POLTAVA STATE MEDICAL UNIVERSITY
Department of Anesthesiology and Intensive Care

Intensive care of acute poisoning

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Lecture plan

1. Basic principles of IT acute poisoning.
2. Basic principles of forced diuresis.
3. Extracorporeal detoxification methods, indications and contraindications, technical means, technique of execution.
4. Principles of antidote therapy.
5. Pathogenesis, clinic and IT in methyl alcohol poisoning.
6. Pathogenesis, clinic and IT poisoning with opiods and barbiturates.

Lecture plan

7. Pathogenesis, clinic and IT in poisoning with organophosphins
8. Pathogenesis, clinic and IT in acid and alkali poisoning.
9. Pathogenesis, clinic and IT in carbon monoxide poisoning.
10. Pathogenesis, clinic and IT for poisonous mushroom poisoning.
11. Features of first aid for insect and animal bites.

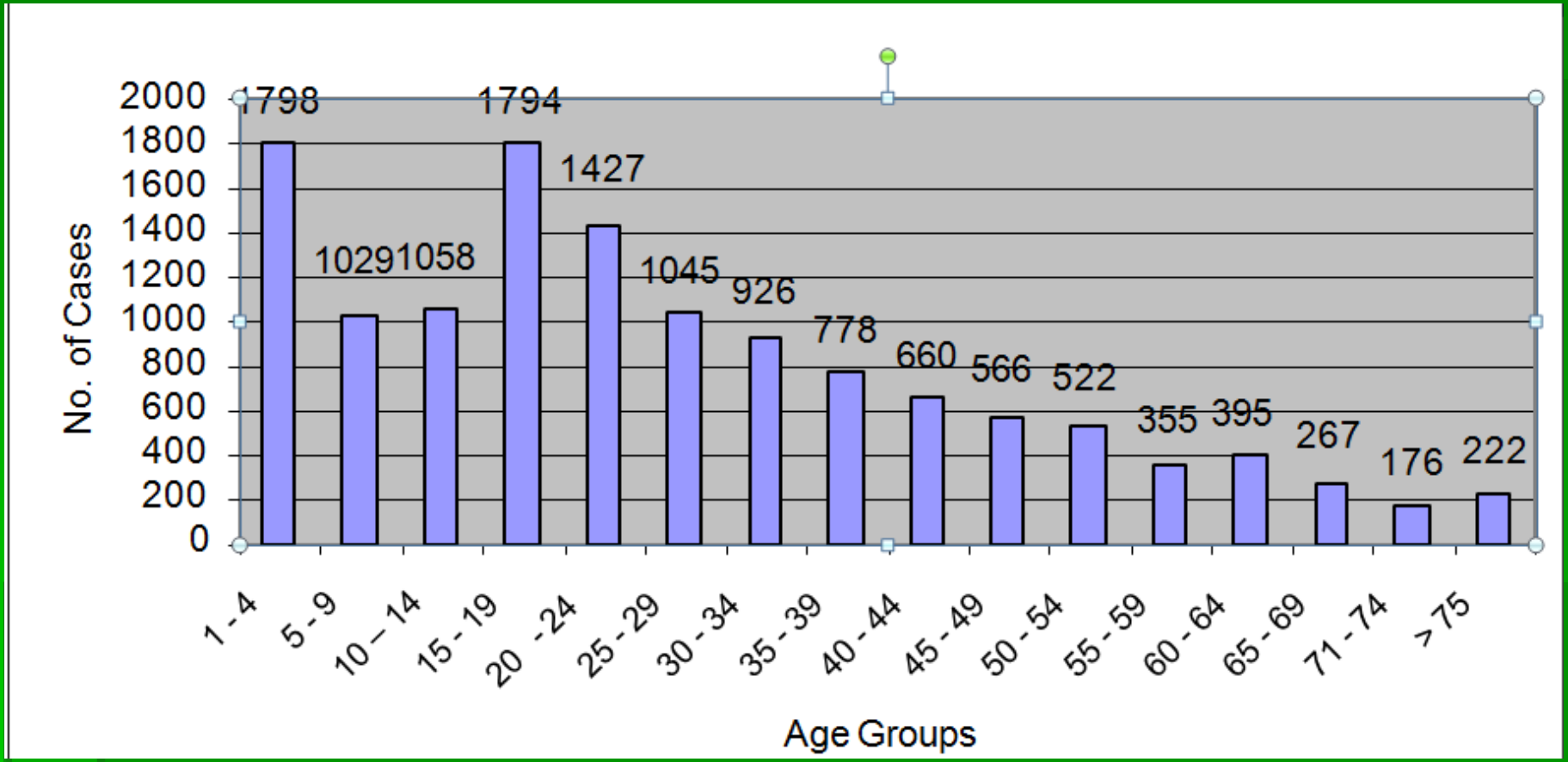
Definition

Any poisonous effect produced from a single or short exposure (24 to 96 hours) resulting in severe biological harm or death

All things are poison and nothing is without poison. It is the dose only that makes a thing not a poison.”

