

Principles of Toxicology

- ☠ Reduce exposure
- ☠ Reduce absorption
- ☠ Increase elimination
- ☠ Know when to intervene
- ☠ Give supportive care
- ☠ Give specific therapy and antidotes when appropriate



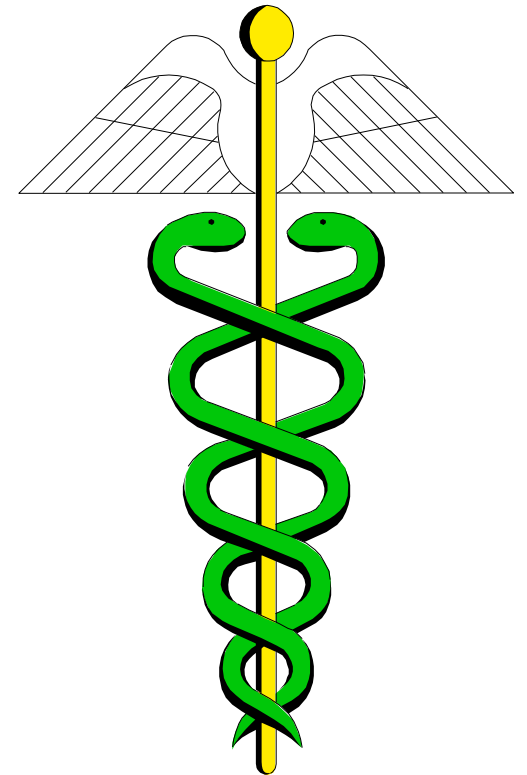
THE COMA COCKTAIL

 **DEXTROSE**

 **NALOXONE**

 **THIAMINE**

 **FLUMAZENIL (NO)**







Hypoglycemia

- ☠ Salicylates
- ☠ Acetaminophen
- ☠ Insulin
- ☠ Alcohol
- ☠ Oral hypoglycemic



**Check glucose in all patients with ODs
and those with altered mental status**

Gastric Decontamination Overview

☠ The vast majority of patients are unlikely to benefit from gastric decontamination:

- They have ingested nontoxic substances
- They have ingested nontoxic amounts of toxic substances
- They present long after decontamination would be expected to be of any benefit



Gastric Decontamination Overview

☠ Patients who theoretically may benefit from decontamination:

- Present early after ingestion (within 1 hour)
- Have taken a delayed-release product
- Are symptomatic before full absorption is likely to have occurred
- Have taken potentially life-threatening overdoses

☠ No prospective studies have demonstrated outcome benefits with gastric decontamination





Syrup of Ipecac



- ☠ Because of many limitations, not recommended in emergency departments
- ☠ Possible role in remote or long transport time settings
- ☠ Contraindications
 - Caustics, low viscosity hydrocarbons
 - Bleeding disorder, seizures, < 6 months
 - Altered mental status, unprotected airway

Gastric Lavage

☠ Now uncommonly used

☠ Possible Indications

- Recent (<1hr) life-threatening ingestion
- Agent not bound by charcoal

☠ Method

- Large-bore orogastric tube
- Left lateral decubitus position
- Protect airway



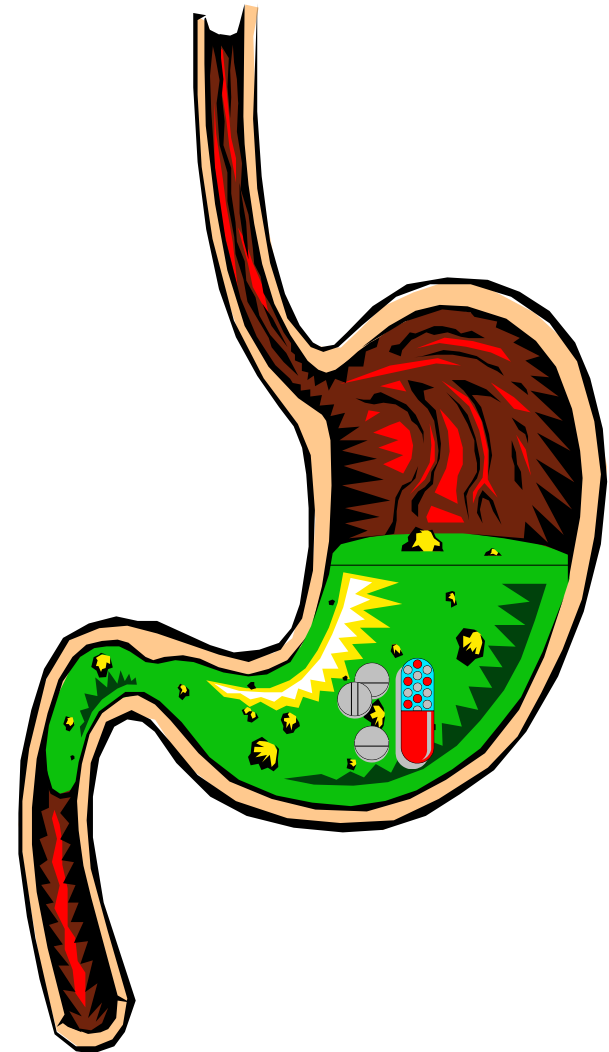
Gastric Lavage

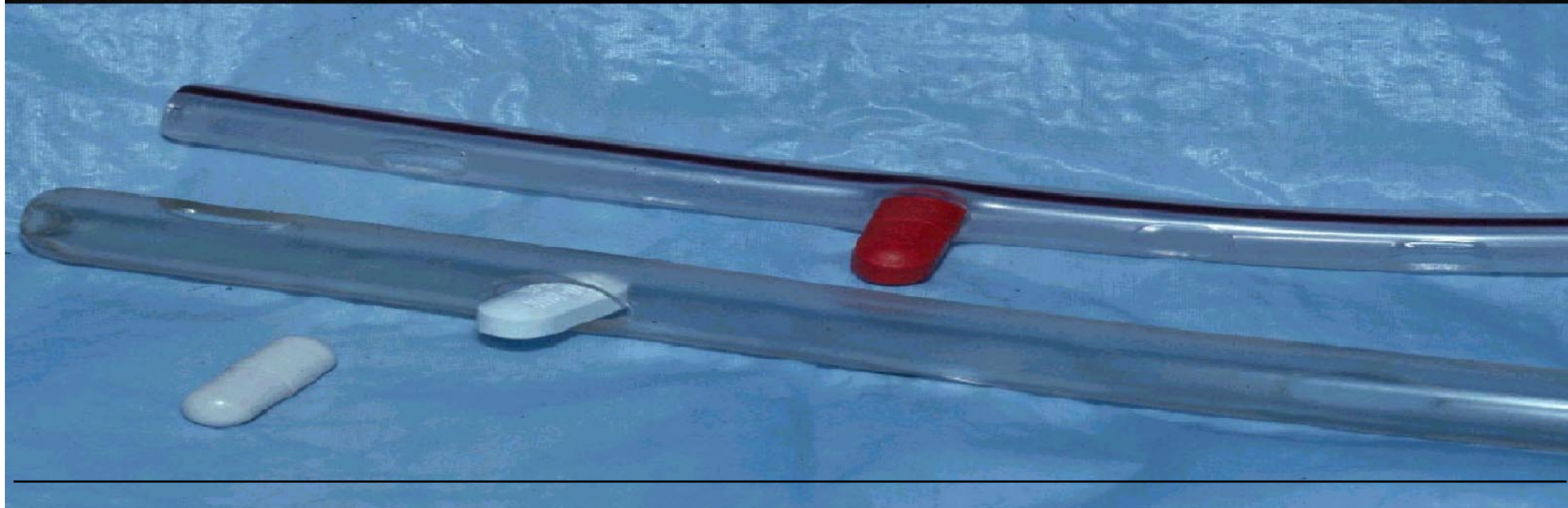
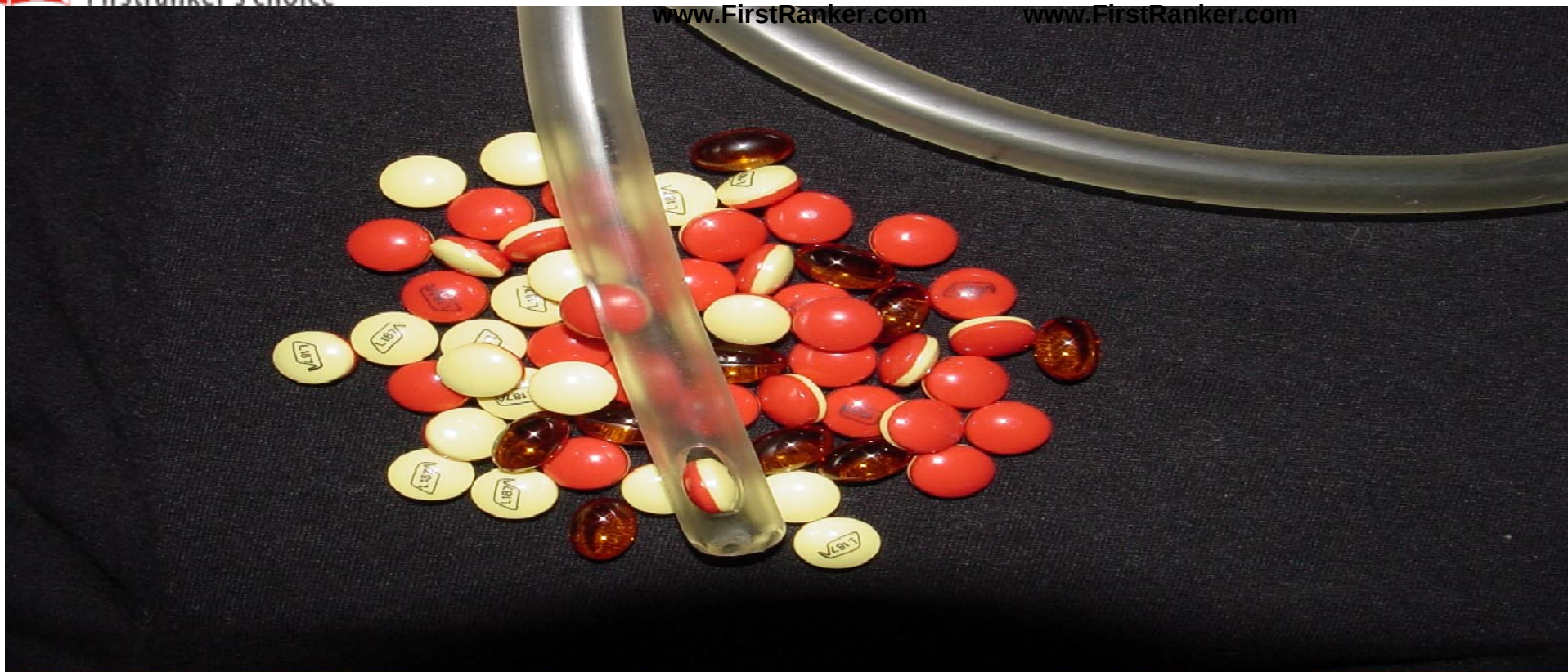
☠️ Contraindications

- Unprotected airway
- Caustics
- Nontoxic agents
- Hydrocarbons (unless they contain a dangerous additive)

☠️ Complications

- Aspiration / esophageal tears







Activated Charcoal

- ☠ Dose: 1-2 g/kg (max 100 g)
- ☠ **The optimal ratio of charcoal to toxin is 10:1**
- ☠ In massive overdoses, may require extra doses of charcoal
- ☠ Administration of oral charcoal is associated with little risk, a low threshold for use is reasonable unless full absorption of the drug has occurred

Activated Charcoal

- ☠ Enhanced elimination
 - Direct binding, enterohepatic recirculation, “gut dialysis”
- ☠ Poorly bound: Lithium, iron, caustics, cyanide, hydrocarbons, alcohols, alkali, acid
- ☠ Contraindications: Caustics, ileus, obstruction
- ☠ Multidose: Sustained release drugs, theophylline, phenobarbital, salicylates, carbamazepine

Avoid multidose cathartics (sorbitol, magnesium citrate) due to dehydration, electrolyte problems



Drug Elimination (1)

Charcoal hemoperfusion

- Protein-bound
- Theophylline, phenobarbital, carbamazepine

Whole bowel irrigation

- Sustained release preparations, bezoars
- Iron, lithium, drug packers, lead
- Contraindications
 - Ileus
 - Obstruction



Drug Elimination (2)

Avoid urine acidification and forced diuresis

- ☠ Urine alkalinization: Salicylates, phenobarb, INH
 - Urine pH goal is 7-8
 - Need to replace K^+ to alkalinize urine
- ☠ Hemodialysis: Salicylates, lithium, methanol, isopropanol, ethylene glycol, theophylline, phenobarbital
 - Low protein binding, low molecular weight
 - Small volume of distribution
 - Water-soluble

Agent / Antidote (1)

<u>Agent</u>	<u>Antidote</u>
Acetaminophen	Acetylcysteine (NAC)
Arsenic	BAL, DMS
Aspirin	Alkaline diuresis, hemodialysis
Beta-blocker	Glucagon, intralipids
Barbiturate	Alkaline diuresis, hemodialysis
Calcium channel blocker	Calcium, glucagon, gluc/insulin, intralipids
Carbamate	Atropine
Carbon monoxide	100% O ₂ , hyperbaric O ₂
Warfarin	FFP / Vit K / PCCs
Cyanide	Sodium nitrite, sodium thiosulfate, hydroxycobalamine
Digitalis	Digibind (fab antibodies)

Agent / Antidote (2)

<u>Agent</u>	<u>Antidote</u>
Ethylene glycol	Bicarbonate, ethanol, dialysis, 4-MP
Heparin	Protamine
Hydrofluoric acid	Calcium, magnesium
Iron	Deferoxamine
Isoniazid (INH)	Pyridoxine (Vit B-6)
Lead	BAL, DMS, EDTA
Mercury	BAL, DMS
Methemoglobin	Methylene blue
Methanol	Bicarbonate, ethanol, dialysis, 4-MP
Nitrites	Methylene blue
Opiates	Naloxone
Organophosphates	Atropine, 2-PAM
Oral hypoglycemic agents	Glucose, glucagon, octreotide
TCA	Sodium bicarbonate, intralipids

Formulas (1)

☠ Anion gap metabolic acidosis

$$(\text{Na}) - (\text{Cl} + \text{CO}_2), \text{ normal } \leq 12$$

☠ Increased

M Methanol, metformin, massive ingestions
U Uremia
D DKA
P Paraldehyde
I Iron, INH
L Lactic acidosis (CO, CN)
E Ethylene glycol
S Salicylates

Formulas (2)

☠ Osmolal gap = (measured) – (calculated)

☠ Normal gap <10

$$\text{Calculated (nl 285-295)} = 2 \text{ Na} + \frac{\text{BUN}}{2.8} + \frac{\text{Glu}}{18} + \frac{\text{EtOH}}{4.6}$$

☠ Increased gap: Acetone, isopropanol, methanol, ethylene glycol, mannitol, ketoacidosis, ethanol (most common)

Toxicology Screens

- ☠ Quantitative urine specimens are superior to blood specimens since drug metabolites can be detected 2-3 days after exposure
- ☠ Urine screens are specifically designed for drugs of abuse
- ☠ A positive or negative screen does not necessarily rule in or rule out an overdose



Toxicology Screens

☠ False positives:

- Amphetamines: Pseudoephedrine
- TCAs: Cyclobenzaprine, carbamazepine, phenothiazines, diphenhydramine
- PCP: Ketamine, dextromethorphan

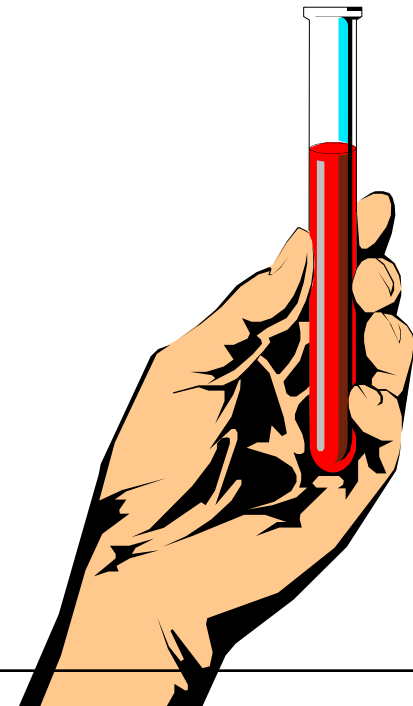
☠ False negatives:

- Dilute urine
- Methadone: Opioid screens
- MDMA: Amphetamines
- Rohypnol: Benzodiazepines



