



GENERAL TREATMENT OF POISONING

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INTRODUCTION

Poison: a substance (solid, liquid or gaseous), which if introduced into the living body ,or brought into contact with any part there it will produce ill health or death by its constitutional or local effects or both.

Most poisons are dose related

A stylized, light gray leaf graphic is positioned in the upper left corner of the slide. It features several elongated, pointed leaflets branching from a central stem.

TYPES OF POISONING

Acute poisoning: excessive single dose or several smaller doses of a poison taken over a short interval of time.

Ingestion of hepatotoxins (i.e.. acetaminophen) may cause acute liver failure that occurs one to a few days later.

Chronic poisoning: smaller doses over a period of time, resulting in gradual worsening. i.e. Arsenic, metals or hydrocarbon solvents.

NATURE OF POISONING

Homicidal: Killing of a human being by another human being by administering poisonous substance deliberately.

Suicidal: When a person administers poison himself/herself to end his/her life.

Accidental: Household poisons (nail polish, acetone).

Occupational: In professional workers, i.e. insecticides.

CLASSIFICATION OF POISONS (BASED ON MOA)

Corrosive Poisons: Cause direct damage to tissues they come into contact with.

- Strong acids: H_2SO_4 , HNO_3 , HCl .
- Strong alkali: Hydrates, carbonates, NH_3 .
- Metallic salts: Zinc chloride, ferric, KCN .

Irritant Poisons: Cause inflammation and irritation to tissues, and are further subdivided into:

- Organic Irritants: Phenol and mustard gas.
- Inorganic Irritants: Ammonia and chlorine.
- Mechanical Irritants: Fiberglass and dust.

Systemic Poisons: Affect the body systemically after being absorbed, and are further categorized into:

- Neurotoxins: Organophosphates, tetrodotoxin, botulinum toxin.
- Cardiotoxins: Aconitine.
- Asphyxiants: CO and cyanide.

Miscellaneous Poisons: Snake venom, lead, mercury, arsenic, bisphenol A (BPA), and phthalates.

POISONS MAY ACT :

Locally: Cause damage at the site of contact or entry. Examples: Corrosives and Irritants.

Remotely: Exert their effects on organs or tissues distant from the site of entry. Examples: Neurotoxins and Cardiotoxins.

Combined Poisons: These have both local and remote effects. They damage the site of entry and also affect other systems in the body. Examples:

- **Cyanide:** Causes local irritation and systemic effects by inhibiting cellular respiration.
- **Arsenic:** Causes skin lesions (local effect) and multi-organ damage (remote effect).

FACTORS MODIFYING THE ACTION OF POISONING

1. **Mode of Administration:** Injectable, inhalation, oral, through unbroken skin, eye contact with toxins, through natural orifices.
2. **Dose / Quantity:** More severe toxic effects are associated with higher doses.
3. **Condition of the Patient:** Age, state of health, sleep and intoxication, state of stomach, cumulative action.
4. **Form of the Poison:** Physical, chemical, mechanical.

CAUSATIVE AGENT	EFFECT (SIGNS/SYMPTOMS)
Opioids:	
Heroin	
Morphine	Hypoventilation
Methadone	Hypotension Miosis
Pentazocine	Sedation
Propoxyphene	Possibly Hypothermia
Primary Antidote = Naloxone	

CAUSATIVE AGENT	EFFECT (SIGNS/SYMPTOMS)
Nicotine	
Black widow spider bite	
Carbamates	Tachycardia Hypertension
	Fasciculations Abdominal pain Paresis
Some Organophosphate	

CAUSATIVE AGENT	EFFECT (SIGNS/SYMPTOMS)
Amphetamines	
Caffeine	Tachycardia Hypertension
Cocaine	Mydriasis
Ephedrine	Agitation and Seizures
	Diaphoresis Hyperthermia
Theophylline (for asthma)	
Benzodiazepines, Beta blockers, Activated charcoal.	

CAUSATIVE AGENT	EFFECT (SIGNS/SYMPTOMS)
Antihistamines	
Atropine	Tachycardia Mydriasis
Scopolamine	Hyperthermia Warm and Dry skin Urinary Retention Constipation
Jimson weed	
Tricyclic antidepressants	Delirium
Many Psychotic drugs	

DIAGNOSIS OF POISONING

1. **History:** Check multiple sources: patient itself, family, friends, witness.
2. **Circumstantial Evidence:** Suicide note, containers & potential toxins at scene of discovery. * What was taken? * How much? * And when?
3. **Physical Examination:** Vital signs, needle tracks, bruising, odor.
4. **Investigations:** Biochemical investigations, ECG abnormalities, toxicologic screening, and urinalysis.

