spray)
- Cetacaine (combination of: tetracaine 2%; benzocaine 14%; Butamben 2%)



Other Topical Anesthetics (*continued*)

- EMLA (Eutectic Mixture of Local Anesthetics)
 - Composed of Lidocaine 2.5% and Prilocaine 2.5% An emulsion of 1:1 of each drug
 - Designed for intact skin and primarily used in pediatrics
 - Must be applied one hour before procedure
 - Reaches maximum at 2-3 hrs and lasts 1-2 hrs
 - Primarily used in medicine, however studies have been done to support its use in dentistry, particularly in pediatric dentistry
 - Now perfected for use in dentistry as a periodontal gel and recommended for use in S&RP (Oraqix Dentsply)



Other Topical Anesthetics (*continued*)



Vasoconstrictors

- Why added to local anesthetics??
 - Constrict blood vessels and decrease blood flow to site of injection
 - Slow absorption of LA to blood stream
 - Lower blood levels decrease the risk of overdose reactions
 - Higher concentrations of LA remain around nerves for longer periods
 - Minimize bleeding at site of administration, therefore useful in procedures where bleeding is anticipated



Vasoconstrictors

IMPORTANT

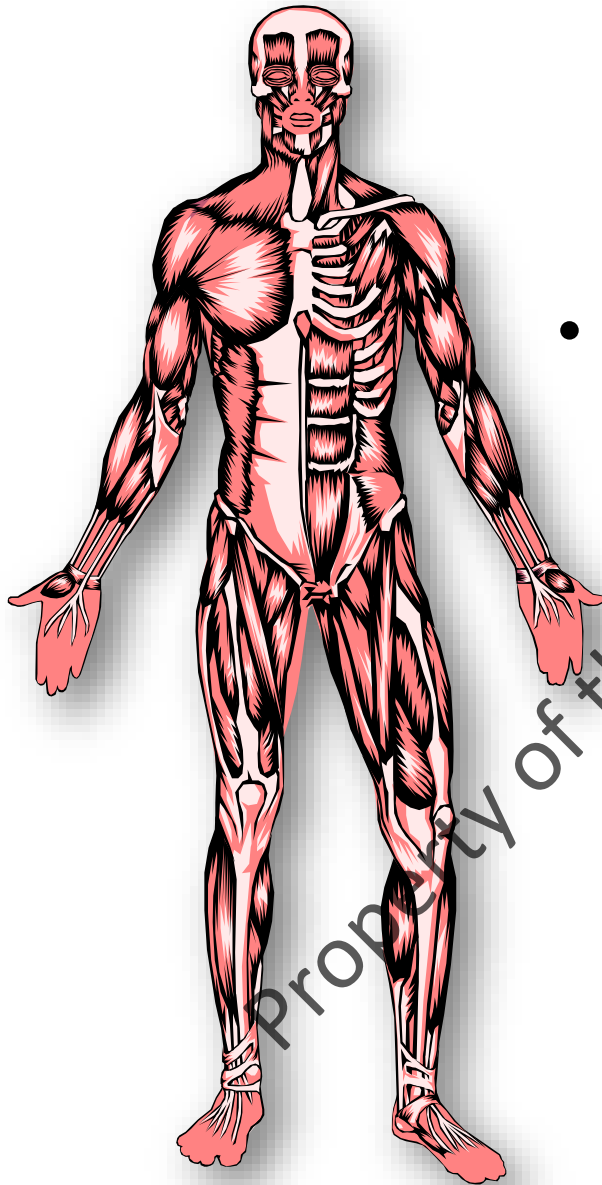
All anesthetics should contain epinephrine unless there is a very good reason not to!!

- E.g., procedures are of very short duration
- E.g., patient is medically compromised

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Concentration of Vasoconstrictors



- Commonly referred to as a ratio
 - i.e., 1:1000 means 1 gram or 1,000 mg of a drug in 1000 ml of solution



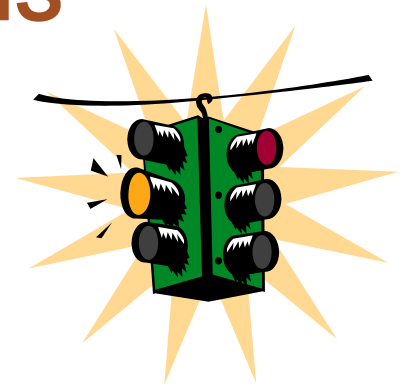
Vasoconstrictor Calculations

- Formula
 - 1.8 ml per cartridge X
 - Number of cartridges X
 - Concentration of Agent =
 - mg administered
- Epinephrine
 - 1:50,000 = 0.02 mg
 - 1:100,000 = 0.01 mg
 - 1:200,000 = 0.005 mg
- Levonordefin
 - 1:20,000 = 0.05 mg
- Example
 - 2 cartridges containing epinephrine 1:100,000
 - $1.8 \times 2 \times 0.01 = 0.036\text{mg}$