Specific Drug Levels

☠ Quantitative blood tests should be limited to those drugs for which levels can predict subsequent toxicity or guide specific therapy (e.g. iron, lithium, acetaminophen, ASA, theophylline, digoxin)



TOXIDROMES

☠ Anticholinergic

☠ Sympathomimetic

☠ Cholinergic

☠ Opioid

☠ Withdrawal



Anticholinergic (1)

☠ Atropine, tricyclic antidepressants, phenothiazines, antihistamines, antiparkinsonian drugs, Jimsonweed

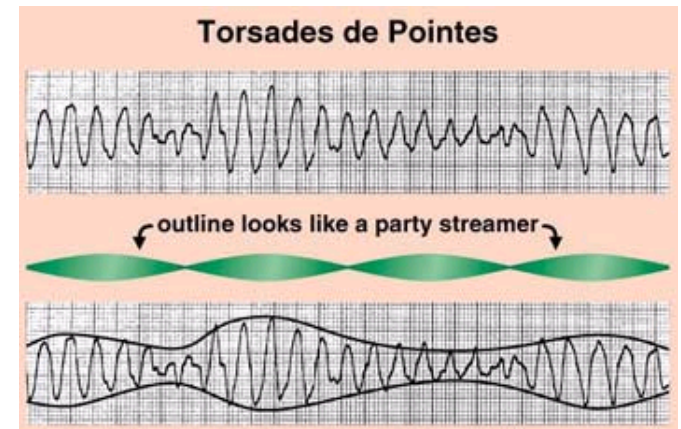
☠ “Hot as hell, blind as a bat, dry as a bone, red as a beet, mad as a hatter”

☠ Dry, flushed skin, dry mucus membranes, mydriasis, decreased bowel sounds, urinary retention

☠ **HOT / DRY / RED / BLIND / MAD**

Anticholinergic (2)

- ☠ Confusion, agitation, delirium, seizures
 - Treatment: Benzodiazepines
 - Avoid phenothiazines (e.g., prochlorperazine) and butyrophenones (e.g., haloperidol)
- ☠ Wide-complex tachycardia
 - Treatment: Sodium bicarbonate
- ☠ Ventricular dysrhythmias
 - Treatment: Lidocaine, amiodarone
 - Avoid procainamide
- ☠ Torsades de pointes
 - Treatment: Magnesium sulfate, overdrive pacing



Anticholinergic (3)

- ☠ Physostigmine: Consider if unresponsive to benzodiazepines; refractory seizures, ventricular dysrhythmias, hyperthermia malignant hypertension, hypotension
- Contraindicated in TCA overdose, heart block



Sympathomimetic

- ☠ Amphetamines, cocaine, PCP
- ☠ CNS excitation, seizures, hypertension, tachycardia, sweating, dilated pupils, hyperthermia



- ☠ Treatment: Benzodiazepines

Anticholinergic: Dry skin
Sympathomimetic: Diaphoresis

Opioids

☠ Codeine, morphine, heroin, demerol, diphenoxylate (Lomotil), propoxyphene (Darvon), hydrocodone (Vicodin), etc.

☠ Respiratory depression, ARDS, hypotension, coma

☠ Pinpoint pupils (not always seen)

☠ **Clonidine can mimic opioid OD**

☠ Treatment: Naloxone



Cholinergic

- ☠ Organophosphates, insecticides, some mushrooms, chemical warfare agents
- ☠ Inhibit cholinesterases → increase acetylcholine
- ☠ Muscarinic effects: **SLUDGE + Killer "B"s**
 - Salivation, Lacrimation, Urination, Diarrhea, Gastrointestinal upset, Emesis
 - Bradycardia, Bronchospasm, Bronchorrhea
 - Miosis (pinpoint pupils)



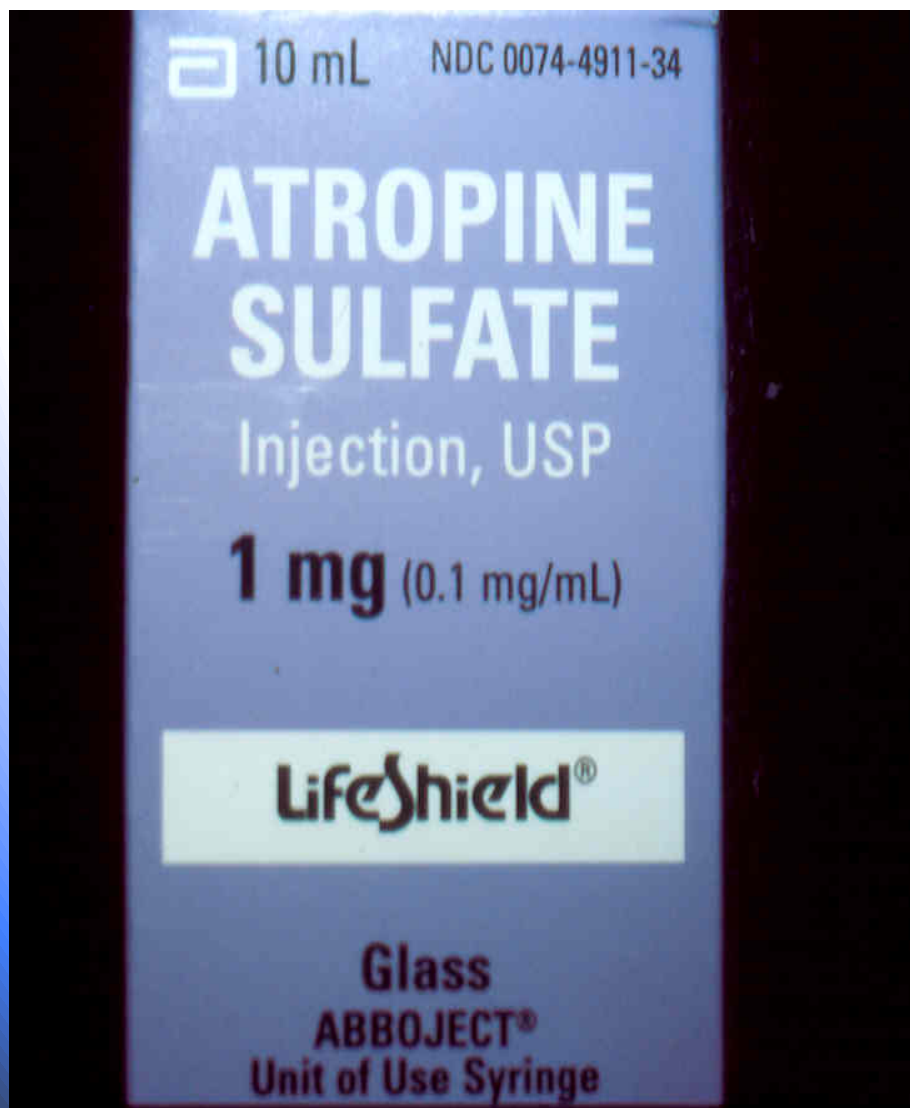
Organophosphates

- ☠ Nicotinic effects: Muscle weakness, fasciculations, respiratory failure
- ☠ Cholinesterase levels helpful in diagnosis but not helpful in management
- ☠ Treatment: Atropine (until secretions dry), Pralidoxime (2-PAM)

Mark-1 Autoinjection Kit

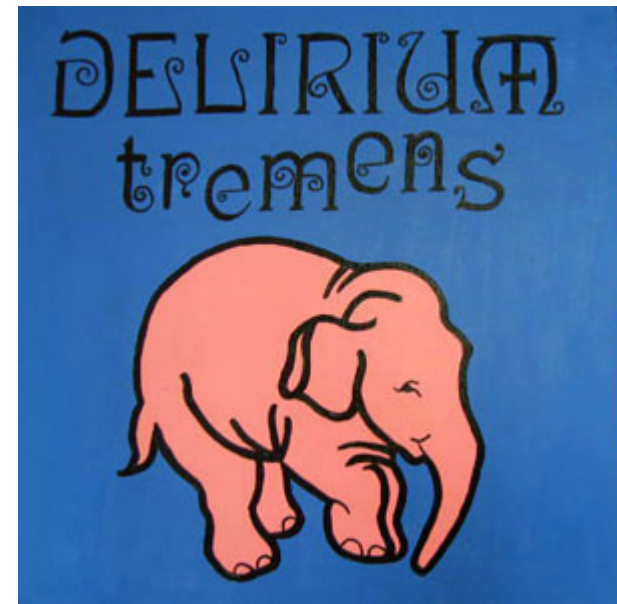


ATROPINE and 2-PAM



Withdrawal Syndromes

- Alcohol, barbiturates, benzodiazepines, cocaine, clonidine
- Diarrhea, mydriasis, hypertension, tachycardia, insomnia, cramps, diaphoresis, agitation, piloerection
- Treatment: Benzodiazepines, clonidine

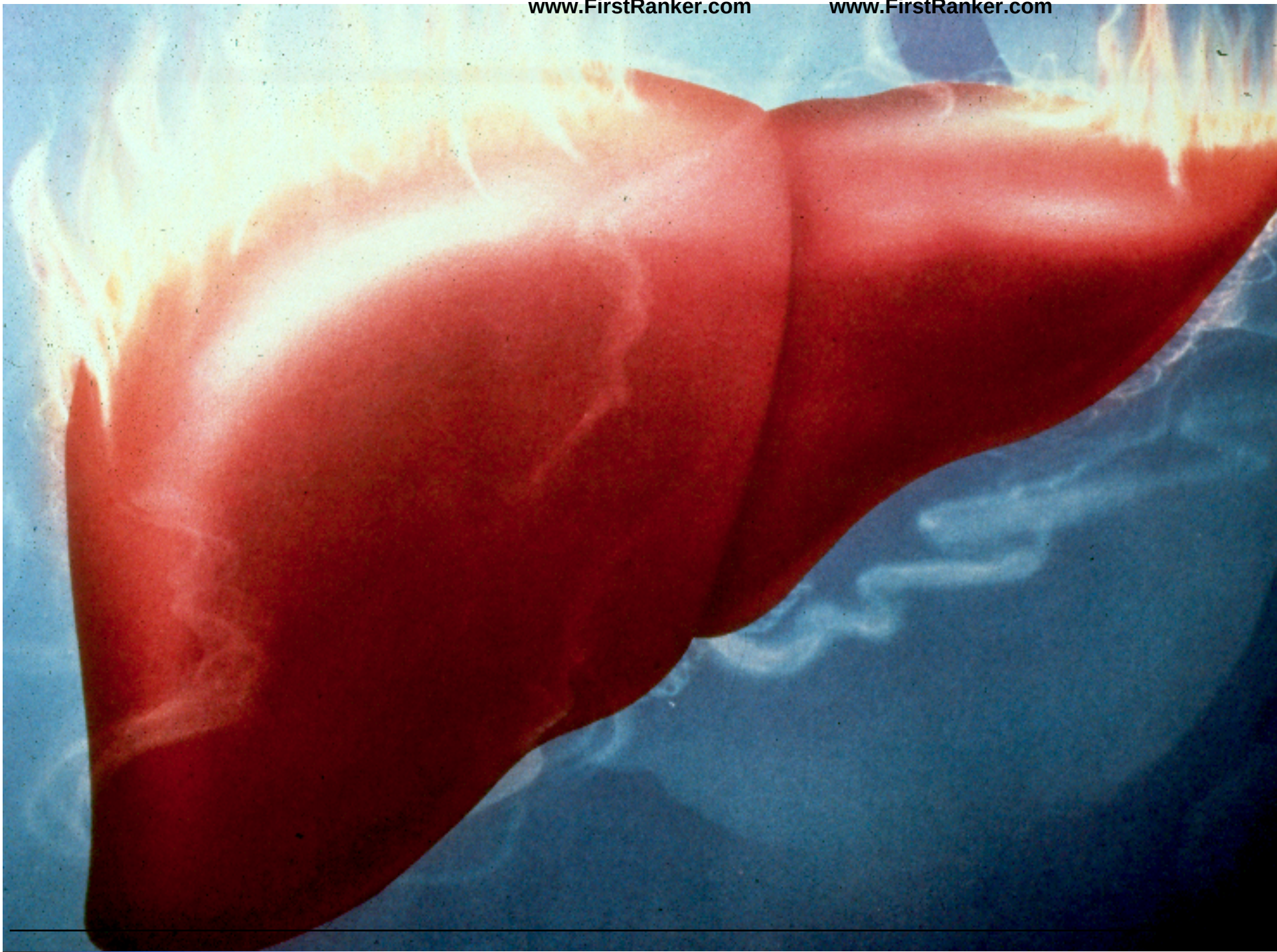


ACETAMINOPHEN



Acetaminophen (1)

- ☠ Toxic metabolite causes liver injury
 - Saturation of glucuronidation and sulfonation pathways (major metabolic pathways)
 - Metabolism shifts to minor pathway (cytochrome P450)
 - Cytochrome P450 metabolism requires glutathione, which depletes rapidly
 - Toxic metabolite NAPQI (n-acetyl-p-benzoquinoneimine) accumulates
 - Is a free radical
 - Direct hepatocellular toxicity



Acetaminophen (2)

☠️ Four clinical stages

Stage I GI symptoms (N/V)

Stage II GI symptoms resolve

Hepatic and renal dysfunction begins

Stage III Peak LFTs, GI symptoms return

Stage IV Recovery phase

STAGE 1	• Nausea & vomiting, abdominal pain, sweating, general discomfort, pale color • Liver function tests may be normal	DAYS 0-1
STAGE 2	• Liver injury develops • Upper right quadrant pain • Rise in liver function tests (ALT, AST, bilirubin, INR)	DAYS 1-3
STAGE 3	• Hepatotoxicity peaks • Rapid & severe hepatic failure • Encephalopathy & hypoglycemia • Glucose, lactate, & phosphate abnormalities • Coma & death	DAYS 3-5
STAGE 4	• Recovery stage for those who survive stage 3	DAYS 5-8

Acetaminophen (3)

☠ Toxic ingestion: 140 mg/kg (7-10 g in adults)

☠ 4 hour level >140: Potentially toxic

☠ N-acetylcysteine (NAC)

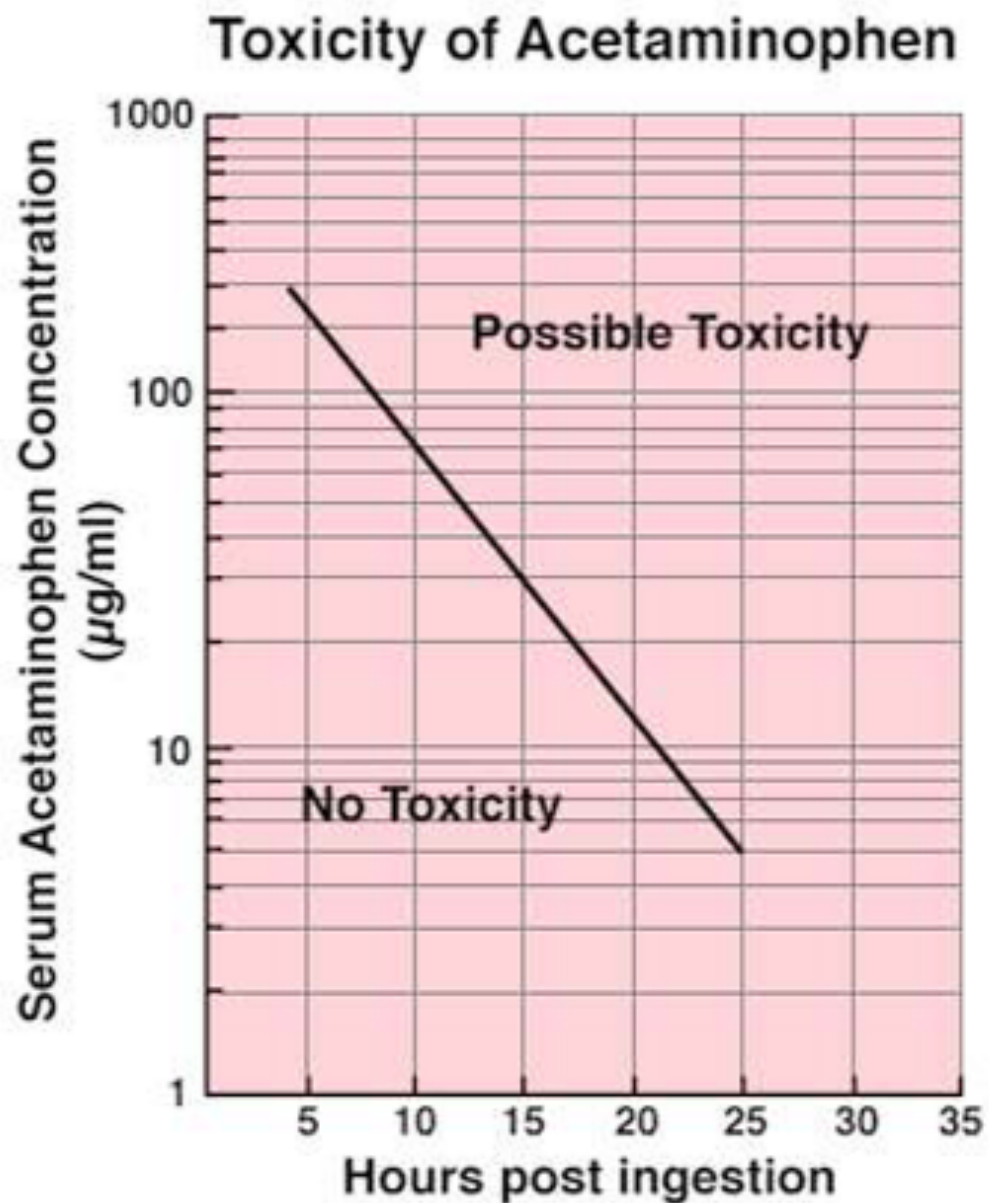
- Prevents binding of NAPQI to hepatic macromolecules
- May also reduce NAPQI back to acetaminophen