Paracelsus (1493-1541)

Epidemiology



Phases Of Poisoning

- Preclinical phase
- Toxic phase
- Resolution phase

Preclinical phase

- Period follows exposure before s/sx
- Aim: to reduce or prevent toxicity
- Decontamination is a priority

Toxic phase

- Period from onset to peak of manifestation of toxicity clinical or laboratory
- Aim: to shorten or lessen the severity of toxicity
- Priority: stabilize airways, breathing and circulation and consider antidote

Resolution phase

- Period from peak toxicity to recovery
- Major goal: shorten the duration of toxicity & supportive care

Suspect intoxication

History of drug overdose or substance abuse
Suicidal ideation or prior suicide attempt
History of other psychiatric illness
Agitation and hallucinations
Stupor or coma
Rotary nystagmus
Delirium or confusion
Seizures
Muscle rigidity
Dystonia
Cardiopulmonary arrest
Unexplained cardiac arrhythmia
Hyper/hypotension
Ventilatory failure

Aspiration
Bronchospasm
Liver failure
Renal failure
Hyper/hypothermia
Rhabdomyolysis
Osmolal gap
Anion gap acidosis
Hyper/hypoglycemia
Hyper/hyponatremia
Hyper/hypokalemia
Polypharmacy

Odor	Poison
Sweet/fruity	Ketone, alcohol
Almond	Cyanide
Gasoline	Hydrocarbon
Garlic	Organophospate
Wintergreen	Methylsalicylate
Pear	Chloral hydrate

Toxicology History

Goal is Identification of Etiologic Agent(s)

- Use all Available Resources
  - Pill bottles
  - Pre-hospital personnel
  - Family and Friends
  - Medical Records
  - Past medication and medical history
- Assess for Suicidal Behavior
  - Must assume suicidal until proven otherwise
  - Low threshold for Psychiatric consultation

Evaluation of Toxicity

Evaluate the **SATSC**

Substance

Amount

Time since ingestion

Symptoms

Co-morbid

Therapeutic approach

A – Airway (mental status, suicidal trauma)

B – Breathing (resp depression, pulm oedema, ARDS)

C – Circulation (dysrhythmias, CV depression)

D – Dysfunction CNS (hypoglycemia, alcohol, opiate & benzodiazepine overdose, seizure control)

E – Exposure (hyperthermia)

Reduce absorption

- Emesis – No role
- Activated charcoal within 1 h
- Gastric lavage
- Whole bowel irrigation

Enterosobtion

Mnemonic: CHARCOAL

Caustics & corrosive

Heavy metals

Alcohol & glycols

Rapidly absorbed substances

Cyanide

Other insoluble drugs

Aliphatic hydrocarbobs

Laxatives

Increase elimination

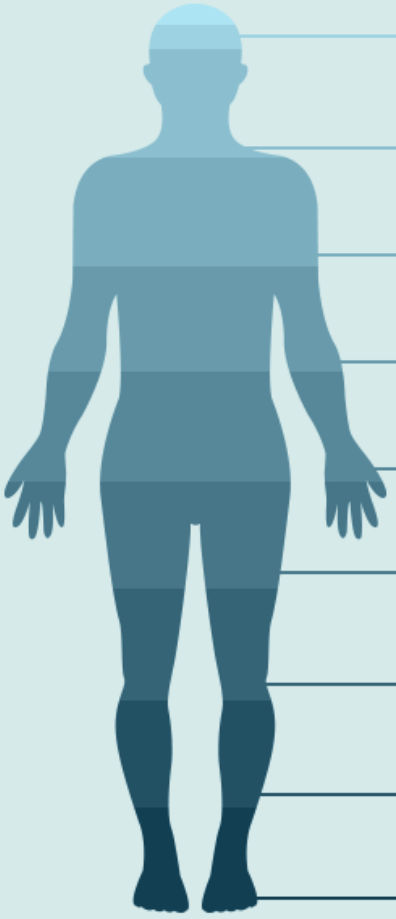
- Urinary alkalinisation
- Multi-dose Activated Charcoal
- Haemodialysis
- Haemoperfusion
- Plasma exchange
- Forced alkaline diuresis

Alcohol

Vomiting
Seizures
Hypothermia
An uneven breathing pattern
Loss of motor coordination
Pale skin
Mental confusion
Stupor which translates to being conscious but unresponsive to stimuli
Loss of consciousness

Alcohol

POISONING | OCCURS DUE TO THE LEVELS OF ALCOHOL IN THE BLOOD



- **0.02-0.039%** Normal behavior, slight increase of euphory
- **0.04-0.059%** Lower inhibition sets in, euphoria increases
- **0.06-0.99%** Loss of coordination, slurred speech, impaired memory
- **0.10-0.129%** Impaired balance, peripheral vision & hearing
- **0.13-0.159%** Blurred vision, onset of dysphoria
- **0.160-0.199%** Major dysphoria and dizziness sets in
- **0.20-0.249%** Can barely walk, vomiting, possibility of blacking out
- **0.250-0.399%** **ALCOHOL POISONING!**
- **0.40%+** Onset of coma, death by respiratory arrest is possible

Alcohol

Life-Threatening Effects

Hypoglycemia
Choking
Dehydration
Cardiac Arrest.
Kindling
Stoppage of Breathing
Brain Damage

Alcohol

Treatment

Don’t!

Don’t give a cold shower. It might cause hypothermia.
Don’t offer hot coffee. Caffeine can increase dehydration.
Don’t feed the person. Food might make him or her choke.
Don’t help or encourage the person to “walk it off.” He or she could fall.
Don’t give medication. It might worsen the person’s condition.
Don’t induce vomiting. The person might gag.