INITIAL STABILIZATION

TREATMENT OF ANY SYSTEMIC POISONING

- Begins with: ABC stabilization
- If patient has apnea or compromised airways (foreign material in the oropharynx, decreased gag reflex), an endotracheal tube should be inserted.
- If patient has respiratory depression or hypoxia, supplemental O₂ or mechanical ventilation should be provided as needed in patients with apnea.
- IV naloxone (2 mg in adults – 0.1 mg/kg in children) should be tried while airway support is maintained.
- In opioid addicts, naloxone may precipitate withdrawal, but withdrawal is preferable to apnea.
- If respiratory depression persists despite use of naloxone → endotracheal intubation and continuous mechanical ventilation are required.

INITIAL STABILIZATION (continued)

- If plasma glucose is low → IV dextrose should be given (50 ml of a 50% solution for adults; 2–4 ml/kg of a 25% solution for children).
- For adults with suspected thiamine deficiency (alcoholics, malnourished patients): IV thiamine (100 mg) is given with or before glucose.
- Hypotension is treated with IV fluids. The first-choice vasopressor for most poison-induced hypotension is norepinephrine (0.5–1 mg/min IV infusion).
- Flumazenil: antidote for benzodiazepine overdose
 - If no response → 0.3 mg IV after 1 minute
 - If no response again → 0.5 mg IV → To max cumulative dose of 3 mg/h.

COMA PATIENTS:

- Check ABC.
- Take blood samples for histology, hematology, and toxicology.
- Give:
 - Dextrose 50% (IV)
 - Thiamine (IM or IV)
 - Naloxone (IV) (2–3 hr duration of action)
 - Flumazenil (IV) (2–3 hr duration of action)
- Even if a patient is known to be poisoned, altered consciousness may have other causes (CNS infection, head trauma, hypoglycemia, stroke, hepatic encephalopathy), which should also be considered.
- If you know the toxin, initiate the specific treatment ASAP.
- If you don't know the toxin, treatment is given on general lines.
- Aim of treatment: → To keep the patient alive by attention to respiration and circulation.

REMOVAL OF UNABSORBED POISON:

- IT DEPENDS ON THE ROUTE OF ADMINISTRATION.

Inhaled poison:

The patient should be removed into fresh air

Artificial respiration and O₂ (6-8 L/min) should be given

Air passages should be kept free from mucus by postural drainage or by suction.

Injected poison:

A ligature should be tied above the point of injection to prevent it from further reaching the circulation.

The ligature is loosened for one minute after every 10 min to prevent gangrene formation.

Contact poison:

In case of affecting the skin, mucus membranes, any wounds: flush with water and repeat at least for 20-30 mins

Saline is preferred for eye irrigation.

if introduced into the body cavities (vagina, rectum): irrigate with water and neutralize by application of suitable chemical

Ingested poison: (gastric decontamination)

Gastric lavage Emetics

Dilution

Other methods

General rule:

< 4 hours → induce emesis because it's in the stomach.

> 4 hours → give laxatives because it's in the bowel.

-Snake bite: snake

Antihistamine, Hydrocortisone, Tetroxide, Antivenom.

-Scorpion bite: scorpion antivenom.

*Best thing is immobilization



TREATMENT OF INGESTED POISONS

TREATMENT OF INGESTED POISONS

- The stomach should be emptied by gastric lavage or emesis. Appropriate antidote should be given.
- Aid elimination of poison by intestines and kidneys of what has been absorbed .
- Symptomatic treatment.
- Egg whites are useful in most poisonings and tannin is antidote to all alkaloids.
- In ingested poisons , the methods of decontamination are:
 - I. Gastric lavage.
 - II. Emesis.
 - III. Catharsis.
 - IV. Administration of activated charcoal.
 - V. Whole bowel irrigation.