NAC provides a cofactor needed to make inert metabolites of APAP

Lack of this cofactor results in the production of hepatotoxic intermediary metabolites

- Oral and IV preparations available
- Safe in pregnancy
- Charcoal does not inhibit effectiveness
- Still indicated in late presentations >24hrs

☠ Hepatotoxicity is rare in children



Alcohol (1) - Ethanol

- ☠ Ethanol intoxication should not give acidosis
- ☠ Search for other causes of altered MS (CNS)

Withdrawal

- ☠ “The shakes” (6-8 hours) / hallucinations (visual)
- ☠ Alcohol withdrawal seizures (6-48 hours)
 - Treat with benzodiazepines; no role for phenytoin
- ☠ Delirium tremens: **Confusion**, fever, tachycardia
- ☠ Treatment: Fluids, thiamine (B1), multivitamins, Mg^{++} , folic acid, benzodiazepines

Wernicke's encephalopathy: Oculomotor crisis
CN VI palsy (lateral rectus), nystagmus, ataxia, global
confusion

Korsakoff's psychosis: Retrograde amnesia, confabulation



Alcohol (2) - Methanol

- ☠ Paint thinner, window washer solvent, wood alcohol, gas tank additive
- ☠ Anion gap acidosis, increased osmolal gap
- ☠ Disc hyperemia (visual symptoms common)
- ☠ Methanol itself is nontoxic but alcohol dehydrogenase forms the toxic metabolite
 - Formaldehyde
 - Formaldehyde is then broken down by formaldehyde dehydrogenase into another toxin = formic acid
- ☠ Symptoms (often delayed 12-18hrs): Seizures, respiratory failure, N/V, pancreatitis, visual changes or blindness

Alcohol (3) - Methanol

Treatment

- Fomepizole (4-methylpyrazole) (Antizol)
 - An alcohol dehydrogenase inhibitor
 - Allows methanol to be excreted intact via the kidneys without being broken down into its toxic metabolites
- Ethanol
 - Occupies alcohol dehydrogenase to limit breakdown of methanol into its toxic metabolites
 - Ethanol is preferentially metabolized by alcohol dehydrogenase over methanol and ethylene glycol
- Dialysis and bicarbonate if severe acidosis refractory to 4-MP or ethanol therapy

Alcohol (4) - Ethylene Glycol

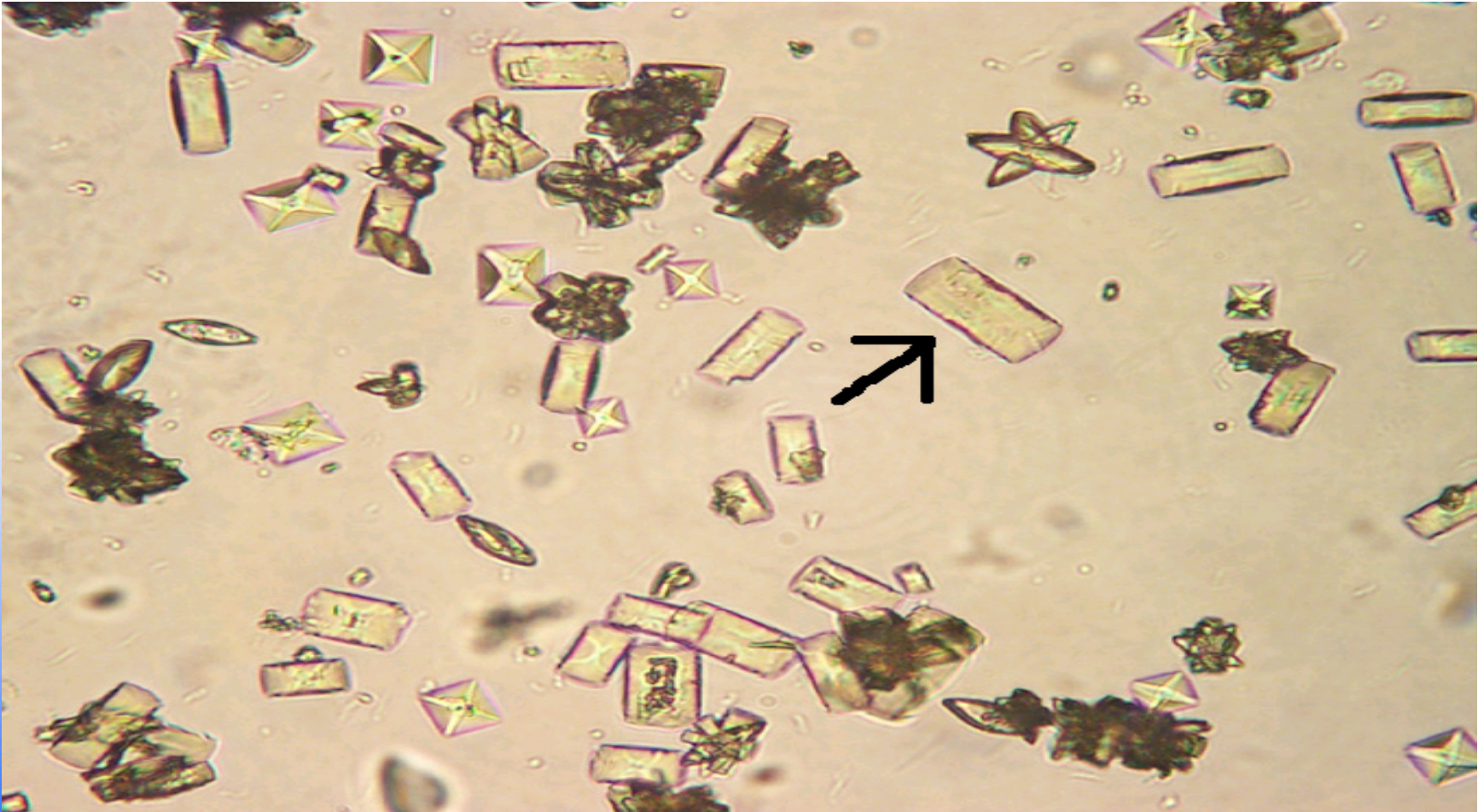
- ☠ Antifreeze, paint, solvents / has a sweet taste
- ☠ CNS depression, intoxication (without alcohol odor), anion gap acidosis, anuria, ↑ osmolal gap
- ☠ Metabolized by alcohol dehydrogenase to the toxic intermediate glycoaldehyde which is then broken down to:
 - Oxalic acid (precipitates in urine and blood with calcium to form calcium oxalate crystals)
 - Glycolic acid (causes profound acidosis)
- ☠ Renal failure, hematuria, hypocalcemia

Alcohol (4) - Ethylene Glycol

Treatment:

- Fomepizole to inhibit the activity of alcohol dehydrogenase, or IV alcohol to preferentially consume alcohol dehydrogenase
 - Treatment allows the renal excretion of intact ethylene glycol
- Requires supplemental pyridoxine and thiamine (both consumed in metabolism of ethylene glycol)
- Bicarbonate for acidosis
- Dialysis if severe acidosis or high blood levels

CALCIUM OXALATE CRYSTALS



4-Methylpyrazole (ANTIZOL)



TOXIC ALCOHOL THERAPY



Alcohol (5) - Isopropyl Alcohol

- ☠ Rubbing alcohol
- ☠ CNS depression greater than ethanol
- ☠ Metabolism: In liver > 50% metabolized to acetone; renal & pulmonary excrete unchanged isopropyl alcohol
- ☠ Hemorrhagic gastritis, pulmonary edema, hypoglycemia
- ☠ Severe hypotension
- ☠ Supportive care, don't give alcohol. Hemodialysis (if severe)

Ketosis but no acidosis / normal anion gap / increased osmolal gap

**Increasing alcohol toxicities:
ethanol < isopropyl alcohol <
ethylene glycol < methanol**

SYMPATHOMIMETICS



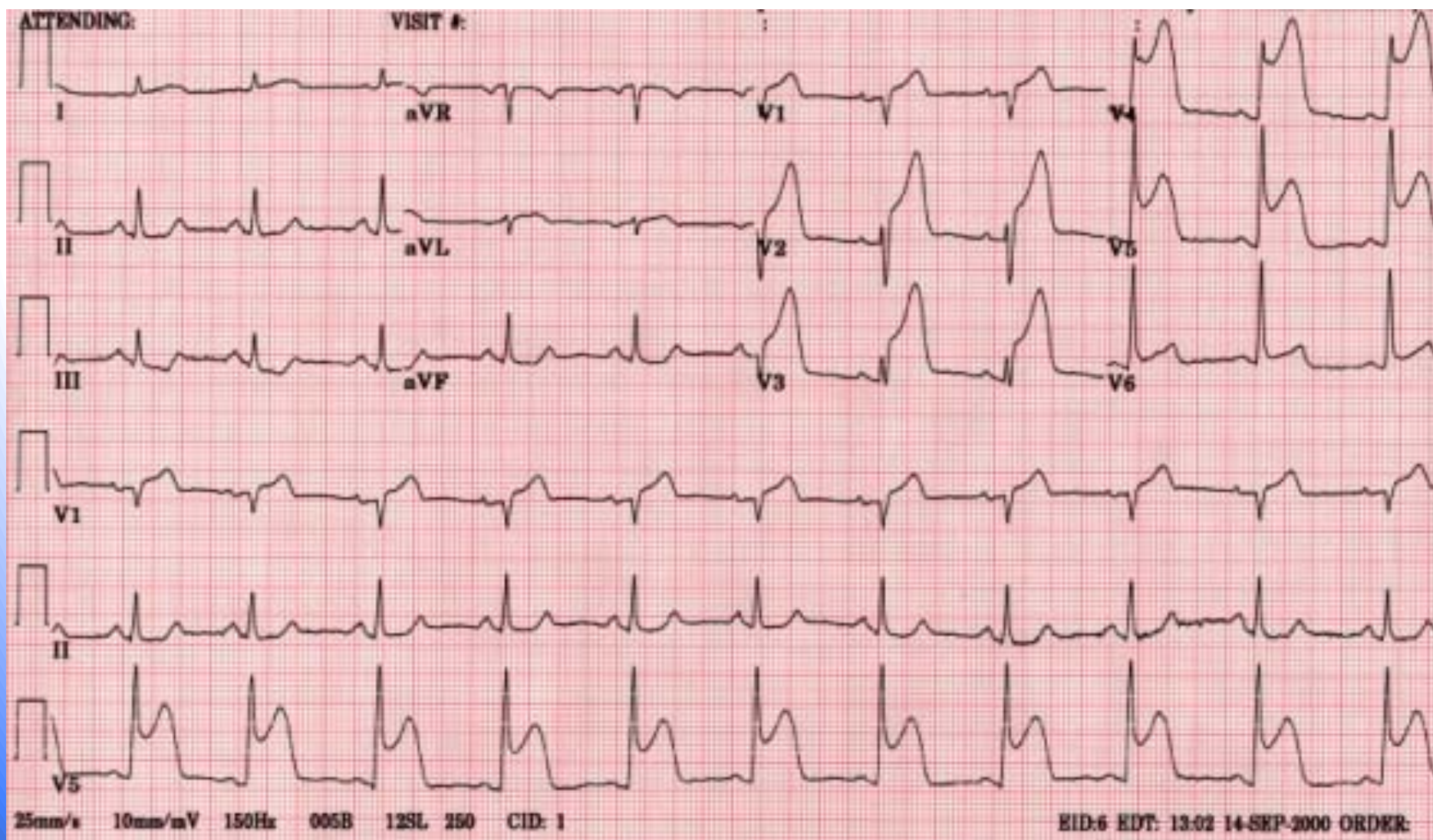
ECSTASY (MDMA)



Amphetamines / Cocaine (1)

- ☠ Cause sympathomimetic syndrome
 - Hypertension, tachycardia, seizures, dilated pupils, hyperthermia, agitation, diaphoresis
- ☠ Treatment: Benzodiazepines, decrease stimulation (dim room), cooling
- ☠ Beta blockers are contraindicated (cause unopposed alpha stimulation: hypertension, ischemia, vasoconstriction)

Cocaine: Abruptio placentae, prematurity, status epilepticus, strokes, MI, rhabdomyolysis



Amphetamines / Cocaine (2)

- ☠ Cocaine is metabolized by plasma pseudocholinesterase and liver enzymes → metabolites excreted in urine
- ☠ Psychiatric complaints are common

Chest pain: Usually noncardiac - consider PE, endocarditis, pneumothorax, pneumonia

- ☠ Body packer usually has positive plain film
- ☠ Formication: “Cocaine bugs”
- ☠ Designer agents: MDA, Ecstasy, MDMA, etc.

Antabuse (Disulfiram) Reaction



Alcohol + metronidazole (Flagyl)

- Flushed face, neck, trunk
- Marked diaphoresis
- Nausea, vomiting, headache
- Severe
 - Hypotension
 - Seizures
 - Dysrhythmias



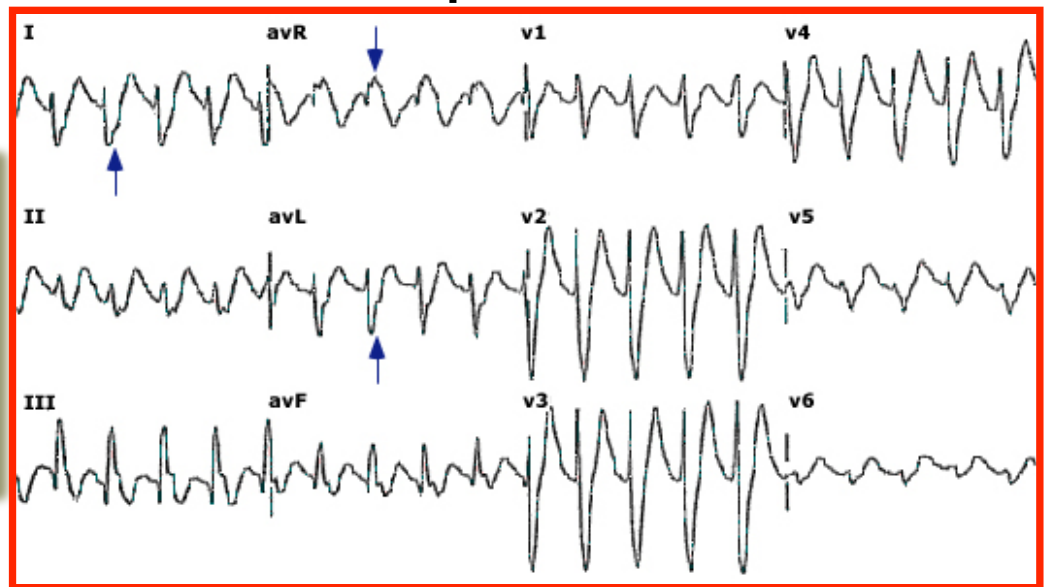
Anticholinergics / Antihistamines (1)