Alcohol

Treatment

- Airway protection
- Gastric lavage
- Oxygen therapy
- IV fluids, tiamin and glucose
- Hemodialysis

Opioids

Substances that act
on **opioid** receptors to produce
morphine-like effects

Opioids

- Opium
- Morphine
- Diacetylmorphine (heroin)
- Codeine
- Fentanyl
- Methadone

Opioid toxidrome

- Bradycardia
- CNS depression
- Reduced gastro-intestinal motility
- Hypotension
- Miosis (pin point pupils)
- Respiratory depression: bradypnoea, apnoea
- Hypothermia
- Needlestick trackmarks

Opioid toxidrome

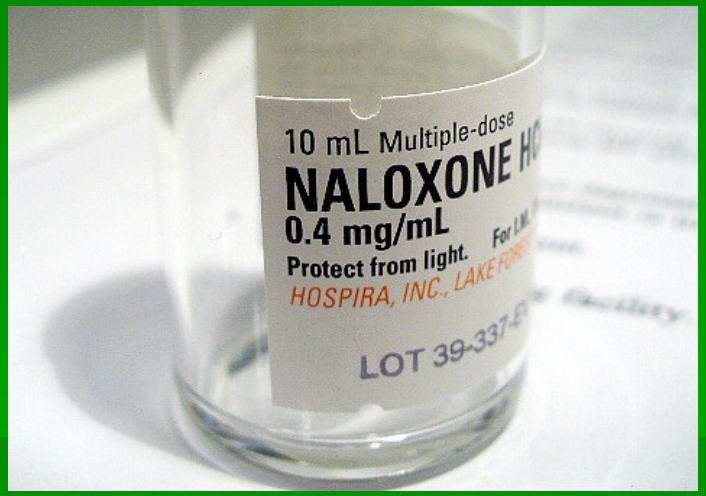
- Ventricular arrhythmias
- Seizures
- Nausea
- Vomiting
- Flushing
- Pruritus
- Constipation

Diagnostic tests

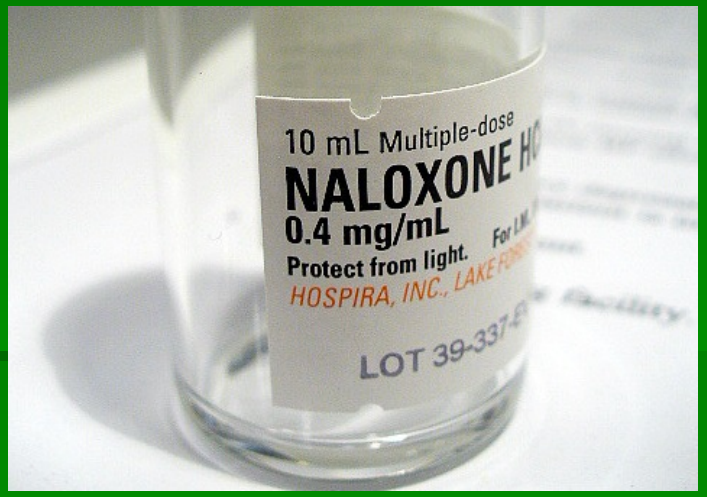
- Drug screens (urine up to 36-48 hours);
- Basic blood and urine tests (Complete blood cell count, Metabolic panel, Creatine kinase level, Arterial blood gas determinations, Urine tests);
- Instrumental studies (chest, abdomen radiographs for swallowed drug packages);
- Electrocardiography (ST abnormalities (19%), QTc prolongation (13%), tall R- and/or S-waves (11%) and missing R progression (10%);
- Echocardiography.

Opioids

- Airway protection
- Naloxone 0.4 - 2 mg IV
- Gastric lavage



Naloxone



Adults

Initial

No respiratory depression: 0.1 to 0.4 mg IV

Respiratory depression: 1 to 2 mg IV

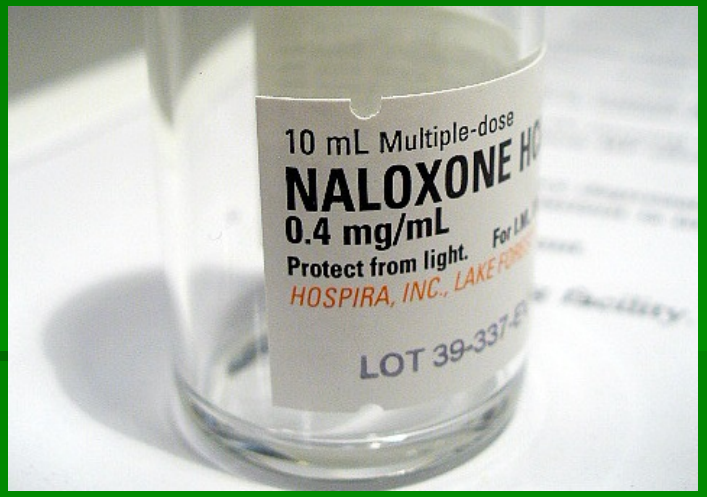
Next, if no response or incomplete response

Give 2 mg IV or IM every 3-5 minutes to a total of 10-20 mg

Infusion

2 mg in 500 ml NS (0.004 mg/ml) titrating to response

Naloxone



- Children
  - Initial
    - No respiratory depression: 0.01 mg/kg IV or IM
    - Respiratory depression: 0.1 mg/kg IV
  - Next, if no response or incomplete response give 0.1 mg/kg IV.

Sedative-hypnotic

- Barbiturates:
  - amital,
  - pentobarbital,
  - phenobarbital,
  - secobarbital

- Benzodiazepines:
  - diazepam (Valium),
  - alprazolam (Xanax),
  - lorazepam (Ativan),
  - temazepam (Restoril),
  - clonazepam (Klonopin)

Sedative-hypnotic toxidrome

- CNS depression-lethargy, obtundation;
- Normal to large, sluggishly reactive, pupils;
- Paradoxical excitement;
- Ataxia;
- Bradycardia;
- Hypotension;
- Hypothermia;
- Respiratory depression;
- Slurred speech

Sedative-hypnotic

Treatment

- Airway protection
- Oxygen therapy
- Gastric lavage
- Multi-dose activated charcoal (20-50 g q4h)
- Alkaline diuresis
- Flumazenil (benzodiazepine)
- Hemodialysis

Stimulants

DEFINITION

CNS stimulants are the psychoactive drugs that induce temporary improvement in either mental or physical function or both

