

2024 ACMT
Board Review Course
Interactive Cram Session #3
July 12, 2024



ACMT

American College
of Medical Toxicology

Interactive Cram Session

Today's goal is to be **interactive**, **engaging**, and **educational**:

Introductions

5-min “Key Takeaways”

Q&A with Speakers

Pop Quiz

Today's session is being recorded and will be accessible on-demand.

DISCLAIMER

According to ABEM policy, the planning committee and faculty for this course are not allowed to have intimate knowledge of the exam or write exam questions. The content of this course is based on the expertise of ACMT members, who are specialists in Medical Toxicology.

We do not have direct knowledge of the exam content. ABEM test question writers are prohibited from participating in any board review or preparatory course. The study materials, including the Quiz Bank and pop quiz questions, are based on years of collective experience from the Board Review Course committee, but we do not guarantee that these questions fully represent the exam content.

CRAM SESSION TOPICS | FRI. JULY 12, 2024

Hydrocarbons

Alcohols & Glycols

Aquatic Toxicity

Radiation

Cardiovascular Toxins

POP QUIZ

10 Qs randomly selected from Quiz Bank

Give it your best guess and then we'll discuss the answers!

Question 1

Which of the following agents is derived from coal tar and associated with the development of methemoglobinemia?

- A. Benzene
- B. Kerosene
- C. Naphthalene
- D. Naphthene
- E. Phenol

Question 1 - Answer

Which of the following agents is derived from coal tar and associated with the development of methemoglobinemia?

- A. Benzene
- B. Kerosene
- C. Naphthalene**
- D. Naphthene
- E. Phenol

EXPLANATION: Naphthalene has been used in mothballs; however, it has been mostly replaced by other compounds in the US. It is a pure white substance with a noxious odor. It is the number one component of coal tar. It is well associated with both hemolysis, particularly in G6PD deficient individuals, and methemoglobinemia. Naphthene is a cyclic hydrocarbon often found in lighter fuels. Benzene is associated with various hematologic problems, but not with methemoglobinemia.

Question 2

Which of the following is the toxic metabolite of n-hexane?

- A. 2,5-hexanedione
- B. Methyl ethyl ketone
- C. 1,2 dibromochloro
- D. 1,3-headione

Question 2 - Answer

Which of the following is the toxic metabolite of n-hexane?

A. 2,5-hexanedione —————→

EXPLANATION: The correct answer is 2,5-hexanedione. It is also the toxic metabolite of Methyl-n-Butyl Ketone. 2,5-hexanedione causes peripheral neuropathy.

B. Methyl ethyl ketone

C. 1,2 dibromochloro

D. 1,3-hexanedione

Question 3

Numerous neonatal deaths occurred after the development of severe metabolic acidosis, respiratory depression, and encephalopathy following flushes containing what pharmaceutical additive?

- A. Benzyl alcohol
- B. Diethylene glycol
- C. Polyethylene glycol
- D. Sorbitol
- E. Tryptophan

Question 3 - Answer

Numerous neonatal deaths occurred after the development of severe metabolic acidosis, respiratory depression, and encephalopathy following flushes containing what pharmaceutical additive?

A. Benzyl alcohol →

B. Diethylene glycol

C. Polyethylene glycol

D. Sorbitol

E. Tryptophan

EXPLANATION: Neonatal gasping syndrome resulted from benzyl alcohol. Symptoms include severe anion gap metabolic acidosis, respiratory depression with gasping, and encephalopathy. All neonates had received bacteriostatic NaCl or water flushes containing 0.9% benzyl alcohol. Benzyl alcohol is normally oxidized rapidly to benzoic acid after being conjugated with glycine in the liver, and excreted as hippuric acid. However, this metabolic pathway is not well developed in premature infants. The benzyl alcohol was metabolized to benzoic acid but not able to be conjugated causing a metabolic acidosis.

Question 4

What was the cause of the 1937 Massengill sulfanilamide disaster which led to the eventual development of the Food, Drug, and Cosmetic Act?

- A. Benzyl Alcohol
- B. Dioxin (2,3,7,8 TCDD)
- C. Diethylene glycol
- D. Diethylstilbestrol
- E. Polysorbate 80

Question 4 - Answer

What was the cause of the 1937 Massengill sulfanilamide disaster which led to the eventual development of the Food, Drug, and Cosmetic Act?