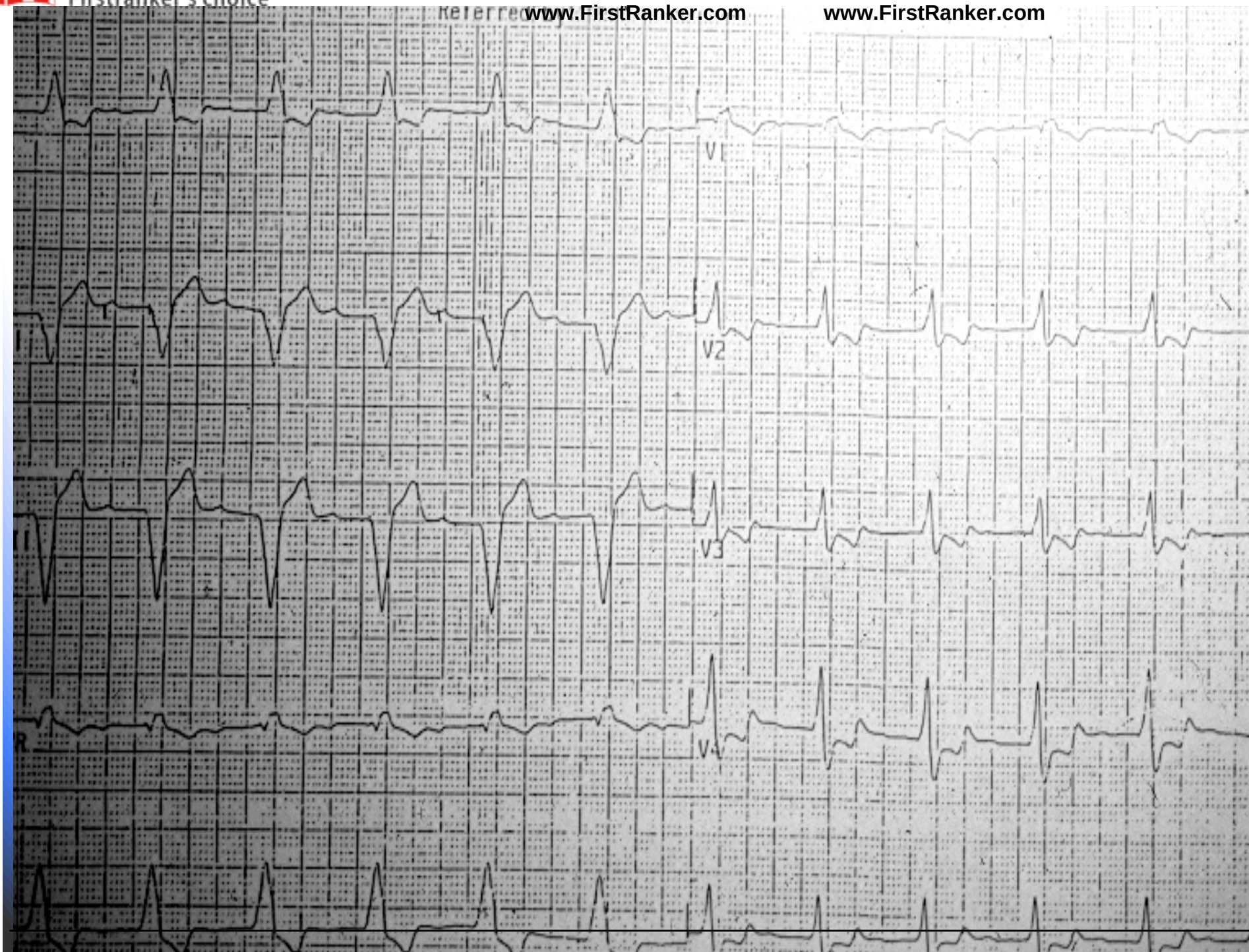
☠ Atropine, scopolamine, diphenhydramine, TCAs

☠ Tachycardia, dryness, ileus, urinary retention, dilated pupils, fever

☠ Treat agitation with benzodiazepines; avoid phenothiazines

**Wide QRS >100 msec?
Terminal R wave axis
rightward?
Consider TCA**





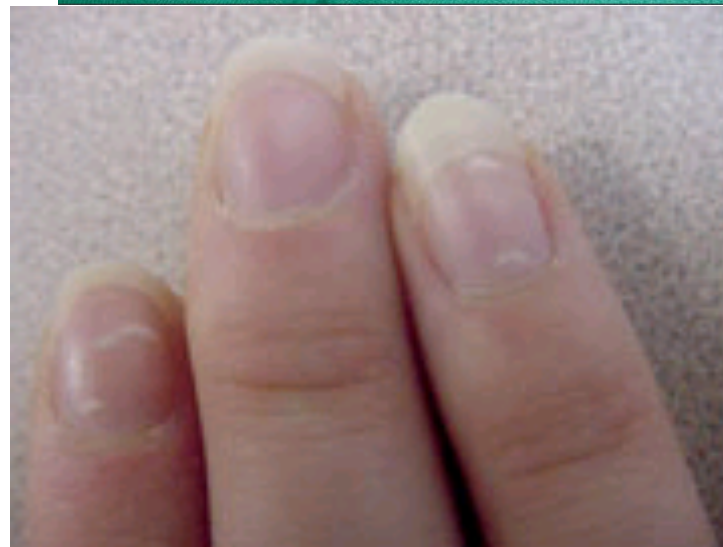
Anticholinergics / Antihistamines (2)

Physostigmine

- Inhibits acetylcholinesterase, increases acetylcholine
- Not first-line drug (benzodiazepines preferred)
- Consider in: Seizures, coma, arrhythmia, agitation
- **Contraindicated in TCA** (may cause asystole)

Arsenic

- ☠ Common heavy metal exposure
- ☠ Wood preservatives, weed killers, pesticides
- ☠ Gastroenteritis, hypotension, muscle twitches, delirium, hepatic and renal failure, death
- ☠ Diagnosis: 24 urine
- ☠ Treatment: Chelation (BAL, DMSA)
- ☠ Chronic effects: Anemia, metallic taste, neuropathy, horizontal Mees' lines in nails (nonspecific – also seen with thallium, other heavy metals and renal failure), alopecia



Barbiturates

☠ Short-acting

- Pentobarbital, secobarbital
- High lipid solubility
- Hepatic degradation

☠ Long-acting

- Phenobarbital
- Renal excretion, dialyzable

☠ Miosis, hypotension, CNS depression

☠ **Cutaneous bullae** (bad prognosis)

☠ Treatment: Lavage, charcoal, urine
alkalinization: long-acting agents, hemodialysis



Benzodiazepines

- ☠ Respiratory depression, ataxia, hypotension, hypothermia, coma
- ☠ Treatment: Supportive

Flumazenil may cause intractable seizures:
chronic users, seizure disorders, seizure-causing
co-ingestants, increased ICP



Beta Blockers

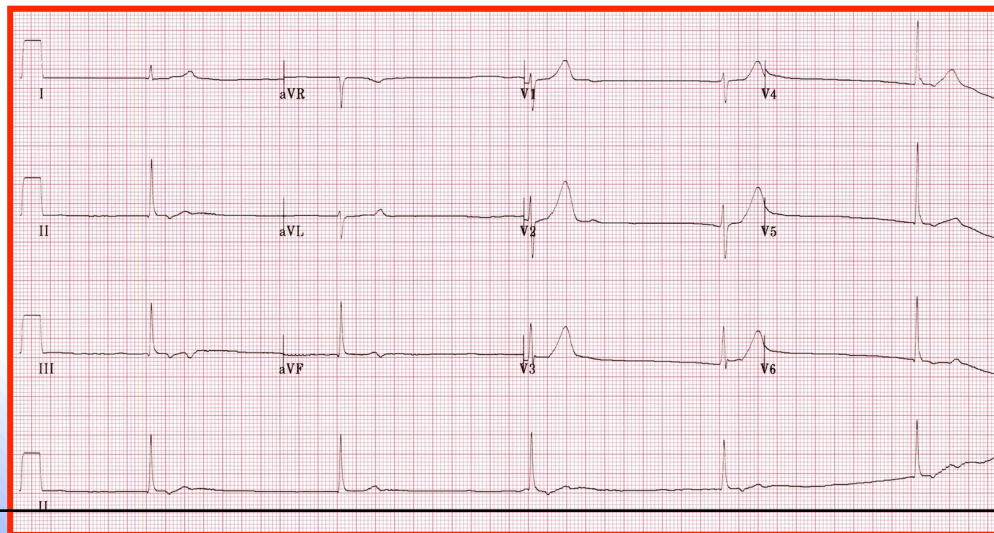
☠ Hypotension, bradycardia, AV block

☠ Antidotes:

- Glucagon 5-10mg IV, (avoid phenol diluent)
- Insulin + dextrose therapy
- Intralipids

☠ Other : Atropine, pressors, pacemaker

☠ Timolol eye drops can cause systemic toxicity



Calcium Channel Antagonists

- ☠ Most common: Verapamil, diltiazem, nifedipine
- ☠ Bradycardia, hypotension, conduction delay
- ☠ Antidotes
 - Calcium gluconate
 - Glucagon
 - Insulin + glucose rescue therapy
 - Intralipids
- ☠ Other treatments: Fluids, pacemaker, pressors

Leading cause of death from cardiovascular drugs

Carbamazepine (Tegretol)

Classic triad

- Dizziness
- Ataxia
- Nystagmus

Mild symptoms

- Ataxia
- Drowsiness
- Slurred speech

• Severe symptoms

- Coma
- Seizures
- Arrhythmia

• Treatment

- Multidose charcoal
- Hemoperfusion
- NaHCO_3 for $\uparrow$ QRS



Carbon Monoxide (CO) (1)

- ☠ Leading cause of toxic death
- ☠ Binds Hgb, cytochrome A3, myoglobin
- ☠ COHgb affinity for O₂ is 250 times more than Hgb
- ☠ Shifts oxyhemoglobin dissociation curve to left (inhibits release of oxygen from hemoglobin)
- ☠ Headache, nausea, confusion, coma, seizures, no cyanosis (may be mistaken for “flu”)
- ☠ Also suspect cyanide toxicity with smoke exposure and profound acidosis

Levels do not always correlate with toxicity

Carbon Monoxide (CO) (2)

- ☠ CO crosses the placenta and fetal Hgb has high affinity for CO
- ☠ Diagnosis: COHgb level (pulse ox not reliable)
- ☠ Half life of CO
 - 320 min (air)
 - 90 min (100% O₂)
 - 20 min (HBO)
- ☠ Delayed neuropsychiatric syndrome
- ☠ HBO *may* prevent delayed CNS injury
- ☠ Treatment: 100% oxygen, HBO (controversial)





Carbon Monoxide (CO) and ABGs

Carboxyhemoglobinemia

- Pulse Ox is normal (oximeter mistakes carboxyhemoglobin for oxyhemoglobin)
- PaO₂ on ABG is normal (oxyhemoglobin is calculated from measurement of dissolved O₂, which is normal)

Key point:
Need to measure carboxyhemoglobin

Caustic Ingestions

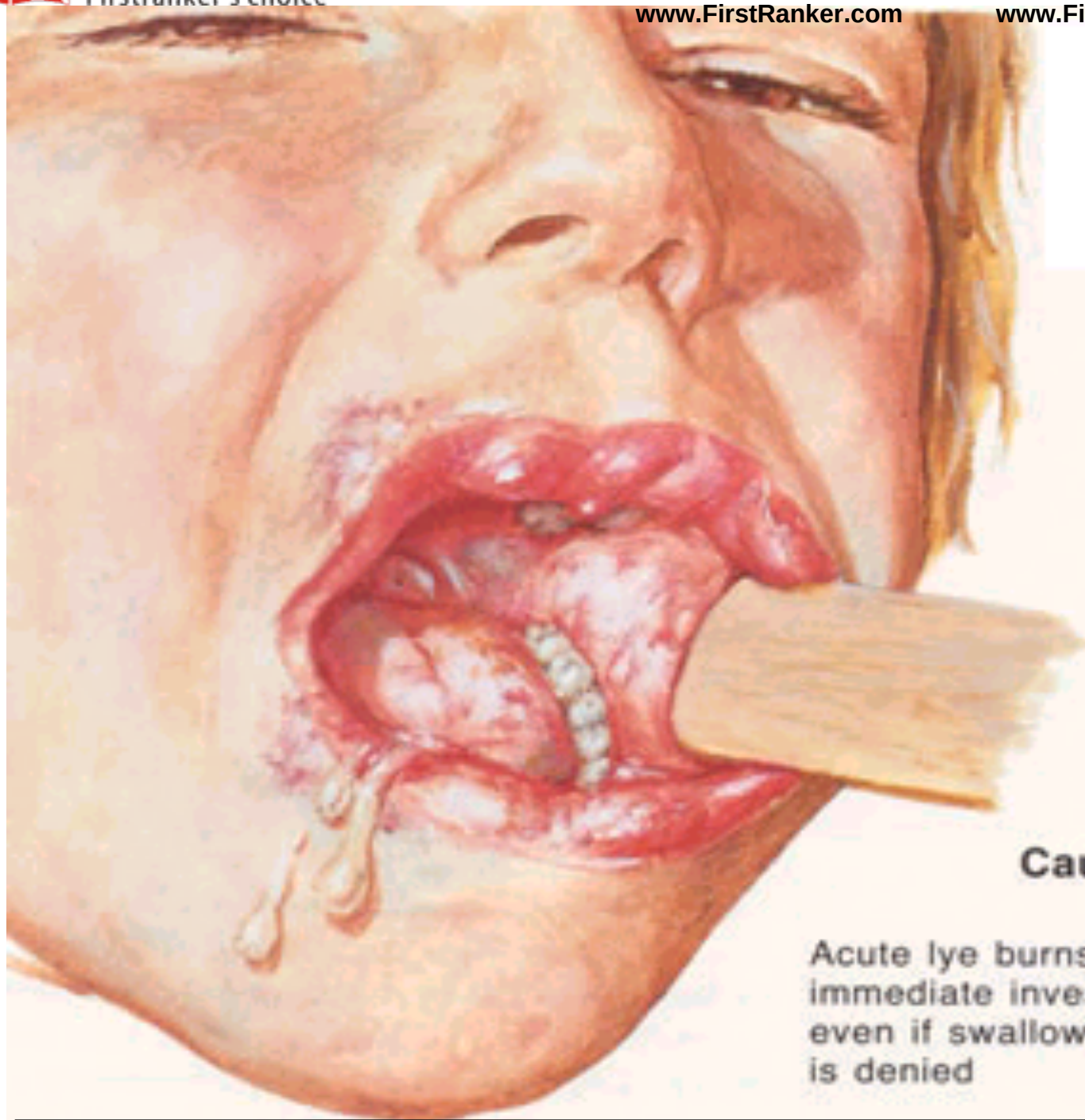
Acids

- Coagulation necrosis
- Eschar formation stops progression

Alkali

- More common
- Liquefaction necrosis facilitates progression
- Full thickness injury

Presence of oropharyngeal burns in alkali ingestion
is not predictive of esophageal injury



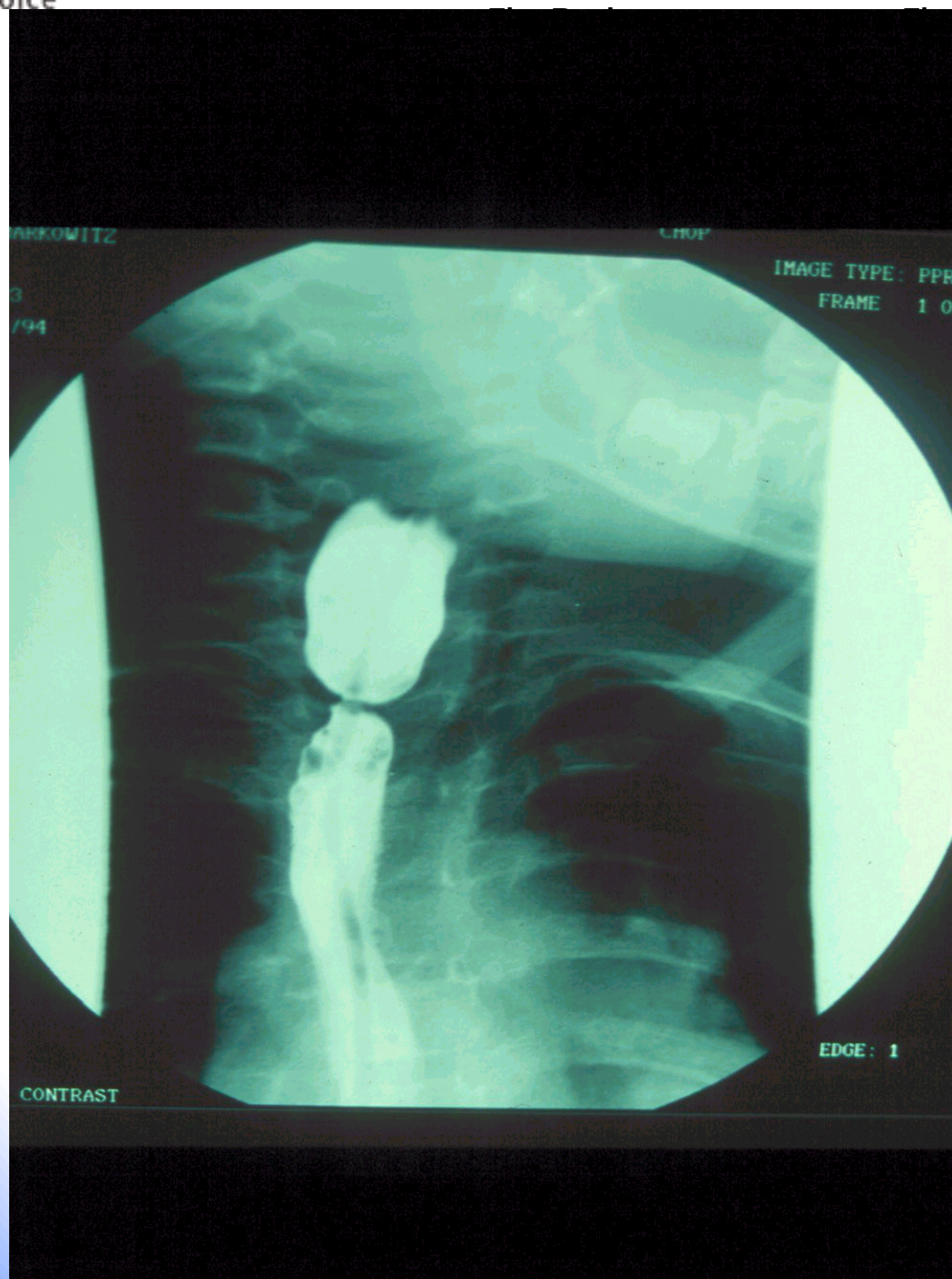
Caustic Poisoning

Acute lye burns in mouth demand immediate investigation and treatment even if swallowing of corrosives is denied