Stimulants

- Cocaine
- Amphetamine
- Atomoxetine
- Methylphenidate

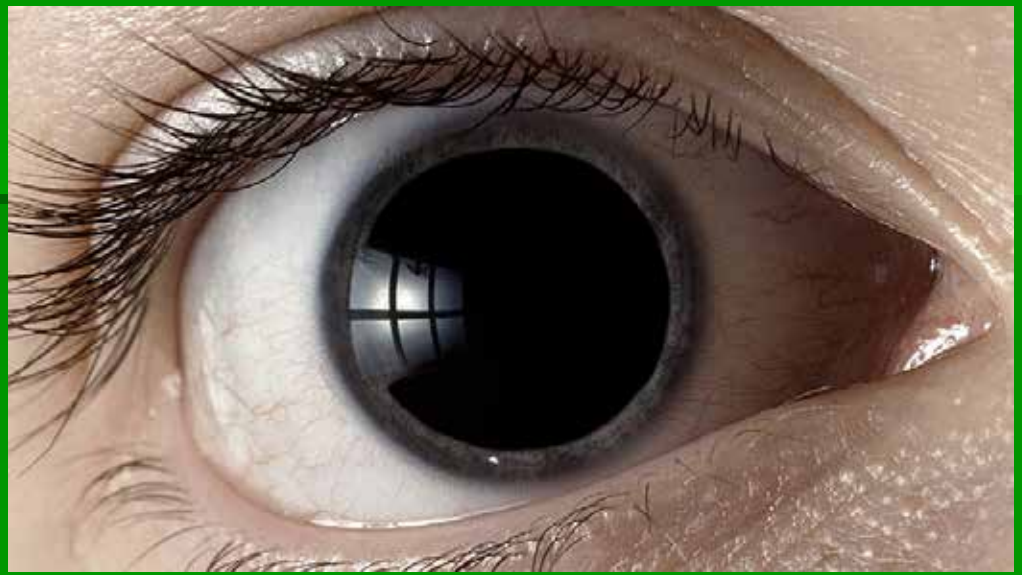
- Tachycardia
- Tachypnoea
- Hypertension
- Hyperthermia
- Sweating (diaphoresis)
- Dry mucosae
- Piloerection
- Mydriasis
-

Stimulants toxidrome

- Agitation
- Delirium
- Paranoid delusions
- Seizures
- Stroke
- Acute coronary syndrome
- Aortic dissection
- Cardiac arrhythmias
- Hyperactive bowel sounds
- Rhabdomyolysis

Stimulants

- Agitation/delusions/paranoia
- Fight/Flight response
- Tachycardia
- Hypertension
- Arrhythmias
- Dilated pupils
- Seizures
- Hyperpyrexia



Stimulants. Treatment

- Airway protection
- Benzodiazepines (Agitation)
- Propranolol (Tachycardia,Hypertension)
- Nitroglycerin (Coronary spasm, Hypertension)

Hallucinogens

Substances that produce changes in perception, thought, and feeling, ranging from distortions of what is sensed (illusions) to sensing objects where none exist (hallucinations)

Hallucinogens

- Lysergic acid diethylamide (LSD)
- Mescaline
- Psilocybin
- Ketamine
- Methoxetamine (MXE)
- Phencyclidine (PCP)
- Dextromethorphan (DXM)
- Tetrahydrocannabinol

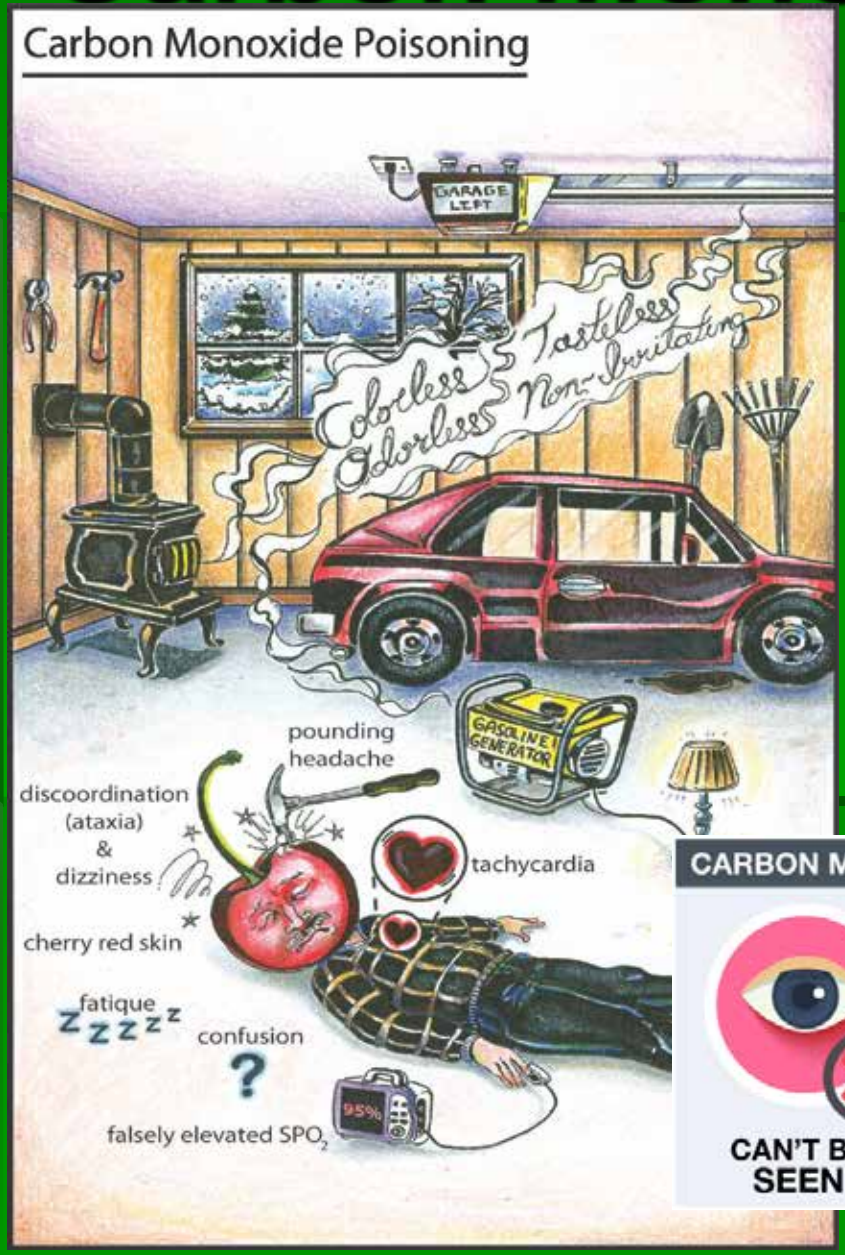
Signs of tricyclic anti-depressant overdose