STOMACH WASH

- GASTRIC LAVAGE IS INDICATED IN PATIENTS WHO PRESENT **WITHIN 3 HOURS OF INGESTION OF POISON.**
- GASTRIC LAVAGE BEYOND 6-12 HOURS IS RECOMMENDED IN INGESTION OF :
 - SALICYLATES, TRICYCLICS, CARBAMAZEPINE AND BARBITURATE. Due to factors that cause delayed and prolonged absorption of the drug from the stomach.
- https://youtu.be/NCTt1HemvOA?si=cFdHQnS0E3kzGQ_t

The gastric lavage tube:
a soft rubber tube.
In adults: 36-40 French size
1 cm diameter
1/2 meter length.
In children: 22-28 French size.



Step 1: The patient should be lying flat lateral position or in a prone position with head hanging over the edge of the bed & the face is positioned downwards so the mouth is at lower level than larynx and chances of aspiration of fluid are eliminated.

Step 2: The gastric lavage tube is gently passed in to the stomach through the mouth. -The position of the tube in stomach can be checked by stethoscope or putting the upper end in a cut of water, if the lower end in the trachea air bubbles will come out.

Step 3: for carrying out stomach wash, initially pass 250-300ml of warm saline or plain water. In children: normal saline is used as children are more at risk of developing hyponatremia if lavaged with water. the stomach contents can be siphoned by the use of suction tube.

- Contraindications:
 - i. Insignificant overdose.
 - ii. Corrosive poisoning
 - iii. EXCEPT carbolic acid as there is danger of perforation.
 - iv. Volatile poisons due to the risk of inhalation.
 - v. Hydrocarbon Ingestion.
 - vi. Comatose patients as there is risk of aspiration.
 - vii. If the patient is hypothermic.
- Complications:
- Aspiration pneumonia.
- Laryngeal trauma/spasm
- Esophageal/stomach perforation.
- Electrolyte imbalance.
- Hypothermia.
- Sinus bradycardia and ST elevation on ECG

Emesis

- ONLY USED IF THERE IS DIFFICULTY IN OBTAINING GASTRIC LAVAGE; DUE TO DANGER OF ASPIRATION OF GASTRIC CONTENTS, VOMITING SHOULD ONLY BE INDUCED IN A CONSCIOUS PATIENT.
- -THE EASIEST WAY TO INDUCE VOMITING IS BY TICKLING THE FAUCES (OROPHARYNGEAL ISTHMUS) - THE ARCHED OPENING AT THE BACK OF THE MOUTH LEADING TO THE PHARYNX.
- IPECACUANHA POWDER:
 - ✓ IT IS DERIVED FROM THE ROOTS OF TWO PLANTS: CEPHALIS IPECACUANHA AND C. ACUMINATA. THE ACTIVE INGREDIENTS ARE: CEPHAELINE, EMETINE, AND TRACES OF PSYCHOTROPINE.
 - ✓ ACTIVATE SENSORY RECEPTORS IN THE GIT.
 - ✓ STIMULATE THE CHEMORECEPTOR TRIGGER ZONE (CTZ) AND THE VOMITING CENTER IN THE BRAIN,

Emetic substances:

- ✓ Ipecac is the most effective.
- ✓ Warm saline water.
- ✓ Mustard powder.
- ✓ Zinc sulphate (no longer used).
- ✓ Apomorphine (no longer used).
- ✓ Ipecacuanha powder

CON

Contraindications:

- Pregnancy.
- Heart disease.
- Hemorrhagic diathesis.
- Cardiotoxic poison ingestion.
- In infants and old patients.
- Comatose or unconscious patients.

Complications:

- Features of cardiotoxicity such as bradycardia, atrial fibrillation and myocarditis.
- Aspiration pneumonitis
- Esophageal tears.

CATHARTICS

Side effects:

Abdominal cramps, N/V.

Complications:

Excessive diarrhea,

Hypermagnesemia

Contraindications:

Corrosives.

Preexisting diarrhea.

- Promote rectal evacuation of GI contents.

- Used substances:

1. Salts:

- ✓ Disodium phosphate,

- ✓ Magnesium citrate & sulfate,

- ✓ Sodium sulfate.

2. Saccharides:

- Mannitol

- Sorbitol (most effective)

Whole bowel Irrigation

- ADMINISTRATION OF BOWEL CLEANSING SOLUTION CONTAINING ELECTROLYTES & POLYETHYLENE GLYCOL
- ORALLY OR THROUGH GASTRIC TUBE.

❖ RATE:

- THE SOLUTION IS ADMINISTERED AT A RATE OF:
 - ✓ 500 ML/H IN CHILDREN (9 MONTHS TO 6 YRS.).
 - ✓ 1000 ML/H IN CHILDREN (9 TO 12 YRS.).
 - ✓ 1500 TO 2000 ML/H IN ADOLESCENTS AND ADULTS.