- A. Benzyl Alcohol
- B. Dioxin (2,3,7,8 TCDD)
- C. Diethylene glycol**
- D. Diethylstilbestrol
- E. Polysorbate 80

EXPLANATION: Diethylene glycol, a nephrotoxin, was substituted for propylene glycol and glycerin in the liquid formulation of sulfanilamide to lower cost. This led to the death of more than 100 individuals from renal failure. Symptoms included vomiting, abdominal pain, anuria, seizures, and coma. Benzyl alcohol was implicated in neonatal gasping syndrome, polysorbate 80 caused a variety of problems in neonates when used in a parenteral vitamin E preparation, dioxin has caused chloracne in exposed individuals, and diethylstilbestrol is a carcinogen implicated in genitourinary cancers of children born to mothers who used this agent.

Question 5

Toxin from this creature is expected to affect which phase of the cardiac cycle?

- A. Phase 0
- B. Phase 1
- C. Phase 2
- D. Phase 3
- E. Phase 4



Question 5 - Answer

Toxin from this creature is expected to affect which phase of the cardiac cycle?

A. Phase 0

B. Phase 1

C. Phase 2

D. Phase 3

E. Phase 4



EXPLANATION: This is a blue ringed octopus; it contains tetrodotoxin which is a sodium channel blocker

Question 6

A patient complains of weakness, drowsiness, incoordination, and shortness of breath two hours after a dinner of steamed mussels at a “Belgian”- cuisine restaurant on the California coast. Which of the following is the most likely cause of her illness?

- A. Amnesic shellfish poisoning
- B. Ciguatera
- C. Neurotoxic shellfish poisoning
- D. Paralytic shellfish poisoning
- E. Tetrodotoxin poisoning

Question 6 - Answer

A patient complains of weakness, drowsiness, incoordination, and shortness of breath two hours after a dinner of steamed mussels at a “Belgian”- cuisine restaurant on the California coast. Which of the following is the most likely cause of her illness?

- A. Amnesic shellfish poisoning
- B. Ciguatera
- C. Neurotoxic shellfish poisoning
- D. Paralytic shellfish poisoning**
- E. Tetrodotoxin poisoning

EXPLANATION: Paralytic shellfish poisoning is from saxitoxin. The poisoning is similar to tetrodotoxin and is the result of decreased sodium channel permeability. In contrast, tetrodotoxin causes paralysis via sodium channel blockade. Symptoms may occur within 30 minutes of ingestion. Neurologic symptoms predominate and include paresthesias, numbness, headache, ataxia, vertigo, and cranial nerve dysfunction, as well as weakness, respiratory failure, and paralysis. GI symptoms are less common. Amnesic shellfish poisoning is from domoic acid. Patients may develop GI symptoms in addition to coma, seizures, hemiparesis, purposeless chewing, and memory loss. Elderly patients are at a higher risk for death and permanent anterograde memory loss. Neurotoxic shellfish poisoning is due to brevetoxin. It is characterized by GI and neurologic symptoms that are similar to ciguatoxin. If the toxin is aerosolized, patients can develop respiratory irritation.

Question 7

Which of the following factors has the greatest effect on the amount of radiation absorbed from a given exposure?

- A. Age of the person
- B. Concomitant explosive device
- C. Distance from source
- D. History of previous exposure
- E. Inhalational dosing

Question 7 - Answer

Which of the following factors has the greatest effect on the amount of radiation absorbed from a given exposure?

- A. Age of the person
- B. Concomitant explosive device
- C. Distance from source**
- D. History of previous exposure
- E. Inhalational dosing

EXPLANATION: Important factors which determine the amount of radiation absorbed include the distance from exposure, the dose rate, the duration of exposure and shielding from the source. Distance makes an exponential difference in radiation absorption.

Question 8

Which of the following radioisotopes is adsorbed by prussian blue?

- A. Cadmium
- B. Cesium
- C. Iodine
- D. Tritium
- E. Uranium

Question 8 - Answer

Which of the following radioisotopes is adsorbed by prussian blue?