- Dry skin and mouth
- Urinary retention
- Tachycardia
- Ataxia
- Jerky limb movements
- Divergent squint
- Altered level of consciousness

Paracetamol

Often asymptomatic
Check blood level at 4 hours
Given IV N-acetylcysteine
150mg/kg

Carbon monoxide



Carbon monoxide

Percent CO in Blood	Typical Symptoms
<10	None
10-20	Slight headache
21-30	Headache, slight increase in respirations, drowsiness
31-40	Headache, impaired judgment, shortness of breath, increasing drowsiness, blurring of vision
41-50	Pounding headache, confusion, marked shortness of breath, marked drowsiness, increasing blurred vision
>51	Unconsciousness, eventual death if victim is not removed from source of CO

Carbon monoxide

- Remove from continued exposure
- 100% oxygen
- Hyperbaric Oxygen Therapy

Organophosphate

Insecticides – Malathion, parathion, diazinon, fenthion, dichlorvos, chlorpyrifos, ethion
Nerve gases – Soman, sarin, tabun, VX
Ophthalmic agents – Echothiophate, isoflurophate
Antihelmintics – Trichlorfon
Herbicides – Tribufos (DEF), merphos
Industrial chemical (plasticizer) – Tricresyl phosphate

Organophosphate

Muscarinic signs

salivation,
lacrimation,
urination,
diaphoresis,
emesis & diarrhea
bronchospasm & bronchorrhea,
blurred vision,
bradycardia or tachycardia,
hypotension, confusion, and shock

Organophosphate

Nicotinic effects

muscle fasciculation
weakness
paralysis
ventilatory failure

Organophosphate Treatment

- **Decontamination**
- **Airway management**
- **Atropine**
- **Pralidoxime**

Organophosphate

Atropine

Start with a 1-2 mg IV bolus, repeat q3-5min for desire effects (drying of pulmonary secretions and adequate oxygenation). Tachycardia and mydriasis must not be used to limit or to stop subsequent doses of atropine.

Organophosphate

Pralidoxime

Reactivator the AChE.

Used as an antidote to reverse muscle paralysis resulting from OP AChE pesticide poisoning but is not effective once the OP compound has bound AChE irreversibly (aged). Start with 1-2 g (20-40 mg/kg) IV in 100 mL isotonic sodium chloride over 15-30 min; repeat in 1 h if muscle weakness is not relieved; then repeat q3-8h if signs of poisoning recur; other dosing regimens have been used, including continuous drip.

Bites by venomous snakes

Elapidae: cobras, kraits, mambas, coral snakes

Viperidae:

Crotalidae rattlesnake, cottonmouth, copperhead;

Viperinae: vipers, adders

Hydrophidae (sea snakes)