❖ INDICATIONS:

- SLOW OR ENTERIC COATED MEDICATIONS.
- PACKETS OF ILLICIT DRUGS.
- HEAVY METALS; IRON, LITHIUM.

Contraindications:

- i. Bowel obstruction.
- ii. Ileus.
- iii. Unprotected airway.

Complications:

- I. Bloating.
- II. Cramping.
- III. Rectal irritation.



ADMINISTRATION OF ANTIDOTES

1. MECHANICAL OR PHYSICAL ANTIDOTES.
 2. CHEMICAL ANTIDOTES.
 3. PHYSIOLOGICAL ANTIDOTES.
 4. UNIVERSAL ANTIDOTES (NOT USED ANYMORE)
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Mechanical Antidote

- Mechanism of action: They counteract the effect of poison mechanically preventing their absorption without inactivating the damaging action of the poison.
- Types:
- Adsorbents: Activated charcoal.
- Demulcents: Milk, egg white, starch,
- Diluents: milk
- Bulky foods: bananas, boiled rice, potatoes.

ACTIVATED CHARCOL

- Activated charcoal: fine , black , odorless powder produced by destructive of various organic materials, usually wood pulp and then treating at high temperature with a variety of activating agents, such as steam or CO_2 , to **increase its adsorptive capacity**.
- Dose: 40-80 g (dose: 0.5-1 g/kg) is mixed with **water to form a soup-like mixture and given orally**.
- **Palatability** may be increased by adding a sweetener (sorbitol) or a flavoring agent (cherry, chocolate, or cola syrup) to the suspension.

Con

Uses: In cases of poisoning with

1. morphine, atropine, nicotine,
2. barbiturates, salicylic acid
3. ,phenol and highly dissociated
4. salts of cyanide.

Side effects:

N/V, aspiration, altered
bowel habit.

Also, Charcoal may also prevent
the absorption of orally
administered therapeutic
agents.

Doesn't adsorb

C- Caustics, Corrosives.

H- Heavy metals.

A- Alcohol.

R- Rapid onset cyanide.

C- Chlorine

O- Others (Iron)

A- Aliphatic hydrocarbon.

L- Lithium

chemical antidotes

albumin

for mercury poisoning

copper sulphates

for phosphorus

Tincture iodine or lugol's iodine

tannic acids

or strong tea used for lead , mercury , nickel , zinc , copper poisoning

the chemical antidotes disintegrate and inactivate poisons by undergoing chemical reactions along with the poisons and forming harmless or insoluble compounds

weak vegetable acids

citric acid , and acetic acid (vinegar , lemon juice) , it can be used for corrosive alkaline poisoning

Weak non-carbonate alkalis

in corrosive acid poisoning , weak non-carbonate alkalis act as neutralizers . Strong alkalis are avoided as they can cause further damage to the stomach

potassium permanganate

used in poisoning with oxidizable substances like cyanides , phosphorus , atropin and other alkaloids

PHYSIOLOGICAL ANTIDOTE

the physiological antidote acts on the tissues and various systems of the body and produce signs and symptoms opposite to the signs and symptoms produced by the poison

examples :

- atropine for organophosphorus compounds , physostigmine
- neostigmine for datura
- barbiturates for strychnine

Toxin	Antidote
Acetaminophen	N- Acetylcysteine
Anticholinergics	Physostigmine
Benzodiazepines	Flumazenil
β-Blockers	Glucagon
Ca channel blockers	Ca IV insulin in high doses with IV glucose
Carbamates	Atropine Pralidoxime chloride
Cyanide	Hydroxocobalamin Cyanide antidote kit(includes amyl nitrate, Na nitrite, and Na thiosulfate)

TOXIN	ANTIDOTE
Heavy metals	Chelating drugs
Iron	Desferoxamine
Isoniazid	Pyridoxine (vitamin B ₆)
Methanol	Ethanol Fomepizole
Methemoglobin-forming agents (eg, aniline dyes, some local anesthetics, nitrates , phenacetin, sulfonamides)	Methylene blue



CHELATING AGENTS

USED AS A SPECIFIC ANTIDOTES AGAINST SOME HEAVY METAL
POISONING, AS THEY HAVE
GREATER AFFINITY FOR THE METALS AS COMPARED TO THE
ENDOGENOUS ENZYMES.