Contraindications for the use of Vasoconstrictors

- Very few contraindications to the concentrations found in dental anesthetics
- There are 4 types of patients for whom the administration of vasoconstrictors must be carefully weighed
- **ABSOLUTE CONTRAINDICATIONS**
 - Patients with high blood pressure
 - Patients with cardiovascular disease
 - Hyperthyroid patients
 - Sulfite sensitivity



High Blood Pressure

- Patients with the following **resting blood pressures** should not receive dental treatment
 - SBP >200 mmHg **OR**
 - DBP >115 mmHg



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Cardiovascular Disease

- Patients with severe cardiovascular disease may be at too great a risk for routine dental therapy
 - Myocardial infarction within 6 mos.
 - Patients who experience angina daily
 - Patients with untreated or uncontrolled congestive heart failure
 - Recent coronary artery bypass surgery
 - Severe cardiac arrhythmias, despite drug therapy

**Epinephrine may be used in patients with mild to moderate cardiovascular disease*



Hyperthyroid Patients

- Epinephrine is **ABSOLUTELY CONTRAINDICATED** for hyperthyroid patients with clinical evidence of the hyperthyroid state
- Signs & symptoms:
 - Exophthalmos
 - Hyperhidrosis
 - Tremor
 - Irritability, nervousness
 - Elevated body temperature
 - Inability to tolerate heat
 - Increased heart rate
 - Increased B.P.



Sulfite Sensitivity

- Shelf-life of an anesthetic with epinephrine is 18 mos.
- If no epinephrine, it is 3 years!
- Sodium Bisulfite is added to prevent oxidation of the anesthetic



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Sulfite Sensitivity

- Several patients have sulfite allergies
- High risk groups are those allergic to dried fruits; red wines and those who are “allergic asthmatics”
- Allergic Asthmatics have a high incidence of sulfite allergies, therefore **DO NOT USE EPINEPHRINE!!**



Relative Contraindications Antidepressant Medications

Tricyclic Antidepressants

- Amitriptyline (Elavil)
- Nortriptyline (Aventyl, Pamelor)
- Imipramine (Tofranil)
- Doxepin (Sinequan)
- Amoxapine (Asendin)
- Desipramine (Norpramin)
- Protrityline (Vivactil)
- Clomipramine (Anafranil)

Monoamine Oxidase Inhibitors

- Isocarboxazid (Marplan)
- Phenelzine (Nardil)
- Tranylcypromine (Parnate)
- Trimipramine (Surmontil)



Relative Contraindications Beta-Blockers (non-selective* only)

*Non-selective

- Propranolol (Inderal)
- Nadolol (Corgard)
- Timolol (Blockadren Timolate, Timoptic, Timoptol)
- Pindolol ((Visken)
- Alprenolol (Aptine)
- Labetalol ((Trandate, Nomodyne)
- Oxprenolol (Trasicor)
- Sotalol (Sotacort)
- Carteolol (Cartrol)
- Penbutolol (Levatol)

Selective

- Metoprolol (Lopressor)
- Atenolol (Tenormin)
- Acebutolol (Sectral)
- Betaxolol (Kerlone)
- Esmolol (Brevibloc)
- Bisoprolol (Zebeta)



Other Medications

Relative Contraindications

- Phenothiazine
- Antiarrhythmic drugs
- Levodopa
- Cocaine—there is a greater chance of developing cardiac arrhythmias & arrest
- Any Drug Abusers!!
- ASA IV, V, & VI

**If you must use a vasoconstrictor for any of these, then use the lowest dose!!*



Signs of Drug Abuse

- Opioids (Heroin, Morphine)
 - Pinpoint pupils (flip light on & off, if no change, think opioids)
- Amphetamines (Crystal Meth, etc.)
 - If light on eyes, 1st constrict then dilate again, sweating, tremors, hyper
- Cocaine
 - Same as amphetamines

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Signs of Drug Abuse

- Hallucinogens (LSD, Angel Dust)
 - Pupils dilated, Increased blood pressure & heart rate, face flushed
- Marijuana
 - Red eyes, hunger

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ASA Categories

(American Society of Anesthesiologists)

These patients
are dentally
treatable.

ASA I	Normal healthy patient <ul style="list-style-type: none">• Everything WNL• BP 140/90 or less
ASA II	Mild systemic disease <ul style="list-style-type: none">• BP 160/95• Well-controlled asthmatic (never hospitalized)• Well-controlled epileptics• Type 2 Diabetes (NIDDM) (80% of Diabetics)
ASA III	More Severe Systemic Disease <ul style="list-style-type: none">• Activity limited but not incapacitated• BP 160-199/95-115 (treatable but must modify care)• Type I Diabetes (IDDM) (controlled)• Heart attack or stroke >6 months ago• Angina (stable) 1-2 episodes/wk but consistent & predictable; pain resolves with 2 tabs nitro.