Bites by venomous snakes

Snake bites

138,000
people thought to die from snake bites every year

450,000 more suffer life-changing injuries

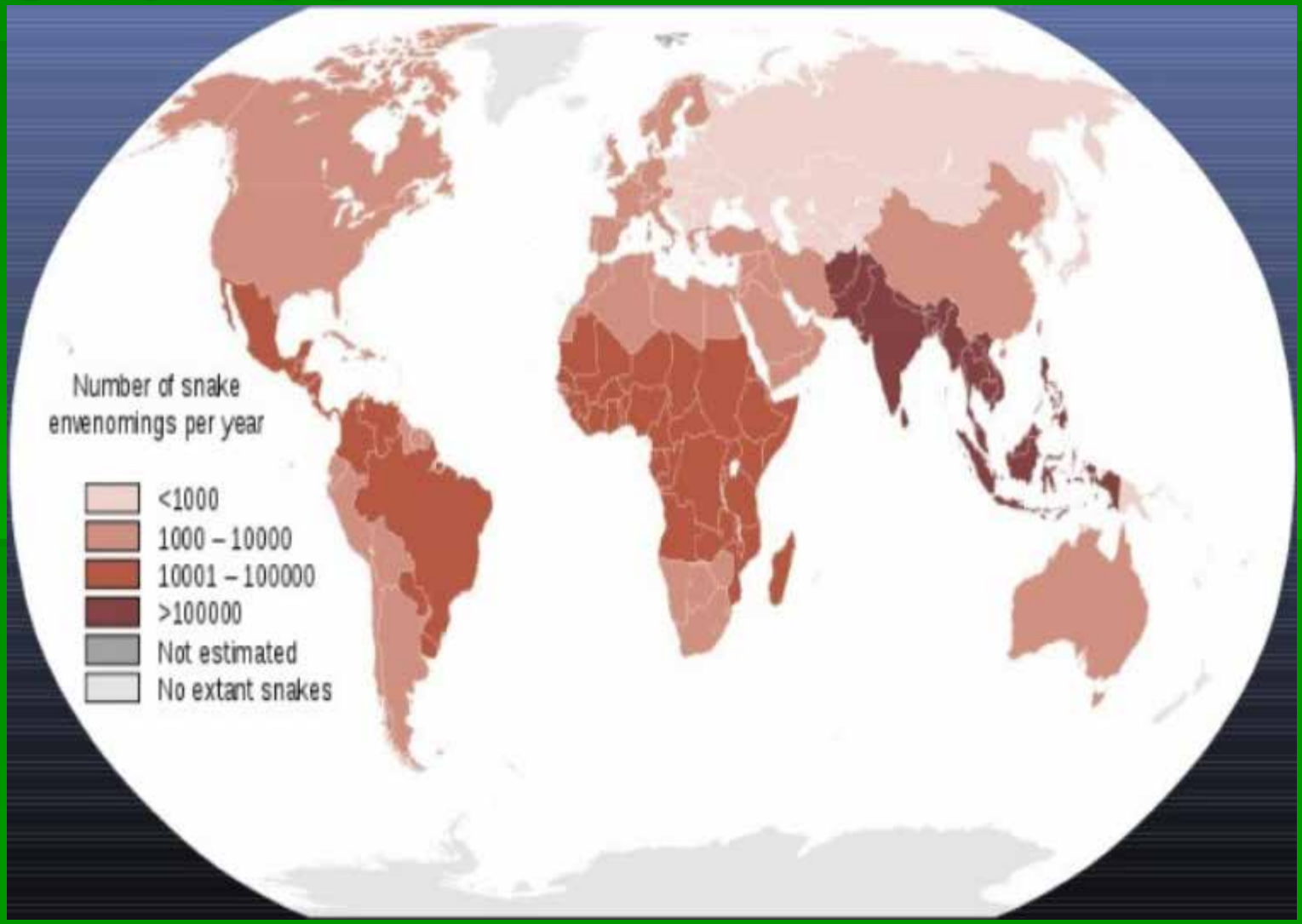
$300 charge for the most expensive vial of antivenom - six months salary for a Swazi farmer

$30 cost of a vial of some newer brands of antivenom, which can be weak or inappropriate

20-30 vials of cheaper antivenom often needed, up to 10 times as much as other brands

Source: WHO

Bites by venomous snakes

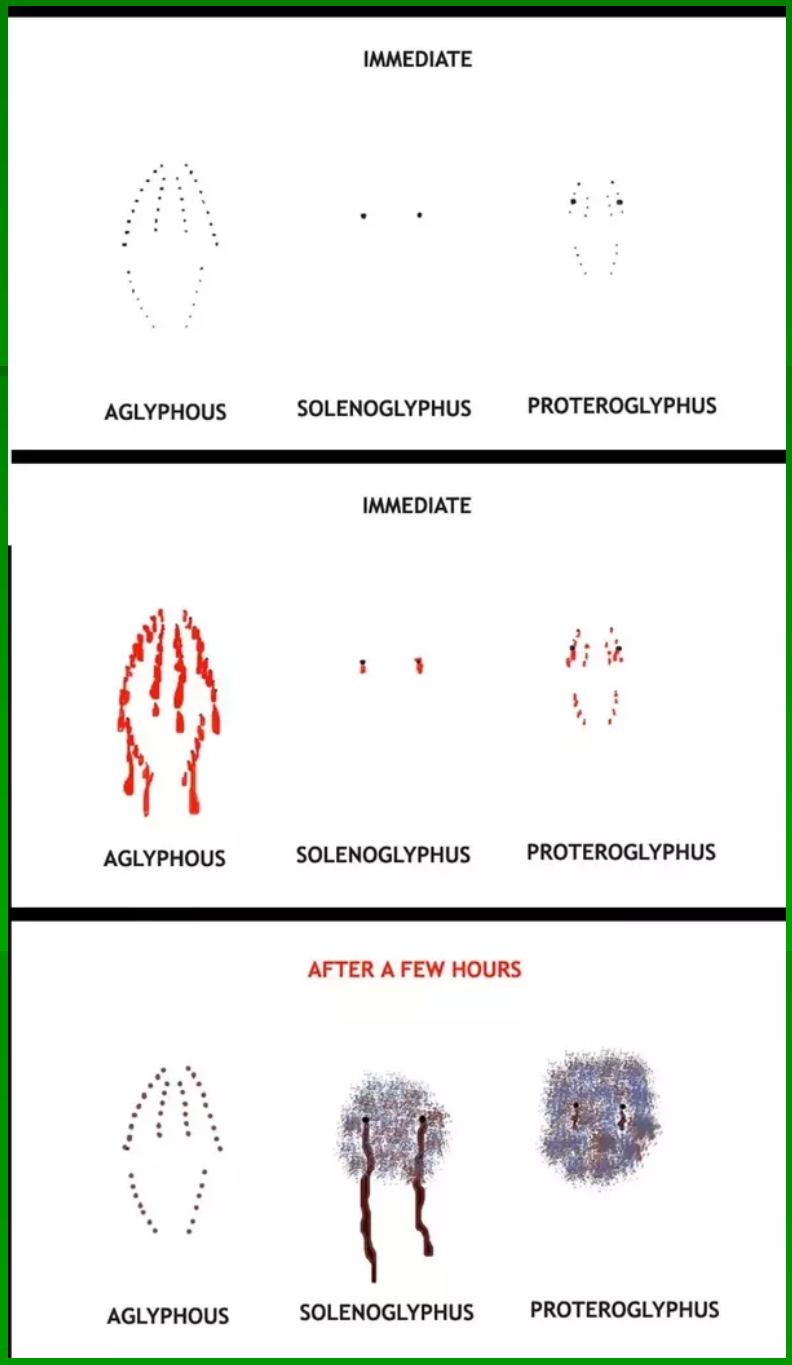


Bites by venomous snakes

Local tissue damage:

fang marks
local pain;
local bleeding;
ecchymoses;
blistering;
progressive swelling;
lymphadenopathy;
necrosis;