CHELATING AGENTS



BRITISH ANTI-LEWISITE
(B.A.L.)
DIMERCAPTOPROPANOL

DEFERRIOXAMINE MESYLATE
OR DEFEROXAMINE

PENICILLAMINE
(CUPRIMINE)

E.D.T.A ETHYLENE
DIAMINE
TETRAACETIC ACID



B.A.L. DIMERCAPTOPROPANOL

MECHANISM OF ACTION

It has two unsaturated $-SH$ groups which combine with the metal and thus **prevent the union of the metal with the $-SH$ group of respiratory enzyme system.**

USES & DOSE

Used in **arsenic, lead, bismuth, copper, mercury, gold** and other heavy metal poisoning.

10% solution in oil (3-5 mg/kg) IM, 4 hourly for 2 days, 6 hourly for 1 day, and then 12 hourly for 10 days.

SIDE EFFECTS

N/V, headache, hypertension, anorexia, generalized aches and pain.

CONTRAINDICATIONS

Liver damage

G-6-PD deficiency

Cadmium and iron poisoning

ETHYLENE DIAMINE TETRAACETIC ACID

PHARMACOKINETICS

Superior to BAL in treatment of poisoning with **mercury and arsenic**.

Distributed only **extracellularly**.

Excreted by urine.

Given **I.V**

USES & DOSE

Effective in **lead, mercury, copper, cobalt, cadmium, iron and nickel poisoning**.

Dose: 25-30 mg/kg in 250-500 ml of 5% glucose or NS over 1-2 hour, twice daily for 5 days and may be repeated after 2-3 days.

SIDE EFFECTS

Thrombophlebitis, N/V, vertigo, fever, headache, hypersensitivity.

CONTRAINDICATIONS:

Renal damage

Raised intracranial pressure

CHELATING AGENTS

Penicillamine (Cuprimine)

For: copper, lead and mercury poisoning.

Other uses: Wilson's disease, cystinuria and scleroderma.

Dose: 30 mg /kg up to a total of 2 g/day in 4 divided doses, orally for about 7 days.

Deferoxamine

In acute iron poisoning

used in transfusional chronic iron overload.

Dose: 2 g in 5% of laevulose solution given I.V. and repeated after 12 hours.

elimination of poison by excretion

○ Indication

- severe poisoning
- progressive deterioration, in spite of full supportive care
- when there is high risk of morbidity and mortality
- when normal route of excretion of poison is impaired
- when poison produces delayed, but serious toxic effects

methods to elimination of poisons

➤ **SALINE DIURESIS**

can enhance the renal excretion of alcohol, Fluoride and thallium

➤ **ALKALINE DIURESIS**

can enhance the excretion of chlorophenoxyacetic acidher bicides, phenobarbital, salicylate.

➤ **ACIDIC DIURESIS**

enhances the excretion of amphetamine, cocaine, quinine phenocyclidine, sympathomimetics, tricyclic antidepressants

Alkaline diuresis

- Alkaline diuresis enhances **elimination of weak acids** (salicylates, phenobarbital)
- **Dose:** A solution made by combining 1L of 5% D/W with 350-mEq ampules of NaHCO₃ and 20 to 40 mEq of K.
- Urine pH is kept at >8.
- **Side effects:** Hypernatremia, alkalemia, and fluid overload may occur but are usually not serious.
- **Contraindications:** renal insufficiency

Acid diuresis

- In forced acid diuresis: ascorbic acid(vitamin C) is used.
- Ammonium chloride has also been, but it is a toxic compound.
- However, this technique only produces a slight increase in the renal clearance of the drug.
- It is rarely done in practice
- For: weak bases (e.g. phencyclidine),amphetamine and strychnine

Dialysis

- ❖ Methods of dialysis include: hemodialysis, peritoneal dialysis, and lipid dialysis (which removes lipid-soluble substances from the blood), as well as hemoperfusion (which more rapidly and efficiently clears specific poisons)
- ❖ BENEFITS:
 - removal of toxins already absorbed by gut ability to remove parent compound and active metabolite

Dialysis

❖ Common toxins that may require dialysis include:

- Ethylene glycol
- Lithicum
- Methanol
- Salicylates
- Theophylline

❖ These therapies are less useful if the poison is:

- Large or charged (polar) molecule
- Has a large volume of distribution (ie, if it is stored in fatty tissue)
- Extensively bound to tissue protein (as with digoxin, phencyclidine, phenothiazines, or tricyclic antidepressants).