Caustic Injuries

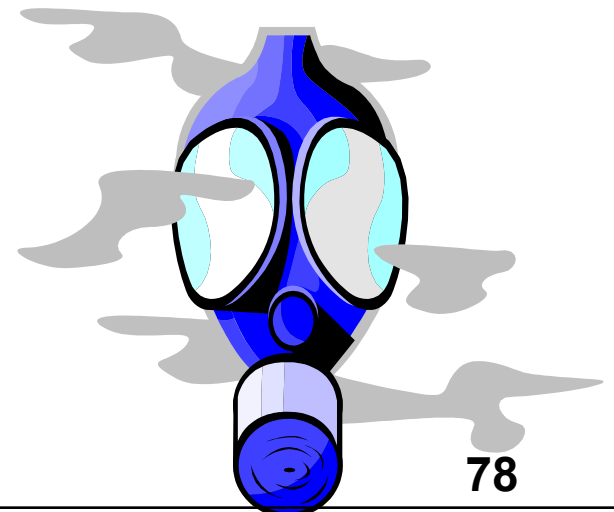
Treatment

- ☠ Charcoal and lavage are contraindicated
- ☠ Blind nasotracheal intubation contraindicated
- ☠ Hydrofluoric acid: Give milk, Mg citrate by NGT
- ☠ Solid alkali ingestion: Dilute with water or milk
- ☠ Other caustic ingestions: Dilution is contraindicated, as it may induce vomiting
- ☠ Upright CXR
- ☠ Endoscopy within first several hrs of ingestion



Chlorine

- ☠ Widely available (labs, paper manufacture, pool chemicals, cleaning products)
- ☠ Yellow-green gas, pungent odor
- ☠ Formation of acid and oxidants on moist membranes
- ☠ Immediate ocular and upper airway irritation
- ☠ Can lead to pulmonary edema in severe exposure
- ☠ Supportive care, humidified O₂



Clonidine

- ☠ Alpha-adrenergic agonist
- ☠ Central effects: Hypotension, bradycardia
- ☠ Peripheral effects: Hypertension followed by hypotension
- ☠ CNS: Lethargy, apnea, miosis, decreased reflexes
- ☠ Cardiac: Bradycardia, hypotension, AV block

**Can mimic opiate OD
coma, miosis, respiratory depression**

- ☠ Treatment: Fluids, atropine, pressors
- ☠ Can mimic opioid OD; Naloxone may be beneficial
- ☠ Withdrawal: Hypertension, tachycardia, anxiety, diaphoresis

Cyanide (1)

- ☠ Binds cytochromes, causes anaerobic metabolism and lactic acidosis
- ☠ Jewelers, chemical labs, apricot pits, smoke inhalation (burning plastic)
- ☠ Suspect in lab worker, suicide, coma with acidosis, use of artificial nail remover, ICU patient on nitroprusside
- ☠ Abdominal pain, nausea, coma, bradycardia, acidosis, **sudden CV collapse**
- ☠ Distinctive odor (bitter almonds)
- ☠ No cyanosis
- ☠ Normal PaO_2 and O_2 sat; acidosis

Cyanide (2)



Lab: Lactic acidosis (with anion gap)



Treatment goal is to create a controlled state of methemoglobinemia to compete for CN

— $\text{CN} + \text{metHgb} \rightarrow \text{cyanometHgb}$



Nitrites create metHgb that binds CN

— Amyl nitrite (inhaled)

— Sodium nitrite IV

— Sodium thiosulfate IV

— $\text{Sodium thiosulfate} + \text{CN} = \text{thiocyanate (renal excretion)}$



Hydroxocobalamin (Cyanokit) is a B12 precursor that combines with cyanide to form cyanocobalamin (B12) which is cleared renally 81

Cyanide Antidotes



Digitalis (1)

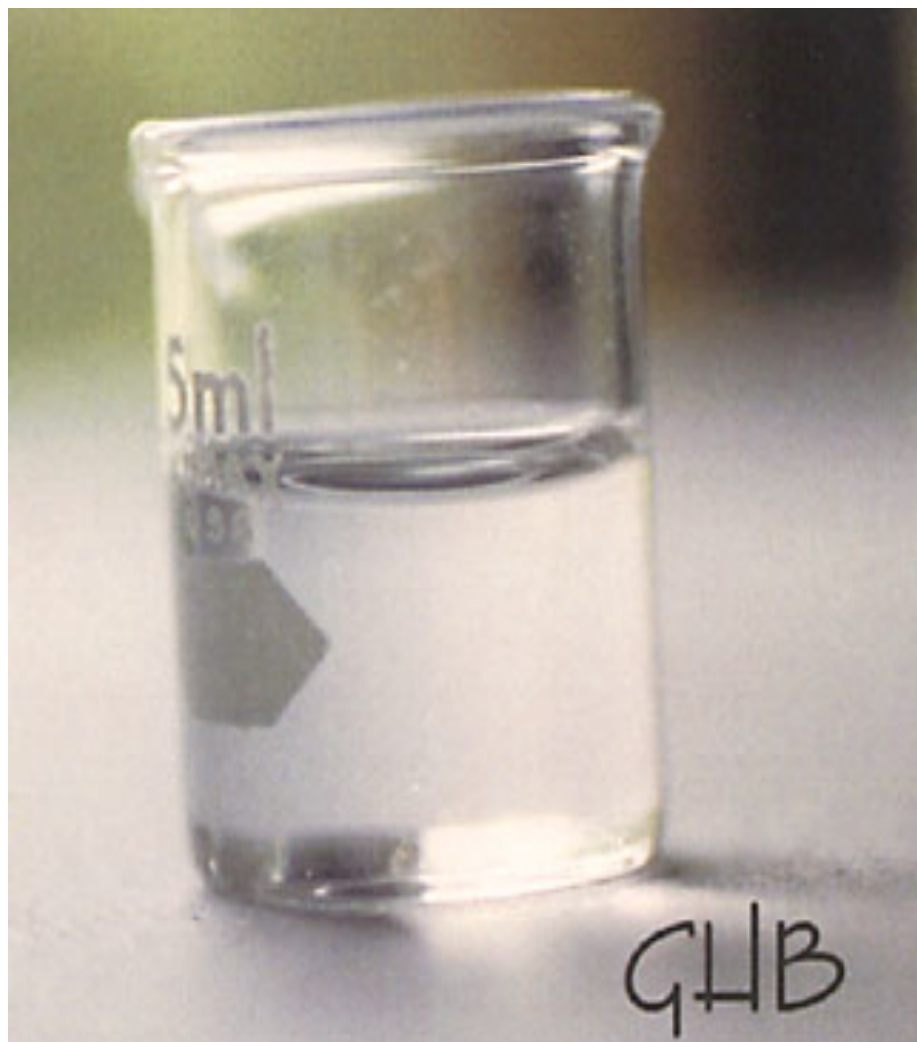
- ☠ Inhibits Na-K ATPase pump
- ☠ K^+ leaks extracellularly; Na^+ and Ca^{++} leak intracellularly
- ☠ Hyperkalemia, AV blocks, arrhythmias
- ☠ Antidote: Digibind (Fab fragments)
 - Each vial binds 0.5 mg digoxin
 - 20 vials should treat any life-threatening toxicity
 - Acute ingestion: Start with 10 vials if dig level is unknown and life threatening dysrhythmia
 - Chronic toxicity: Start with 6 vials



Digitalis (2)

- ☠ Class 1A antiarrhythmics are contraindicated
- ☠ Low K^+ , Low Mg, High Ca increases toxicity
- ☠ High K^+ indicates severe OD
- ☠ Pathognomonic: PAT with AV block
- ☠ Calcium administration contraindicated if digoxin toxicity suspected or confirmed

**Most common dig toxic
dysrhythmia is PVCs**



Gamma Hydroxybutyric Acid (GHB)

- ☠️ A naturally occurring analog of the neuroinhibitor gamma-aminobutyric acid (GABA)
- ☠️ Street names: Grievous bodily harm, Georgia home boy, Liquid X
- ☠️ “Date rape” drug / “rave” drug
- ☠️ Typically – white male, mid- to late 20s, nightclub, present to ER after midnight
- ☠️ Also used by body builders



Gamma Hydroxybutyric Acid (GHB)

- ☠ Not routinely detected on drug screens
- ☠ CNS depressant, rapid onset, lowered gag reflex, severe respiratory depression
- ☠ Treatment: Airway control, no role for naloxone, flumazenil, or physostigmine
- ☠ Usual **rapid resolution within 8 hours**
- ☠ If co-ingestion of ETOH observe longer
- ☠ Withdrawal from chronic use is similar to ethanol or sedative/hypnotic withdrawal

Hallucinogens

- ☠ LSD, mushrooms, mescaline, morning glory seeds, designer drugs
- ☠ Serotonin antagonism
- ☠ Psychosis, anxiety
- ☠ Sympathomimetic signs
 - Tachycardia
 - Hyperthermia
 - Dilated pupils
- ☠ Treatment: Sedation, benzodiazepines, haloperidol (Haldol), minimal stimulation
- ☠ Amanita mushrooms account for most deaths

TLSD

Digital Surround Reality

Hydrocarbons (1)

- ☠ Increased toxicity with
 - Halogenated hydrocarbons or other toxic additives
 - Low viscosity
 - High volatility
 - Low surface tension
- ☠ Aspiration pneumonitis (coughing, tachypnea)
 - Do not induce vomiting

**Exposure (sniffing, huffing) can sensitize myocardium
→ dysrhythmias (catecholamines contraindicated)**

- ☠ Lavage if halogenated (carbon tet, PVC) or aromatic (benzene, toluene, xylene)
- ☠ CXR: Deceptively clear- delayed findings (4-6 hours)
- ☠ Steroids and prophylactic antibiotics are not indicated

Hydrocarbons (2)

☠ The general rule is NOT to empty the stomach, unless the hydrocarbon solution contains a toxic substance such as: (CHAMP)

Camphor

Halogenated (carbon tet, PVC, vinyl chloride)

Aromatic (benzene, toluene, xylene)

Metals (leaded gas)

Pesticides

☠ Kwell (lindane)

- Chlorinated hydrocarbon
- CNS toxicity, seizures
- Avoid in young children, pregnancy

Hydrogen Fluoride

- ☠ Used in glass etching
- ☠ One of the strongest inorganic acids
- ☠ Can cause deep cutaneous injury, systemic hypocalcemia
- ☠ **Skin may appear normal despite significant burn – causes significant delayed injury**
- ☠ Treatment
 - Calcium gluconate (infiltration, gel, or intra-arterial infusion) / avoid intradermal CaCl
 - Calcium binds with fluoride ion → stops pain
 - If pain returns, repeat treatment



Hydrogen Sulfide (H₂S)

- ☠ Oil refinery, hot asphalt, sewage, mines
- ☠ Rapid knockdown agent with rotten egg odor
- ☠ Inhibits cellular respiration, blocks cytochrome oxidase (leading to anaerobic metabolism)
- ☠ Has higher affinity for methemoglobin than cytochrome oxidase, increase elimination by production of methemoglobin
- ☠ Treatment: Remove from source, increase elimination using methylene blue in cyanide antidote kit to generate methemoglobin (do not use the thiosulfate)

Isoniazid (INH)

Overdose

- ↓ vitamin B6 (required for GABA synthesis)
- ↓ seizure threshold (can present in status)
- Coma
- Metabolic acidosis

 Consider in pediatric seizures (unintentional OD)

 Consider in populations likely to be on INH

 Little role for bicarbonate therapy in acute toxicity

Antidote: Pyridoxine (vitamin B6)

Iron (1)

Common cause of poisoning death in children

- ☠ Free circulating iron damages blood vessels
 - Direct GI corrosive effect
 - Inhibits aerobic metabolism
 - Vasomotor collapse, hepatorenal failure