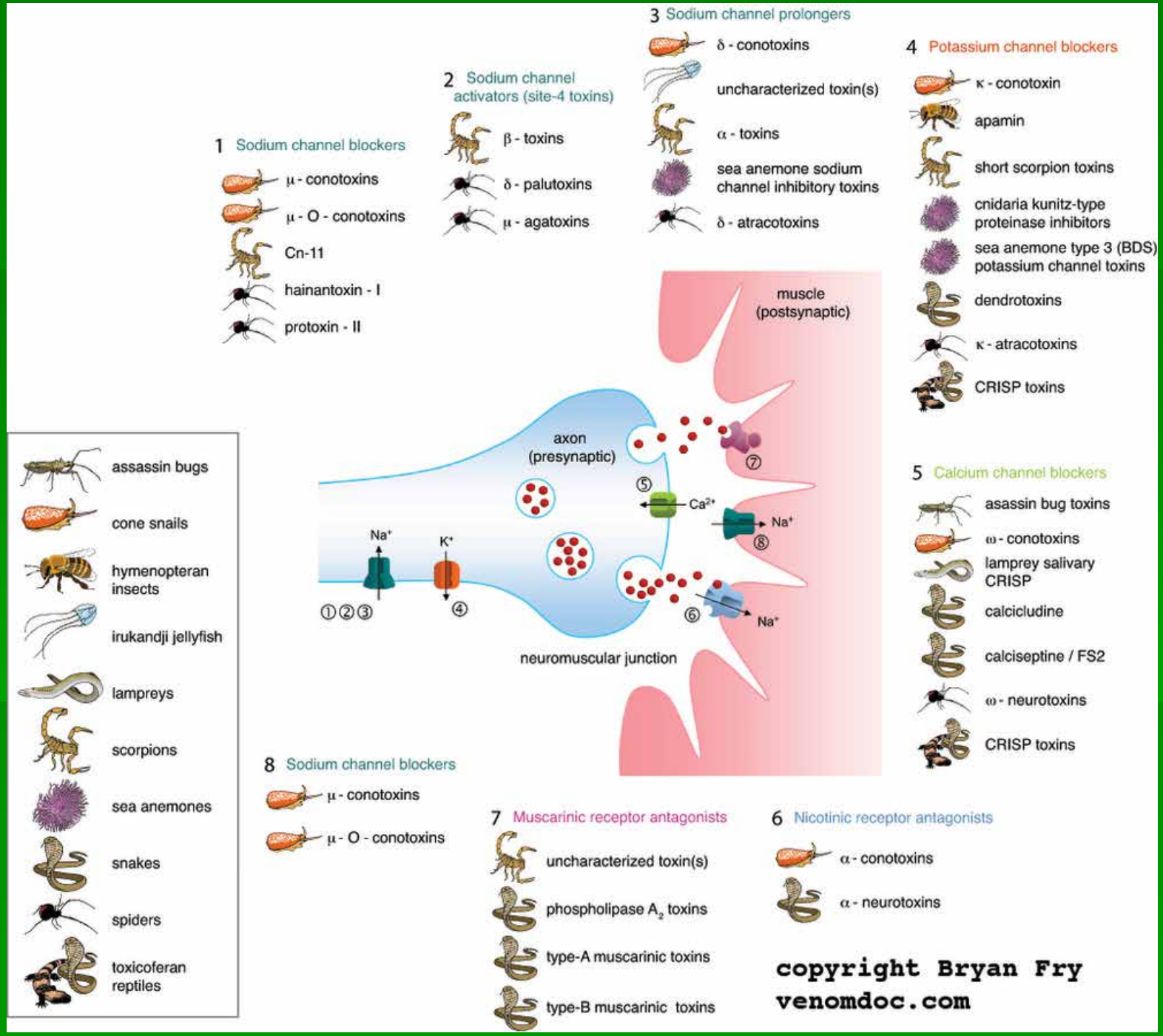
Bites by venomous snakes



Bites by venomous snakes

Paralytic neurotoxicity:

ptosis,
ophthalmoplegia,
diplopia,
dysphagia,
dysarthria,
drooling of saliva,
limb weakness,
respiratory muscle paralysis


1 Sodium channel blockers
μ - conotoxins
μ - O - conotoxins
Cn-11
hainantoxin - I
protoxin - II
2 Sodium channel activators (site-4 toxins)
β - toxins
δ - palutoxins
μ - agatoxins
3 Sodium channel prolongers
δ - conotoxins
uncharacterized toxin(s)
α - toxins
sea anemone sodium channel inhibitory toxins
δ - atracotoxins
4 Potassium channel blockers
κ - conotoxin
apamin
short scorpion toxins
cnidaria kunitz-type proteinase inhibitors
sea anemone type 3 (BDS) potassium channel toxins
dendrotoxins
κ - atracotoxins
CRISP toxins
muscle (postsynaptic)
axon (presynaptic)
Na+
K+
Ca2+
Na+
Na+
①②③
④
⑤
⑥
⑦
⑧
neuromuscular junction
5 Calcium channel blockers
asassin bug toxins
ω - conotoxins
lamprey salivary CRISP
calcicludine
calciseptine / FS2
ω - neurotoxins
CRISP toxins
assassin bugs
cone snails
hymenopteran insects
irukandji jellyfish
lampreys
scorpions
sea anemones
snakes
spiders
toxicoferan reptiles
8 Sodium channel blockers
μ - conotoxins
μ - O - conotoxins
7 Muscarinic receptor antagonists
uncharacterized toxin(s)
phospholipase A2 toxins
type-A muscarinic toxins
type-B muscarinic toxins
6 Nicotinic receptor antagonists
α - conotoxins
α - neurotoxins
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Bites by venomous snakes