ASA Categories

(American Society of Anesthesiologists)

These patients
are **NOT**
dentally
treatable.

ASA IV	Incapacitating disease which is a constant threat to life, even at rest. Same as ASA III but worse. <ul style="list-style-type: none">• Recent heart attack or stroke within last 6 months• Recent coronary bypass surgery• BP 200/115• End stage HIV• End stage renal disease; liver disease; carcinoma
ASA V	Very ill patient, not expected to live >24 hours (end-stage disease).
ASA VI	Clinically dead (being kept alive until organs are transplanted).



Important Note

- Vasoconstrictors are ***not contraindicated*** for a patient whose medical condition has been diagnosed and is ***under control*** through medical or surgical means
- And if Vasoconstrictor is ***administered***:
 - Slowly
 - In Small Quantities
 - And After Negative Aspiration



Three Major Methods by which Drugs Produce Adverse Reactions

- Toxicity caused by Direct Extension of Pharmacological effects
 - Side effects
 - **Overdose**
 - Local toxic effects
- Toxicity caused by Alteration in the Recipient
 - Presence of disease (hepatic dysfunction, heart disease)
 - Emotional disturbances
 - Genetic aberrations (atypical plasma cholinesterase, malignant hyperthermia)
 - **Idiosyncrasy**



Three Major Methods by which Drugs Produce Adverse Reactions

- Allergy to the Drug
 - ***Major difference between toxicity and allergy***
 - Toxicity is **DOSE-DEPENDENT**
 - Allergy is **NOT DOSE-DEPENDENT**

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Toxicity

- “The amount of anesthetic agent required to produce a toxic overdose”

**The greater the potency, the greater the risk of toxic overdose!*

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Toxicity

- Toxicity will occur if:
 - Too much is given
 - Absorbed too quickly
 - Metabolized too slowly
 - Unable to be excreted

**Toxic overdose may be due to either faulty technique or medical compromise*

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Local Anesthetic Overdose

- The most common of all true adverse reactions (up to 99%)
- The drug must gain access to the circulatory system in quantities sufficient to produce adverse effects on tissues of the body
- The reaction continues only as long as the level of the agent remains above the threshold for overdose

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Idiosyncrasy

- An unexplained adverse drug reaction that is neither an overdose or an allergy
- Cannot predict
- Thought to be genetic
- Treat symptomatically
 - i.e., A,B,C.

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Causes of Overdose

- Total dose too large
- Absorption unusually rapid
- Intravascular administration of LA
- Biotransformation unusually slow (impaired liver function)
- Drug slowly eliminated through kidneys (impaired kidney function)

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Prevention of Overdose

- Thorough assessment of medical history
- Use of an aspirating syringe
- Use of a needle no smaller than a 25 gauge (may be impossible to aspirate with a 30 gauge)
- Aspiration on at least two planes before injecting (45-degree rotation of needle to change direction of bevel)
- **Slow injection of the solution!**



Prevention of Overdose

- **Slow injection is the most important factor in the prevention of adverse drug reactions!**
- **Under no circumstances should injection of 1.8 ml be attempted in <1 minute!**

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Clinical Manifestations of LA Overdose

- Talkativeness
- Apprehension
- Excitability
- Slurred speech
- Generalized stutter, leading to muscle twitching & tremor of facial muscles and extremities
- Elevated blood pressure
- Elevated heart rate
- Elevated respiratory rate



Progressive Symptoms with Increased Blood Levels

- Generalized feeling of lightheadedness and dizziness
- Visual disturbances (inability to focus)
- Auditory disturbances
- Drowsiness and disorientation
- Loss of consciousness

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Moderate to High Overdose Levels

- Generalized tonic-clonic seizures followed by:
 - Generalized CNS depression
 - Depressed BP, heart rate and respiratory rate
- **NOTE**: Possibility that excitatory phase may be brief or missing! First manifestation of overdose could be drowsiness, progressive unconsciousness and respiratory arrest. This is more common with Lidocaine.