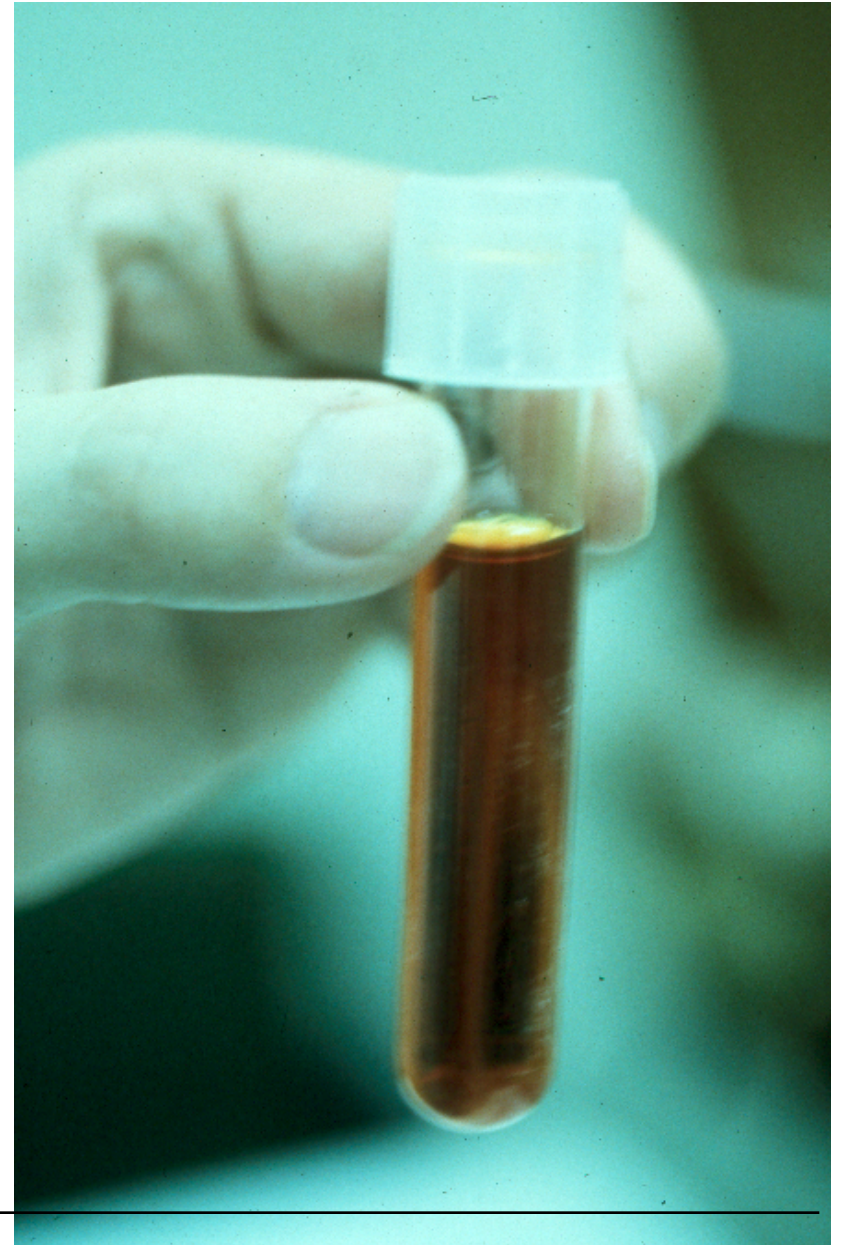
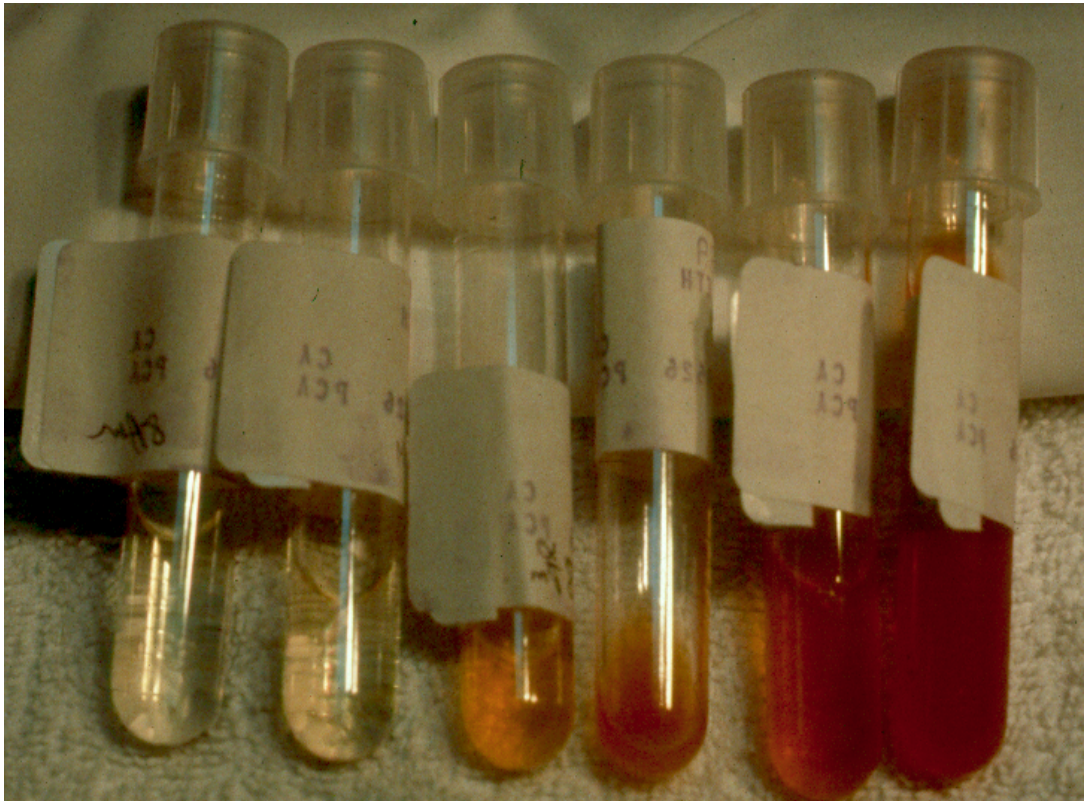
- ☠ Toxic dose is 30-40 mg/kg elemental iron
 - 0-6 hrs - Gastroenteritis, dehydration, shock
 - 6-48 hrs - Quiescent phase
 - 12-48 hrs - Acidosis, coma, hepatic injury
 - 2-6 weeks - Recovery, GI scarring, obstruction

Iron (2)

GI symptoms suggest significant exposure

- ☠ 4 hour serum Fe level (TIBC not reliable)
- ☠ Avoid bicarb lavage
- ☠ Charcoal doesn't bind Fe
- ☠ Labs suggestive of OD: ↑gap, ↑glucose, ↑WBC
- ☠ Liver has greatest injury due to portal circulation
- ☠ Abdominal X-ray: Bezoar
- ☠ Antidote: Whole bowel irrigation, deferoxamine (chelating agent)
- ☠ Deferoxamine indications
 - Severe GI symptoms, acidosis, shock
 - Serum iron > 500, significant history

VIN ROSE URINE



Lead (1)

- ☠ Common cause of chronic poisoning
- ☠ Suspect in children with headache, encephalopathy, anorexia, abdominal pain, paint exposure, lead lines
- ☠ Combines with sulfhydryl (SH) groups, interferes with enzyme activity
- ☠ CNS, GI tract, hematologic





Lead (2)

- ☠ Peripheral neuropathy (classic - wrist drop)
- ☠ Diagnosis: Serum level (>50 mcg/dL is severe), basophilic stippling on blood smear



- ☠ Treatment: Chelation (BAL, DMSA, EDTA)
- ☠ If KUB shows lead paint chips, whole bowel irrigation



Lithium (1)

- ☠ Most patients under long-term treatment develop toxicity at some point
- ☠ Renal elimination
- ☠ Hyponatremia and hypovolemia increase toxicity
- ☠ In acute ingestion, toxicity may be delayed (lags rise in serum levels).
- ☠ May be toxic at low levels (chronic ingestion)
- ☠ Serum levels do not predict CNS levels
- ☠ Clinical: Tremor, vomiting, diarrhea, confusion, slurred speech, coma, arrhythmias
- ☠ Seizures: Use benzodiazepines, phenobarbital
 - Dilantin decreases excretion of Li^+

Lithium (2)

Clinical status more important than Li^+ level

- ☠ Treatment: Hold drug, no diuretics, lavage, Kayexalate
- ☠ Aggressive hydration with normal saline
- ☠ Hemodialysis (severely toxic patient)
 - Best based on clinical indications: : Coma, seizures, myoclonus, hemodynamic collapse
 - Consider if acute ingestion level $> 3.5 \text{ mEq/L}$
 - High risk in renal failure, rapidly increasing levels, sustained release ingestion

Mercury

- ☠ Toxicity depends on dose, form, and route of exposure (elemental, organic, inorganic)
- ☠ CNS, GI, renal toxicity
- ☠ Metallic mercury is benign if ingested, but very dangerous when inhaled or injected
- ☠ Diagnosis: 24 hour urine collection
- ☠ Treatment: Lavage, chelation (BAL, succimer)

**Mercury from thermometer is inert
(not absorbed by GI tract)**

Monamine Oxidase Inhibitors (MAOI)

- ☠ Block degradation of catecholamines (norepi, epi, serotonin)
- ☠ Delayed symptoms 6-12hrs post-ingestion: Headache, HTN, tachycardia
- ☠ Hypertensive crisis: Amphetamines, cheese, wine, fava beans (tyramine)

**Meperidine and dextromethorphan are
contraindicated
with MAOI (can cause severe hyperthermia)**

Methemoglobinemia (1)

- ☠ Results from the oxidation of normal Fe^{++} state to Fe^{+++}
- ☠ Methemoglobin is unable to bind oxygen
- ☠ Causes: Nitrites, nitrates, benzocaine, dapson, phenazopyridine (Pyridium)
- ☠ Presents often with vague symptoms
- ☠ May present with signs and symptoms of hypoxia, including cyanosis at metHgb levels $>15\%$

Methemoglobinemia (2)

- ☠ Cyanosis unresponsive to O_2
- ☠ Chocolate brown blood (also seen with SO_4Hgb)
- ☠ Pulse ox unreliable, arterial PaO_2 normal
- ☠ Definitive identification with co-oximetry

**Cyanosis reversible with methylene blue
(Reduces Fe^{+++} to Fe^{++})**

**Methylene blue is contraindicated in G6PD
deficiency**

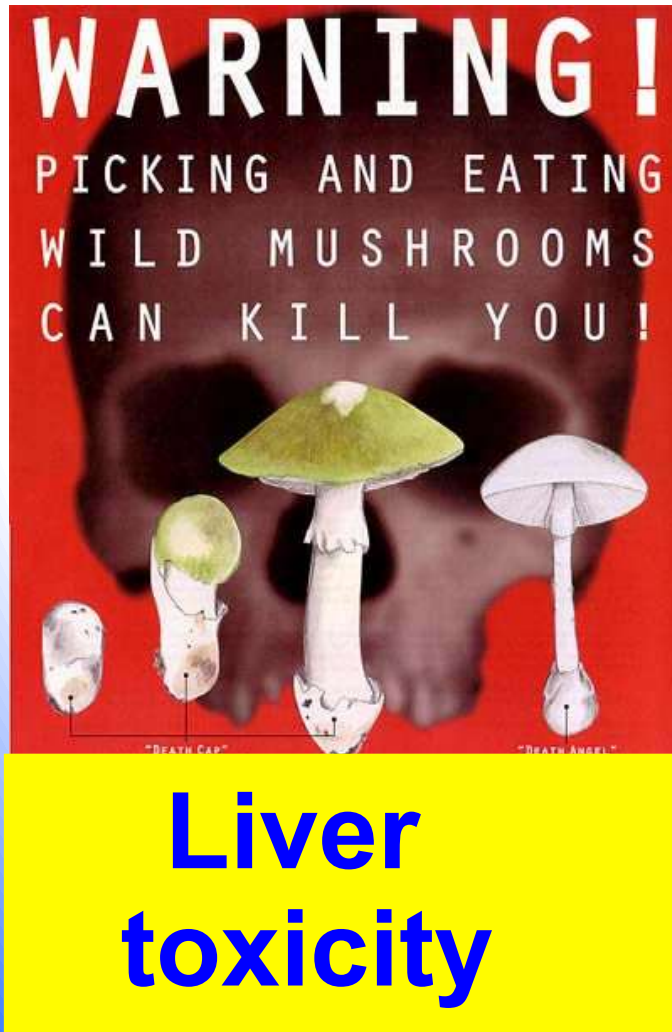
METHEMOGLOBINEMIA



Mushrooms

- ☠ If lethal, usually Amanita species
- ☠ Nausea, vomiting, diarrhea, hepatic failure, renal failure
- ☠ **Time from ingestion to symptom onset helps determine risk of significant toxicity**
 - Symptoms within 2 hours of ingestion indicate benign course if only one type of mushroom ingested
 - Symptoms delayed more than 6 hours indicate a toxic ingestion with risk of hepatic and renal failure

Amanita Phalloides “Death Cap”



Mushrooms

Amanita Phalloides

- ☠ Nausea, vomiting and diarrhea 12 hours post-ingestion, then latent period
- ☠ GI symptoms return in 24-48 hours with liver necrosis and renal failure
- ☠ Toxin kills GI epithelial cells, hepatocytes
- ☠ Supportive care, liver transplantation

Amanita muscaria



Mushrooms

Amanita Muscaria / Pantherina

- ☠ Toxin is GABA agonist
- ☠ Mental status changes
(delirium, hallucinations,
seizures)
- ☠ No hepatic toxicity



Psilocybin

- ☠ Hallucinations, euphoria,
nausea
- ☠ Supportive care



False Morels (Gyromitridin)

Good

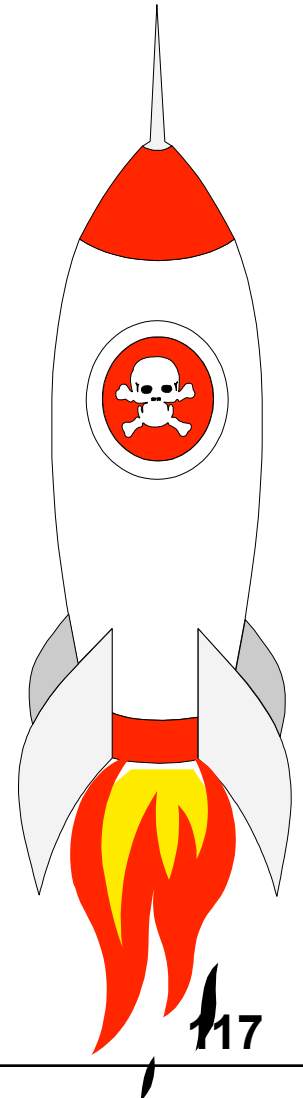


Bad



MUSHROOMS (Gyromitrin)

- ☠ Species: Gyromitrin (false morels)
- ☠ Toxin: Monomethylhydrazine (rocket fuel... really!)
- ☠ Clinical effects: Gastroenteritis (delayed), headache and seizures
- ☠ Treatment: Pyridoxine (Vitamin B6)



Neuroleptics / Phenothiazines

Clinical effects

- Dystonic (extrapyramidal) reactions
 - Tremor, torticollis, rigidity, facial grimacing, dysphagia, oculogyric crisis
- Akathesia (inability to sit still)
- Tardive dyskinesia (involuntary lip and tongue movements)
- Anticholinergic symptoms
- Quinidine-like, anticholinergic, peripheral alpha-blockade, antihistaminic , prolonged QT, cardiac dysrhythmias
- Other: Heat stroke, hypotension, sedation, psychosis, urinary retention, seizures

 Treatment: Benzotropine (Cogentin), diphenhydramine (Benadryl), benzodiazepines, lavage, bicarbonate

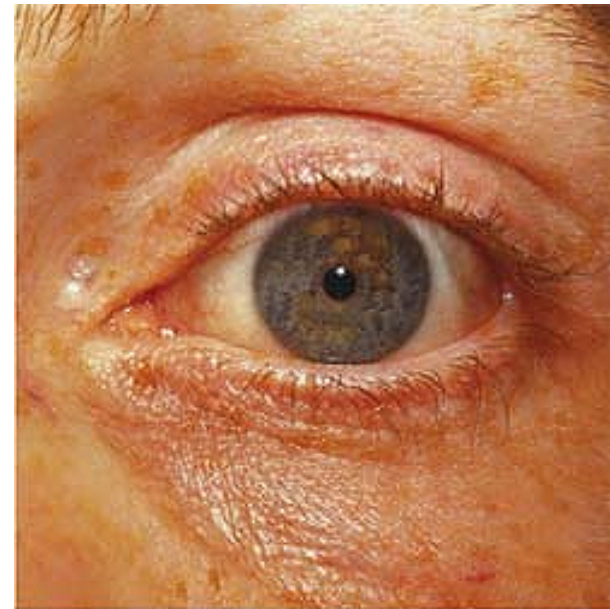
Neuroleptics / Phenothiazines

- ☠ Neuroleptic malignant syndrome (NMS)
- Rare, not dose-related (idiosyncratic)
 - **Slow onset** (hours to days)
 - Altered mental status, severe hyperthermia, **lead-pipe rigidity**, opisthotonos, hypertension, tachycardia
 - Elevated creatine kinase
 - Treatment: Fever control, benzodiazepines, dantrolene, paralysis, bromocriptine



Opioids

- ☠ Coma, apnea, pinpoint pupils (hypoxia can cause dilated pupils), hypotension
- ☠ Can cause noncardiogenic pulmonary edema
- ☠ Naloxone: Duration of action 30-60 minutes
- ☠ Diphenoxylate (Lomotil): slow onset of symptoms
- ☠ Increased dose of naloxone
 - Propoxyphene
 - Methadone
 - Fentanyl
 - Codeine
 - Pentazocine



Naloxone

- ☠ Dose: 0.2-2mg IVP
- ☠ Reflexive large doses discouraged
 - Will precipitate withdrawal
 - Smaller doses, titrating to effect preferred
- ☠ Continuous drip may be required
- ☠ Other routes of administration
 - IM, IO, SC, ET, Neb



Oral Hypoglycemic Agents (1)

- ☠️ Sulfonylureas cause prolonged hypoglycemia
 - Glyburide (Micronase), glipizide (Glucotrol), glimepiride (Amaryl), chlorpropamide (Diabinese)
 - Increase release of insulin from pancreatic cells
 - Risk factors for toxicity: Extremes of age, poor nutrition, EtOH, hepatic and renal insufficiency, beta blockers
 - Particularly dangerous in children
- ☠️ Non-sulfonylureas do NOT cause hypoglycemia
 - Metformin [Glucophage], acarbose [Precose], repaglinide [Prandin], rosiglitazone [Avandia], pioglitazone [Actos]

Oral Hypoglycemic Agents (2)

- ☠ Metformin can cause lactic acidosis
 - IV contrast will not induce lactic acidosis unless renal failure/insufficiency develops
- ☠ Admit symptomatic patients with sulfonylurea or metformin overdose
- ☠ Treatment of sulfonylurea overdose
 - Glucose bolus and drip
 - Glucagon (not used as empiric therapy)
 - Octreotide (refractory sulfonylureas overdose)
- ☠ Accidental ingestion without risk factors for toxicity can be observed in ED
 - If hypoglycemia does not occur, may discharge home (does not apply to pediatric patients)

Organophosphates

- ☠ Insecticides, pesticides
- ☠ Inhibit cholinesterases. Increase acetylcholine (cholinergic toxidrome)
- ☠ Muscarinic effects: pinpoint pupils, **SLUDGE + Killer "B"s – Bradycardia, Bronchospasm, Bronchorrhea**