Excitatory neurotoxicity:

perioral paraesthesiae,
sweating,
salivation,
piloerection,
pulmonary oedema,
autonomic storm

Bites by venomous snakes

Haemotoxicity:

prolonged bleeding from wounds,bite or venipuncture sites;

asymptomatic coagulopathy with prolonged INR;

haematemesis;

melaena;

haematuria

Bites by venomous snakes

Myotoxicity:
muscle pain and tenderness,
weakness,
rhabdomyolysis,
acute kidney injury

Bites by venomous snakes

Bites by venomous snakes

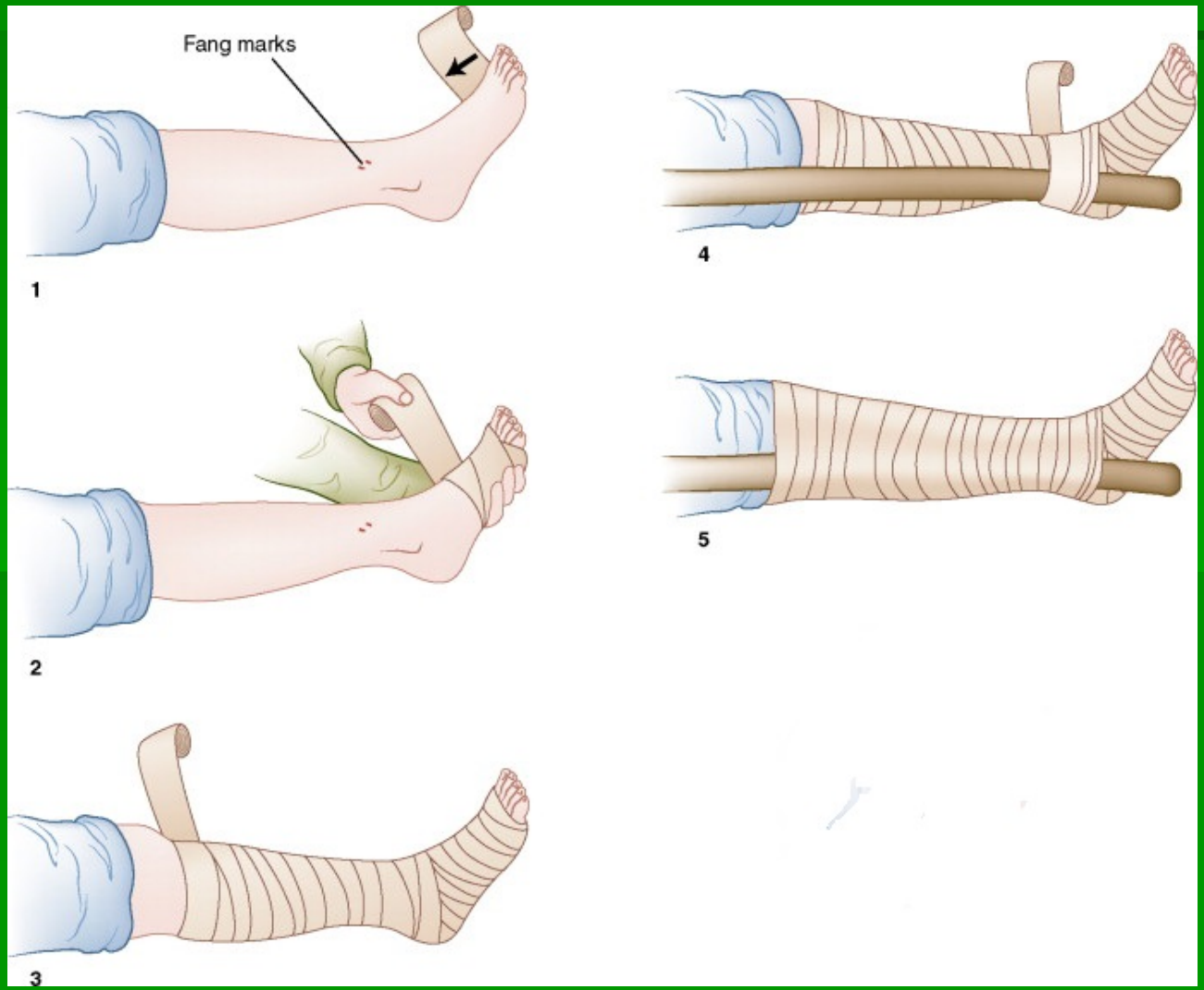
Don’t

try to catch the snake
cut the wound
use a tourniquet
use ice on the wound
drink any alcohol or caffeine

Bites by venomous snakes

Remove clothing, jewelry, or constricting items
Clean the wound
Immobilize and support the area of the bite

Bites by venomous snakes



Bites by venomous snakes

Bites by venomous snakes

1. There is no weight based calculation for antivenom - the snake delivers the same amount of venom regardless of the size of the body.
2. One vial of antivenom is enough to neutralize the venom that can be delivered by one snake.
3. Clinical recovery takes time after antivenom administration and multiple vials do not speed recovery.

Literature

- Brent, J., Burkhart, K., Dargan, P., Hatten, B., Megarbane, B., Palmer, R., & White, J. (2017). . Cham: Springer International Publishing.
- Emergency Management of Poisoning. Haddad and Winchester's Clinical Management of Poisoning and Drug Overdose. 2007;13-61. doi:10.1016/B978-0-7216-0693-4.50007-4