Clinical Manifestations of Epinephrine Overdose

- Fear, anxiety
- Tenseness, restlessness
- Throbbing headache
- Tremor
- Perspiration
- Weakness
- Dizziness
- Respiratory difficulty



Allergy

- Allergic Responses to local anesthetics include:
 - Allergic dermatitis
 - Asthmatic attack
 - Systemic anaphylaxis
- Most frequent is dermatological reaction
- Life-threatening responses can occur, although this is rare
- Most frequent hypersensitivity is to ester-type anesthetics
- Allergic reactions documented to the contents of the cartridge



Clinical Manifestations of Allergic Reaction

- Skin reactions
- Cramps, nausea, vomiting
- Respiratory distress
- Cardiovascular distress

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Summary of Significance of Medical History

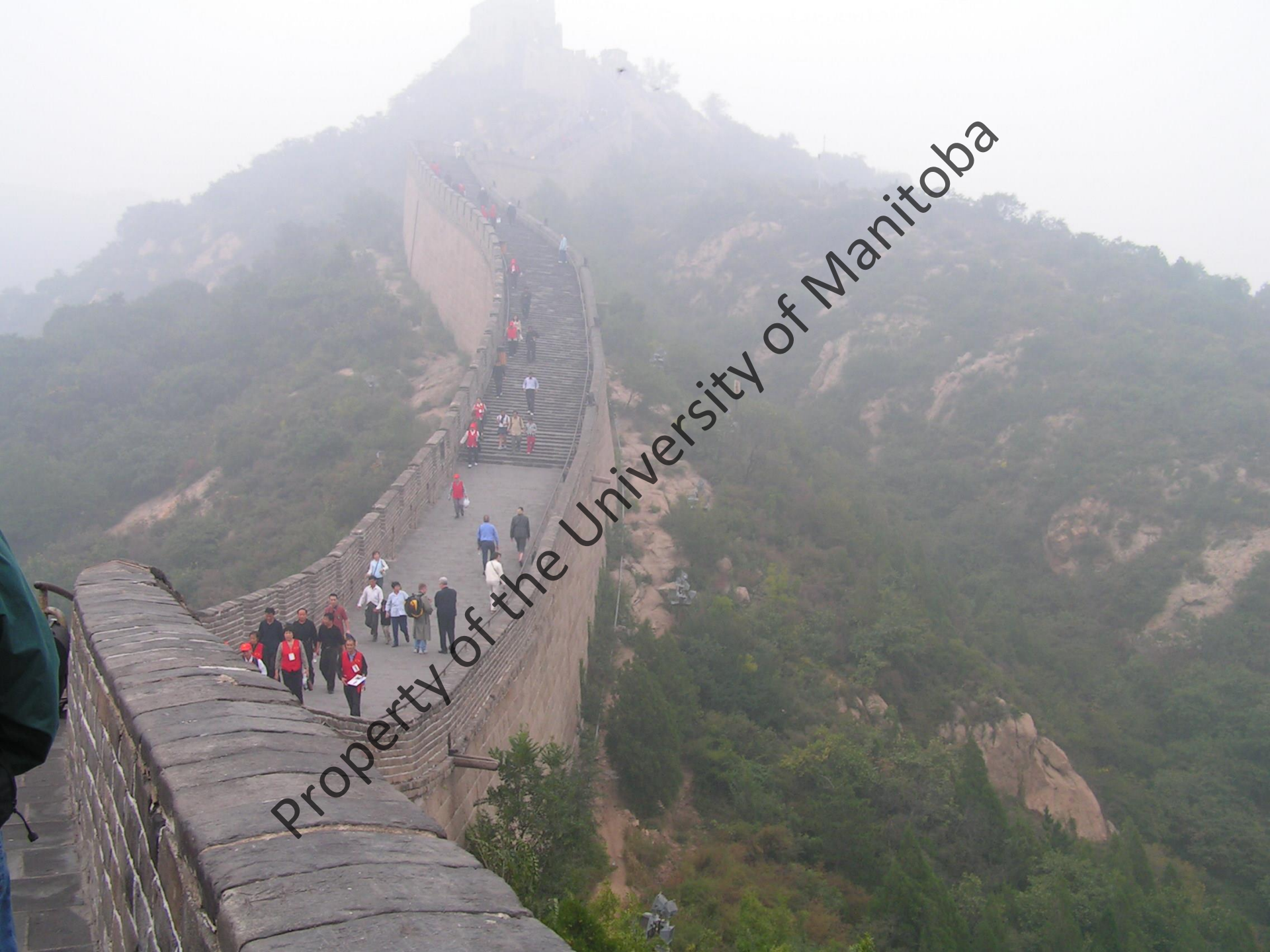
- Liver Dysfunction (*relative for amide*)
- Kidney Dysfunction (*anesthetic with caution*)
- Malignant Hyperthermia (*relative for amides*)
- Plasma Pseudocholinesterase deficiency (*no ester*)
- Uncontrolled hyperthyroidism (*no vasoconstrictor*)
- Cardiac arrhythmias (*no vasoconstrictor*)
- Asthmatics and documented allergies (*no vasoconstrictor*)
- Sulfite sensitivity/allergy (*no articaïne or vasoconstrictor*)



- All Photos and Diagrams were copied from the HYG 2380 2014 Pain Management Study Guide by Salme Lavigne and Malamed's 6th Edition of the Handbook of Local Anesthesia, 2013

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