S – Salivation

L – Lacrimation

U – Urination

D – Diarrhea

G – Gastrointestinal upset

E – Emesis



Organophosphates

- ☠ Nicotinic effects: Muscle weakness, fasciculations, respiratory failure
- ☠ Avoid use of succinylcholine with neuromuscular blockade
- ☠ Treatment: Atropine (until secretions dry)
- ☠ Pralidoxime (2-PAM)
 - Not indicated with carbamates



Phencyclidine (PCP)

☠ Findings

- Rotatory nystagmus
- Agitation, seizures
- Hyperthermia, rhabdomyolysis

☠ Treatment

- Hydration, cooling
- Benzodiazepines
- Haloperidol
- Ketamine toxicity similar
(without violent presentation)



Do not acidify urine

Phenytoin (Fosphenytoin)

☠ Side effects are related to dose and rate of infusion

- Nystagmus, ataxia, vomiting, slurred speech, dystonias, lethargy, coma
- Paradoxical seizures are rarely seen
- Too-rapid infusion can cause dysrhythmias and hypotension due to propylene glycol diluent

☠ Treatment: Lavage, multidose charcoal

☠ Hemodialysis and hemoperfusion are NOT useful

Poisonous Plants (1)

- ☠ Pits of apricot, cherry, peach and plum
 - Contain hydrocyanic acid (CN)
 - Cyanide toxicity, supportive care, cyanide kit

- ☠ Anticholinergic plants
 - Deadly nightshade (belladonna) berries
 - Henbane (seeds in pod)
 - Jimsonweed (dried leaves and seeds)

- ☠ Digitalis toxic plants (FOLLY)
 - Foxglove, Oleander, Lilly of the Valley



Jimsonweed *Datura stramonium*



Anticholinergic

Digitalis toxic plants (FOLLY)

Foxglove, Oleander, Lilly of the Valley



Lilly of the Valley



Digitalis Toxicity

Poisonous Plants (2)

 Holly (bright red berries)

- GI toxicity: N/V, abdominal cramps

 Philodendrons

- Common house plants
- Immediate pain or burning in mouth and throat
- GI irritation

 Poinsettia

- Vomiting, cramps, contact dermatitis



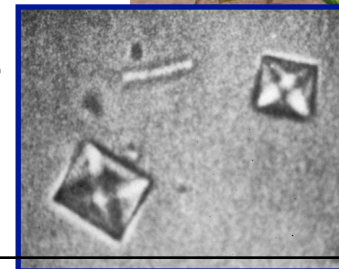
Poisonous Plants (3)

☠ Pokeweed (purple and black berries)

- Minor toxicity: Nausea, vomiting, cramps and diarrhea
- Severe toxicity: Respiratory depression, seizures, hemorrhage gastritis, hemolytic anemia

☠ Diffenbachia

- GI mucosal membrane irritation
- Calcium oxylate crystals



Pokeweed



Poisonous Plants (4)

Tobacco

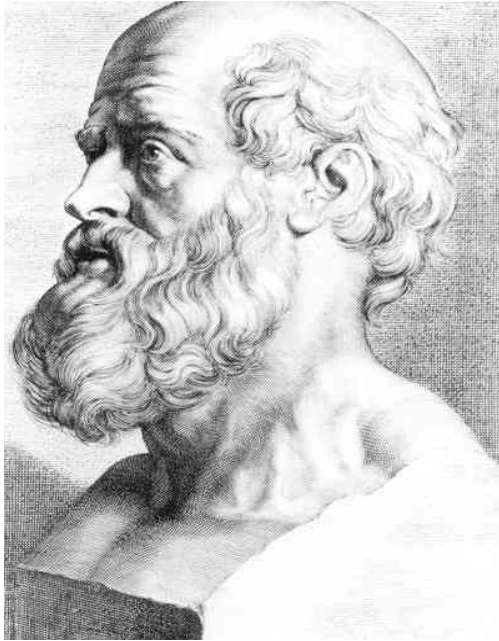
- Pediatric ingestion of cigarettes
- Dermal exposure in tobacco workers
- Cholinergic symptoms: Salivation, N/V/D, miosis, confusion, seizures in children
- Lavage, charcoal, benzodiazepines for seizures



Morning Glory - LSD



Poison Hemlock





Rodenticides -Anticoagulants

Warfarin

- PT increases over 8-24 hrs, peaks at 32 hrs, lasts as long as 5 days

Super warfarin (rat poison)

- Duration as long as 6-8 weeks with significant suicidal ingestions
- Pediatric “tastes” usually benign

Block synthesis of vitamin K-dependent clotting factors (II, VII, IX, X)

Treatment: Lavage, activated charcoal, vitamin K, FFP, PCCs



SALICYLATES



Salicylates (1)

- ☠ Uncouple oxidative phosphorylation, causing metabolic acidosis
- ☠ Tachypnea, coma, seizures, pulmonary edema, fever, tinnitus, tachycardia
- ☠ Acute poisoning: GI symptoms, level correlates better with toxicity - suspect in unknown OD
- ☠ Chronic poisoning: Fewer GI and more CNS symptoms, level may not correlate with toxicity
 - Consider in elderly pt with ALOC
- ☠ Children: Hyperventilation, diaphoresis, behavioral changes - caution oil of wintergreen

**Anion gap metabolic acidosis
and respiratory alkalosis**

Salicylates (2)

- ☠ Hypoglycemia common; check glucose
- ☠ May see hypokalemia, ↑T3/T4 levels
- ☠ Done nomogram no longer recommended
 - Doesn't apply to enteric-coated forms
 - Absorption is erratic
 - Treat the patient, not the level
- ☠ Treatment: **Urine alkalization**, fluids, glucose, potassium; hemodialysis for severe cases

Oil of wintergreen contains high concentrations of salicylates (very toxic)

Serotonin Syndrome

- ☠ SSRI (selective serotonin reuptake inhibitors)
 - Fluoxetine (Prozac, Sarafem)
 - Sertraline (Zoloft)
 - Citalopram (Celexa)
 - Escitalopram (Lexapro)
 - Paroxetine (Paxil)
- ☠ Combination of 2 or more drugs increases serotonin levels
 - SSRI, MAOI, meperidine, dextromethorphan, cocaine, amphetamines, St. John's wart

Serotonin Syndrome

- ☠ Clinical syndromes (rapid onset)
 - CNS (altered mental status, coma, seizures)
 - Autonomic (hyperthermia, tachycardia, hypertension)
 - Neuromuscular (myoclonus, hyperkinesia)
- ☠ Treatment: Cooling, cyproheptadine (Periactin)
- ☠ Distinguish from NMS
 - NMS: Slow onset, lead pipe rigidity
 - Serotonin syndrome: Rapid onset, myoclonus

Strychnine Toxicity

- ☠ Highly toxic alkaloid
- ☠ Blocks inhibition of postsynaptic brainstem glycine receptors and spinal cord motor neurons
- ☠ Results in CNS hyperstimulation
- ☠ Muscle twitching, facial grimacing, extensor spasm, opisthotonos (back muscle spasm, body arched)
- ☠ Patient remains conscious
- ☠ Medullary paralysis and death occur
- ☠ Treatment: Airway, benzodiazepines, may require paralytics



Tricyclic Antidepressants (1)

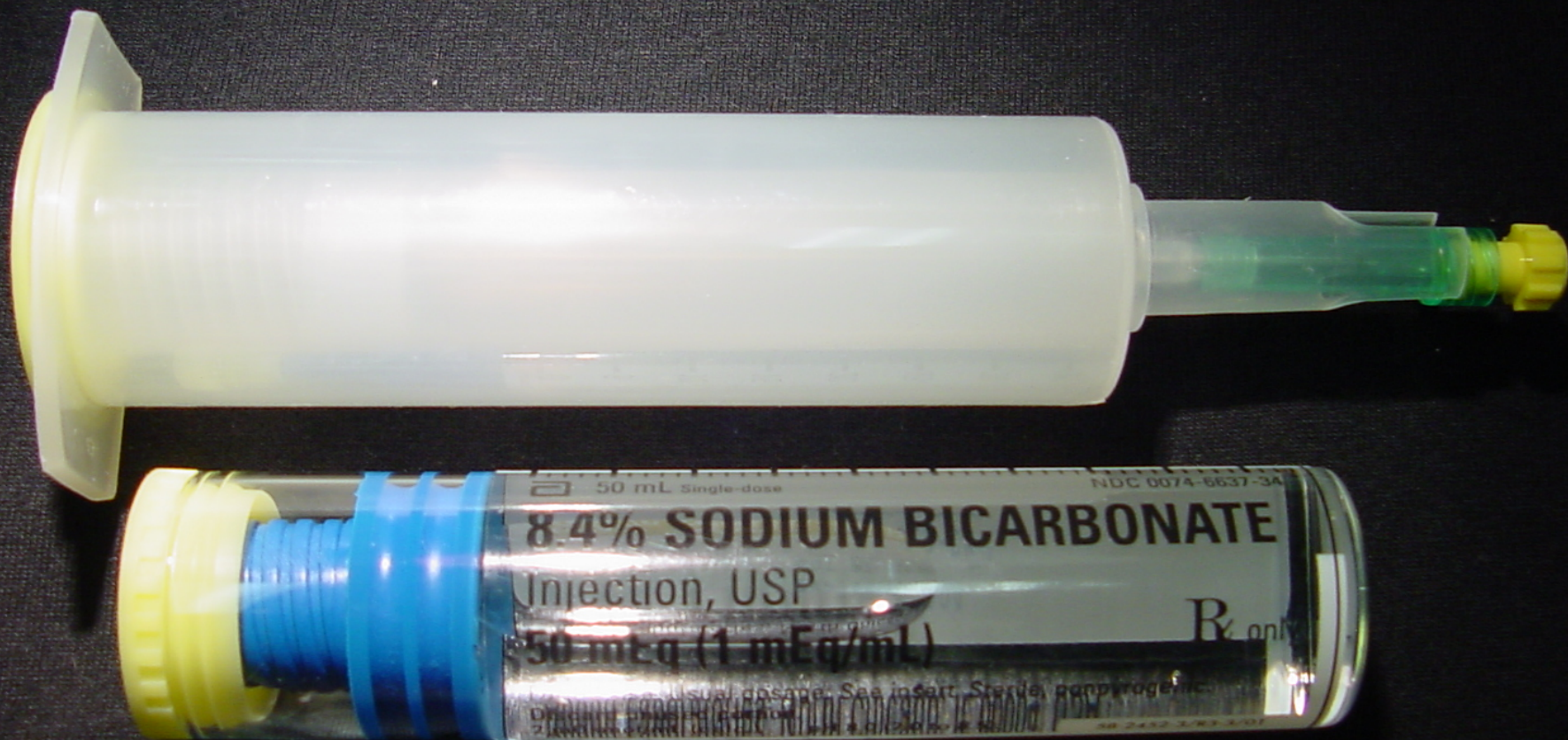
- ☠ Signs of serious toxicity manifested within 6 hours of ingestion - often earlier
- ☠ Anticholinergic effects
 - Flushed skin, dry mouth, mydriasis, decreased bowel sounds, urinary retention
- ☠ CNS effects
 - Drowsiness, confusion, ataxia, delirium, seizures, coma
- ☠ Cardiovascular effects
 - Hypotension, tachycardia, wide QRS, V-tach, torsades
 - Right axis deviation (negative S in I, positive R in aVR)

Tricyclic Antidepressants (2)

Treatment

- Lavage, multidose charcoal (avoid ipecac & flumazenil)
- Hypotension: IV saline, bicarbonate, norepinephrine
- Wide QRS, ventricular dysrhythmias: Bicarbonate, lidocaine (avoid procainamide)
- Torsades: Magnesium sulfate, overdrive pacing
- Delirium, seizures: benzodiazepines (avoid phenytoin and physostigmine)

**Sodium bicarbonate decreases
cardiac toxicity**



Xanthines

- ☠ Inhibits breakdown of cyclic AMP (the adrenergic transmitter) causing excess adrenergic excess
- ☠ Theophylline, caffeine
- ☠ Tachycardia (also multifocal atrial tachycardia = treat with IV Mg), vomiting, seizures
- ☠ Increased theophylline levels caused by erythromycin, ciprofloxacin, cimetidine, CHF, liver failure
- ☠ Acute ingestion: Hypokalemia, acidosis 2° to seizures. Levels usually correspond to toxicity
- ☠ Chronic ingestion: Toxicity seen with lower serum levels
- ☠ Lab: Metabolic acidosis, ↓K, ↓Mg, ↓PO₄
- ☠ Treatment: Multidose charcoal, phenobarbital, beta blockers, Mg⁺⁺, dialysis, hemoperfusion

Toxicology Pearls (1)

Bradycardia

- P Propranolol (beta blockers), poppies (opioids)
- A Anticholinesterase drugs
- C Clonidine, calcium channel blockers
- E Ethanol, other alcohols
- D Digoxin



Rhythm strip 1—sinus bradycardia.

Tachycardia

- F Freebase (cocaine)
- A Anticholinergics, antihistamines, amphetamines
- S Sympathomimetics, solvent abuse
- T Theophylline

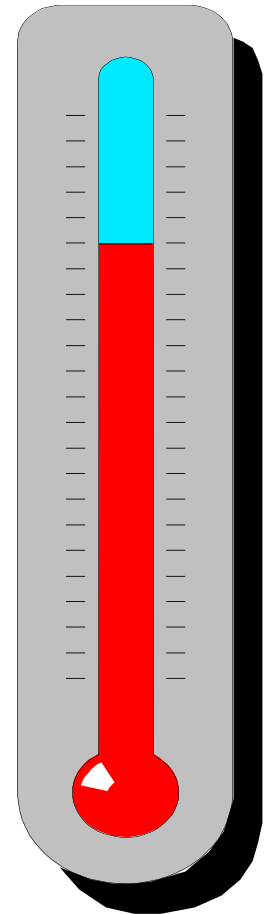


Rhythm strip 2—sinus tachycardia.

Toxicology Pearls (2)

Hyperthermia

- N NMS, nicotine
- A Antihistamines
- S Salicylates, sympathomimetics
Serotonin syndrome
- A Anticholinergics, antidepressants



Toxicology Pearls (3)

Hypothermia

- C Carbon monoxide
- O Oral hypoglycemics, insulin
- O Opioids
- L Liquor
- S Sedatives, hypnotics



Hypotension

- C Clonidine, calcium channel blockers
- R Reserpine (antihypertensive agents)
- A Antidepressants, aminophylline
- S Sedatives, hypnotics
- H Heroin (opioids)

Toxicology Pearls (4)

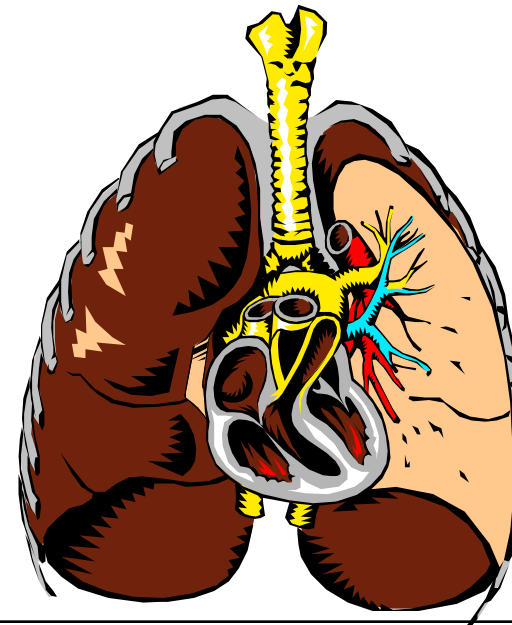
☠ Hypertension

- C Cocaine
- T Thyroid supplements
- S Sympathomimetics
- C Caffeine
- A Anticholinergics, amphetamines
- N Nicotine



☠ Hypoventilation

- S Sedatives, hypnotics
- L Liquor
- O Opioids
- W Weed (marijuana)



Toxicology Pearls (5)