HOSPITAL ADMISSION

- General indications for hospital admission include:
 - * altered consciousness
 - * persistently abnormal vital signs
 - * and predicted delayed toxicity

For example, admission is considered if patients have ingested sustained-release preparations, particularly of drugs with potentially serious effects (eg, cardiovascular drugs).

However, if ingestion was intentional, patients require a psychiatric evaluation



ACETAMINOPHEN POISONING

Acetaminophen poisoning is common

□ It can cause gastroenteritis within hours and hepatotoxicity 1 to 3 days after ingestion

To cause toxicity, an acute overdose must total ≥ 150 mg/kg (about 7.5 g in adults) within 24 h

Severity of hepatotoxicity after a single acute overdose is predicted by Serum acetaminophen levels.

□ Treatment is with N-acetylcysteine to prevent or minimize hepatotoxicity.



Stages of Acute Acetaminophen Poisoning

Stage	Time Post-ingestion	Description
I	0–24 h	Anorexia, nausea, vomiting
II	24–72 h	Right upper quadrant abdominal pain (common) AST, ALT, and, if poisoning is severe, bilirubin and PT (usually reported as the INR) sometimes elevated
III	72–96 h	Vomiting and symptoms of liver failure Peaking of AST, ALT, bilirubin, and INR Sometimes renal failure and pancreatitis
IV	> 5 days	Resolution of hepatotoxicity or progression to multiple organ failure (sometimes fatal)

ACETAMINOPHEN POISONING DIAGNOSIS

- Diagnosis considered in all patients with non?accidental ingestions that may be suicide attempts .

- * Serum acetaminophen levels
- * Rumack-Matthew nomogram :

If the time of ingestion is known, the Rumack?Matthew nomogram is used to estimate likelihood of hepatotoxicity ; if the time of ingestion is unknown, the nomogram cannot be used.



TREATMENT

- Oral or IV N- acetylcysteine □ is an antidote for acetaminophen poisoning. □
- The drug is most effective if given within 8 hrs of acetaminophen ingestion. After 24 h, the benefit of the antidote is questionable. □ It is equally effective given IV or orally
- □ Activated charcoal may be given if acetaminophen is likely to still remain in the GI tract. □
- Liver failure is treated supportively. Patients with fulminant liver failure may require liver transplantation.



Salicylate poisoning

Acute ingestion of > 150 mg/kg can cause severe toxicity. Salicylate tablets may form bezoars(Bezoars are tightly packed collections of partially digested or undigested material that can become stuck in the stomach or intestines), prolonging absorption and toxicity.