☠ Hyperventilation

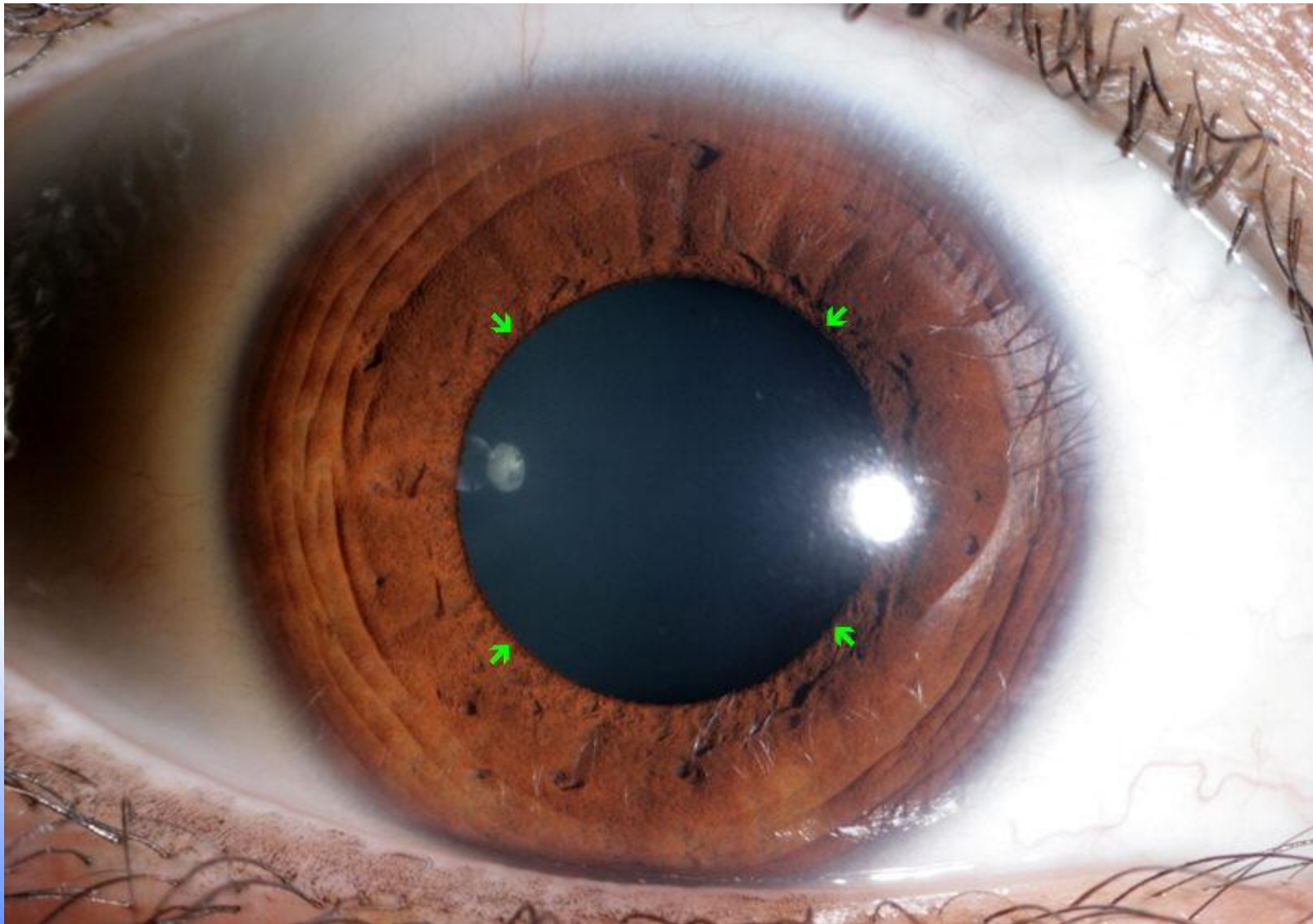
- P PCP, pneumonitis (chemical)
- A ASA (salicylates)
- N Noncardiogenic pulmonary edema
- T Toxic metabolic acidosis

☠ Miosis

- C Cholinergics, clonidine
- O Opioids, organophosphates
- P Phenothiazines, pilocarpine, pontine bleed
- S Sedatives, hypnotics



MYDRIASIS



Toxicology Pearls (6)

Mydriasis

- A Antihistamines
- A Antidepressants
- A Anticholinergics, atropine
- S Sympathomimetics (cocaine, amphetamines)

Diaphoretic skin

- S Sympathomimetics
- O Organophosphates
- A ASA (salicylates)
- P PCP

Toxicology Pearls (7)

Skin Findings



Red skin:

Anticholinergics, boric acid,
carbon monoxide



Blue skin:

Cyanosis, methemoglobinemia
(nitrates, nitrites, aniline dyes,
dapson, phenazopyridine)



Blistering:

Barbiturates, CO,
spider bites, snakes envenomations

Toxicology Pearls (8)

Seizures

- O Organophosphate
- T Tricyclic antidepressants
- I INH, insulin
- S Sympathomimetics
- C Cocaine, camphor
- A Amphetamines
- M Methylxanthines (theophylline)
- P Phencyclidine (PCP)
- B Benzo withdrawal, botanicals
- E Ethanol withdrawal
- L Lindane, lidocaine
- L Lead, lithium





Toxicology Pearls (9)

Odors

- ☠ Bitter almonds or “silver polish”: Cyanide
- ☠ Mothballs: Camphor
- ☠ Garlic: Arsenic, organophosphate, DMSO
- ☠ Rotten eggs: Sulfur dioxide, hydrogen sulfide
- ☠ Wintergreen: Methyl salicylate
- ☠ Peanuts: Vacor (rat poison)
- ☠ Carrots: Water hemlock
- ☠ Gasoline: Hydrocarbons
- ☠ Fruity: DKA, isopropanol
- ☠ Pears: Chloral hydrate



Toxicology Pearls (10)

Radiopaque Drugs

C Chloral hydrate
Cocaine packets

O Opiate packets

I Iron

(Heavy metal: Pb, As, Hg)

N Neuroleptic agents

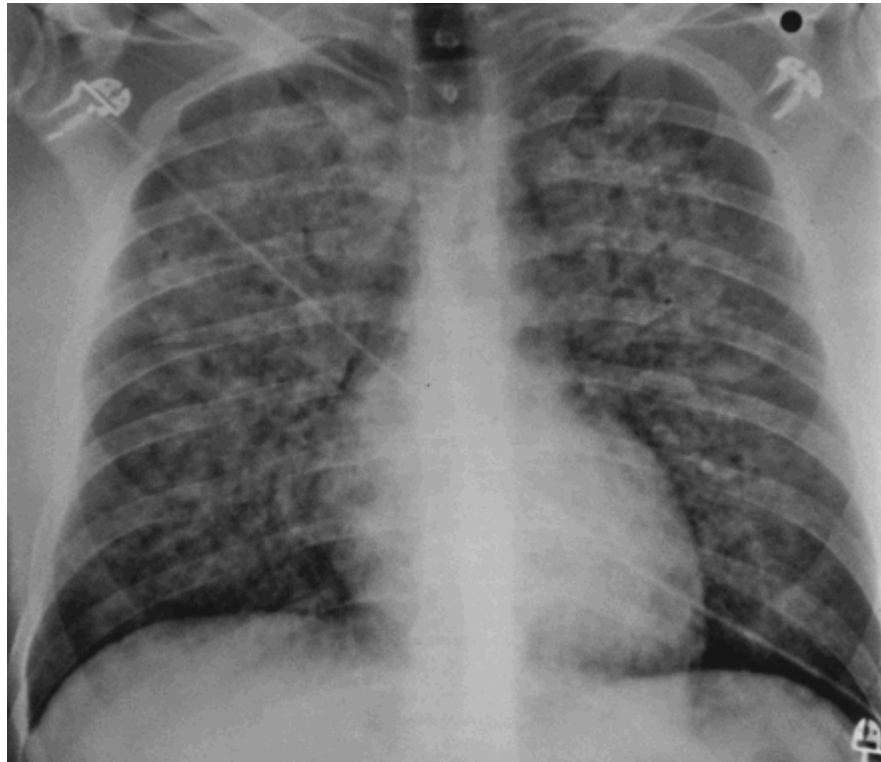
S Sustained release
products, enteric coated
preparations



Toxicology Pearls (11)

☠ Noncardiogenic pulmonary edema

- M Meprobamate, methadone
- O Opioids
- P Phenobarbital, propoxyphene
- S Salicylates



Toxicology Pearls (12)

Charcoal Not Useful

- Alcohols
- Cyanide
- Iron
- Lithium
- Arsenic

Charcoal Contraindicated

- Caustics

Multidose Charcoal

- Theophylline
- Sustained release drugs
- Phenobarbital
- Salicylates

Whole Bowel Irrigation

- Sustained release drugs
- Not absorbed by charcoal
- Body packer

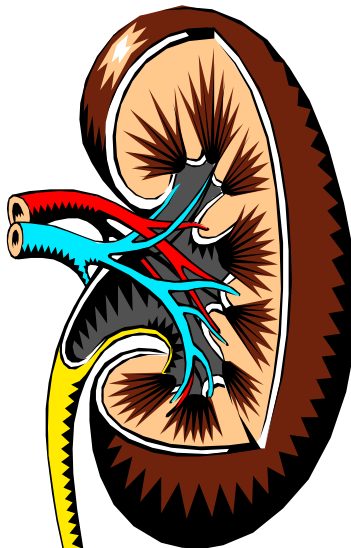
Toxicology Pearls (13)

Dialysis

- Methanol
- Isopropyl alcohol
- Ethylene glycol
- Lithium
- Salicylate
- Theophylline
- Phenobarbital

Nondialyzable

- CN
- TCAs
- Iron
- Benzodiazepines
- Phenothiazines
- Hallucinogens



Toxicology Pearls (14)

Delayed toxicity

- Acetaminophen
- Digoxin
- Ethylene glycol
- Heavy metals
- Methanol
- Mushrooms
- Narcotics
- Iron
- Salicylates
- Slow release compounds



TOXICOLOGY QUESTIONS

A 24 y/o patient presents with an unknown ingestion 2 hours prior to arrival. Which of the following statements is true?

- A. Regardless of the method, gastric emptying is always 100% effective
- B. Too much time has elapsed in this case for gastric emptying to have any benefit
- C. If the patient has a normal mental status at presentation, syrup of ipecac is indicated
- D. Sorbitol should be routinely administered with activated charcoal
- E. Activated charcoal administration may still be indicated and has the fewest adverse side effects

A 32 y/o dialysis patient presents to the ED following an overdose. Which of the following agents can be eliminated via hemodialysis?

- A. Secobarbital
- B. Lithium
- C. Digoxin
- D. Phenytoin
- E. Iron

Regarding whole bowel irrigation, which of the following statements is true?

- A. Should not be utilized in drug packers
- B. Has been shown to be clinically effective in well designed clinical trials
- C. May be utilized for iron ingestions with a positive abdominal radiograph
- D. Is contraindicated in lead ingestions since it may exacerbate encephalopathy
- E. Can be used cautiously in patients with bowel obstructions

Regarding antidotes, which statement is correct?

- A. Warfarin toxicity is treated with protamine
- B. Beta blocker toxicity is treated with glucagon
- C. Cyanide toxicity is responsive to methylene blue
- D. Calcium channel blocker toxicity is not treated with glucagon
- E. Carbamate toxicity should always be treated with 2-PAM

A 17 y/o patient presents following an unknown ingestion. His electrolytes are as follows: $\text{Na}^+ = 145$, $\text{Cl}^- = 92$, $\text{CO}_2 = 13$. What toxin did this patient most likely ingest?

- A. Acetaminophen
- B. Phenobarbital
- C. Salicylates
- D. Isopropanol
- E. Mercury

Which of the following therapies is likely to be most efficacious in the previous patient's management?

- A. N-acetylcysteine
- B. Urinary alkalization
- C. Urinary acidification
- D. 4-Methylpyrazole
- E. BAL chelation

A 15 y/o patient presents with confusion, agitation, mydriasis and dry, flushed skin. Which is the most likely etiology?

- A. MDMA toxicity
- B. *Amanita phalloides* mushroom ingestion
- C. Methadone toxicity
- D. Jimson weed exposure
- E. Cannabis exposure

A 44 y/o patient presents 30 hours after an acute on chronic acetaminophen ingestion. He reports abdominal pain, vomiting and is jaundiced. Which of the following statements is true regarding the proper management?

- A. The acetaminophen nomogram can still be used to plot serum levels to guide therapy
- B. The antidote indicated in this scenario can only be administered orally
- C. The patient's hepatotoxicity unlikely to be due to acetaminophen toxicity
- D. N-acetylcysteine is still indicated and may have clinical benefit in this delayed setting
- E. N-acetylcysteine therapy is contraindicated after 24 hours post ingestion

A 29 y/o female presents to the ED complaining of nausea, vomiting and headache. She is flushed and diaphoretic. She reports being seen & treated 24 hours ago for a vaginal discharge. Which is the most likely cause of her symptoms?

- A. Hypersensitivity reaction to doxycycline
- B. Subarachnoid hemorrhage
- C. Viral meningitis
- D. Jarisch-Herxheimer reaction
- E. Disulfiram reaction

A 25 y/o patient presents with headache, vomiting and somnolence. She reports using 2 gas space heaters for warmth last night. You order a carbon monoxide level which is measured at 25%. Which statement is true?

- A. The patient's pulse ox will be low
- B. CO shifts O₂ saturation curve to the right
- C. HBO is always indicated in this case due to CO neurotoxic effects
- D. If the patient is pregnant, the fetal hemoglobin has a high affinity for CO
- E. Administration of the cyanide antidote kit should also be considered in this case

A 39 y/o alcoholic male presents with nausea, vomiting and visual disturbances. His $\text{CO}_2 = 5$. His osmolal gap = 40. What is likely to improve this patient's condition ?

- A. Intravenous 4-methylpyrazole
- B. Intravenous octreotide
- C. Intravenous methylene blue
- D. Intravenous N-Acetyl cysteine
- E. Intravenous physostigmine

Which of the following entities causes a normal anion gap acidosis?

- A. Isoniazid toxicity
- B. Salicylates
- C. Renal tubular acidosis
- D. Uremia
- E. Metformin

An 18 year old patient presents to the ED comatose with midrange pupils following a rave party on a Saturday night at 2am. After no response to D50 and naloxone therapy, the patient is orally intubated without complications. At 6am the patient suddenly becomes agitated and extubates himself. What drug of abuse is this scenario most consistent with?

- A. LSD-laced psilocybe mushrooms
- B. Ketamine
- C. Gamma hydroxybutyric acid
- D. Ecstasy MDMA
- E. Fentanyl

A 47 y/o patient presents with an altered mental status following an ingestion of an unknown quantity of clonidine. Which of the following is true?

- A. This patient's symptoms will be similar to an anticholinergic overdose
- B. The patient will experience hypotension, followed by hypertension
- C. The patient will be agitated with dilated pupils
- D. Naloxone may be beneficial
- E. This overdose does not share any similarities with a opiate toxicity

A 23 y/o fire fighter was involved in a extinguishing a large 5 alarm factory fire. He presents to the ED comatose with profound metabolic acidosis despite high flow oxygen therapy. What is the most appropriate intervention?

- A. Sodium thiosulfate and hydroxycobalamine
- B. Methylene blue
- C. Pyridoxine therapy
- D. Pralidoxime therapy
- E. Emergent hemodialysis

A 78 y/o patient is hyperkalemic with a digoxin level = 8.5. Her ECG shows PAT with AV block. Which of the following would be most appropriate?

- A. Administer Digibind
- B. Administer CaCl for this patients hyperkalemia
- C. Withhold further digoxin awaiting clearance of the patient's toxicity
- D. Cardiovert the patient with 50 Joules
- E. Administer procainamide

A 45 y/o patient was working with a liquid while etching glass. He was not wearing his gloves. He is complaining of intense hand pain. Which of the following is indicated?

- A. Copious irrigation with copper sulfate
- B. Intradermal calcium chloride
- C. Topical calcium gluconate
- D. Intravenous potassium phosphate
- E. An immediate surgical intervention

A 36 y/o migrant worker presents in status epilepticus despite benzodiazepines, phenytoin and phenobarbital administration. His ABG pH is 6.8 on high flow oxygen therapy. What is the next most appropriate therapeutic step?

- A. Vitamin B6 pyridoxine therapy
- B. Vitamin B12 hydroxycobalamine
- C. Vitamin B3 niacin
- D. Vitamin B1 thiamine
- E. Bicarbonate drip

Which of the following agents causes miosis?

- A. Cocaine
- B. Jimson weed
- C. Atropine
- D. Methamphetamine
- E. Fentanyl

A 39 y/o Type II diabetic presents following an overdose. He is unresponsive with a GLC = 25 mg/dl. Which therapy may be indicated if this patient's hypoglycemia persists despite aggressive dextrose administration?

- A. Octreotide
- B. Cyproheptadine
- C. Insulin rescue therapy
- D. Metformin administration
- E. Emergent pancreatectomy

Toxicology Answer Key

1. E

2. B

3. C

4. B

5. C

6. B

7. D

8. D

9. E

10.D

11.A

12.C

13.C

14.D

15.A

16.A

17.C

18.A

19.E

20.A