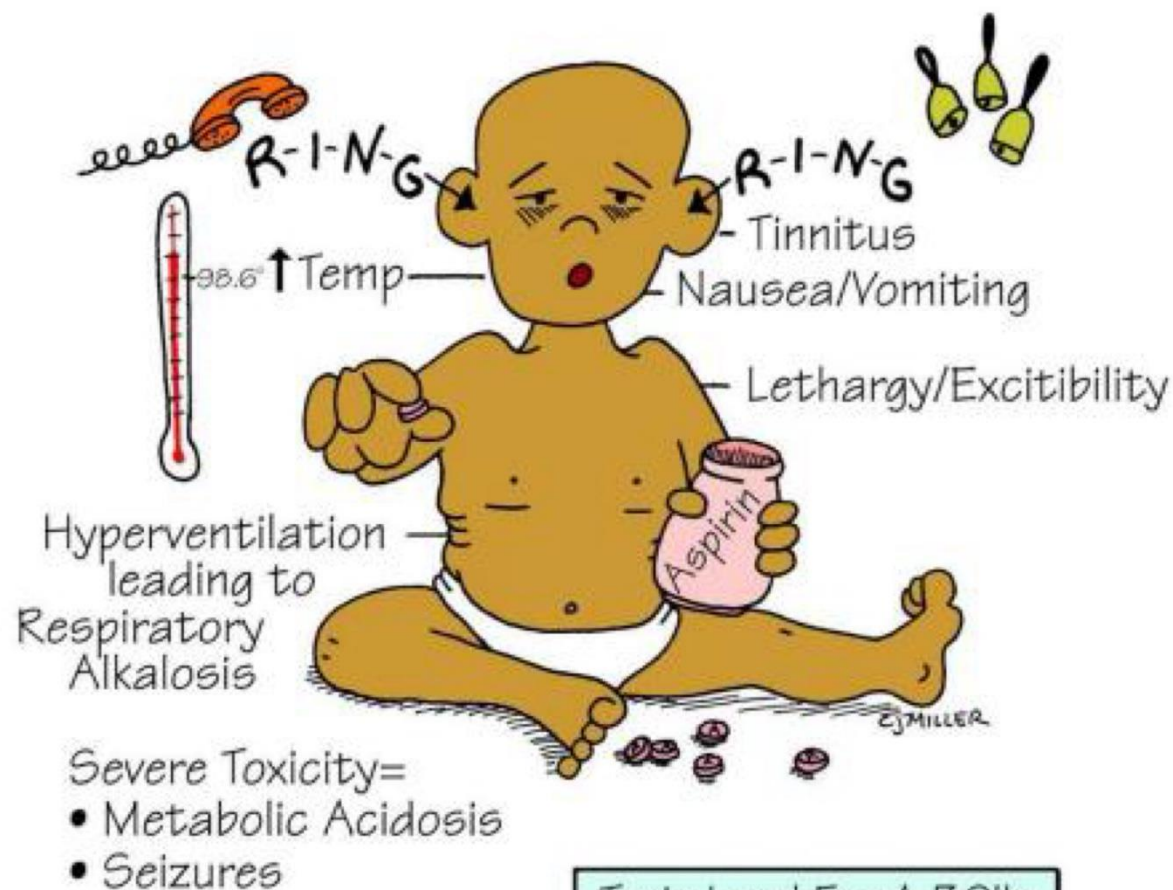
2. Chronic toxicity can occur after several days of high therapeutic doses; it is common, often undiagnosed, and often more serious than acute toxicity.

Chronic toxicity tends to occur in elderly patients



SIGNS AND SYMPTOMS

SALICYLATE POISONING



Toxic Level For A 30lb.
Child = 12 Adult Aspirin
or 48 Baby Aspirin.

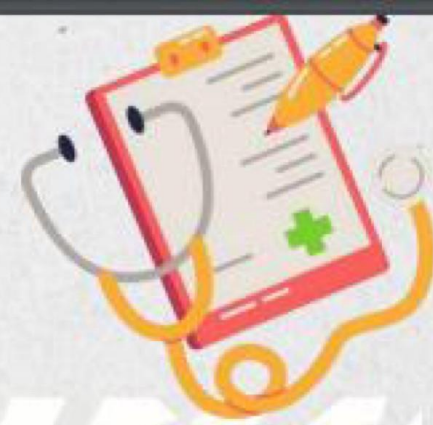
DIAGNOSIS OF SALICYLATE POISONING

1. ABGs (usually suggest **primary respiratory alkalosis** during the **first few hours** after ingestion. **Later**, they suggest **compensated metabolic acidosis** or **mixed metabolic acidosis/respiratory alkalosis**).

2. If poisoning is suspected serum salicylate (drawn at least a few hours after ingestion), urine pH, ABGs, serum electrolytes, serum creatinine, plasma glucose, and BUN are measured.

3.If rhabdomyolysis is suspected:
serum CK and urine myoglobin are measured.





TREATMENT

ACTIVATED CHARCOAL

Activated charcoal is given as soon as possible and, if bowel sounds are present, may be repeated every 4 hrs until charcoal appears in the stool

ALKALINE DIURESIS

a diuretic like **furosemide** is given intravenously and **sodium bicarbonate** is added to the infusion fluid to make blood and, in turn, urine alkaline ($\text{PH} \geq 8$)

Potassium replacement becomes of most importance in this setting because potassium is usually also lost in urine and hypokalemia promotes **bicarbonate** ion retention and prevents bicarbonate excretion, thus interfering with alkalization of the urine

SPECIAL NOTES ABOUT THE TREATMENT

- Fever can be treated with physical measures such as external cooling.
- Seizures are treated with benzodiazepines
- Hemodialysis may be required to enhance salicylate elimination in patients with severe neurologic impairment, renal or respiratory insufficiency
- Drugs that decrease respiratory drive should be avoided
- Drugs that increase urinary HCO_3^- (eg, acetazolamide) should be avoided because they worsen metabolic acidosis and decrease blood pH.



Thank you