A. Cadmium

B. Cesium



EXPLANATION: Prussian blue was demonstrated to significantly decrease the half-life of radioactive cesium in the famous 1987 Goiania (Brazil) radiation incident.

C. Iodine

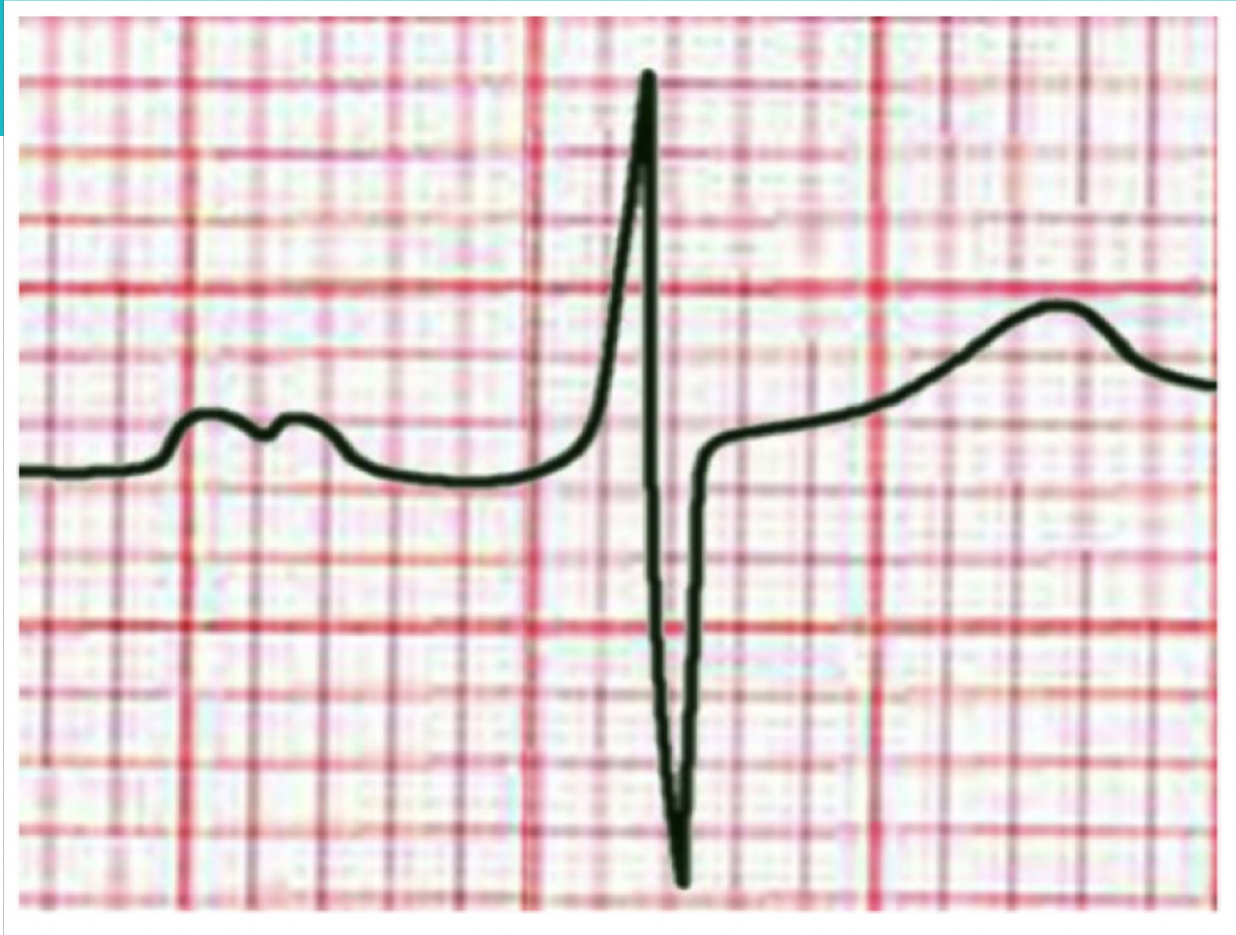
D. Tritium

E. Uranium

Question 9

Take a look at the image. The ECG abnormality, assuming not related to an underlying valvular disease, may be seen with which of the following medications?

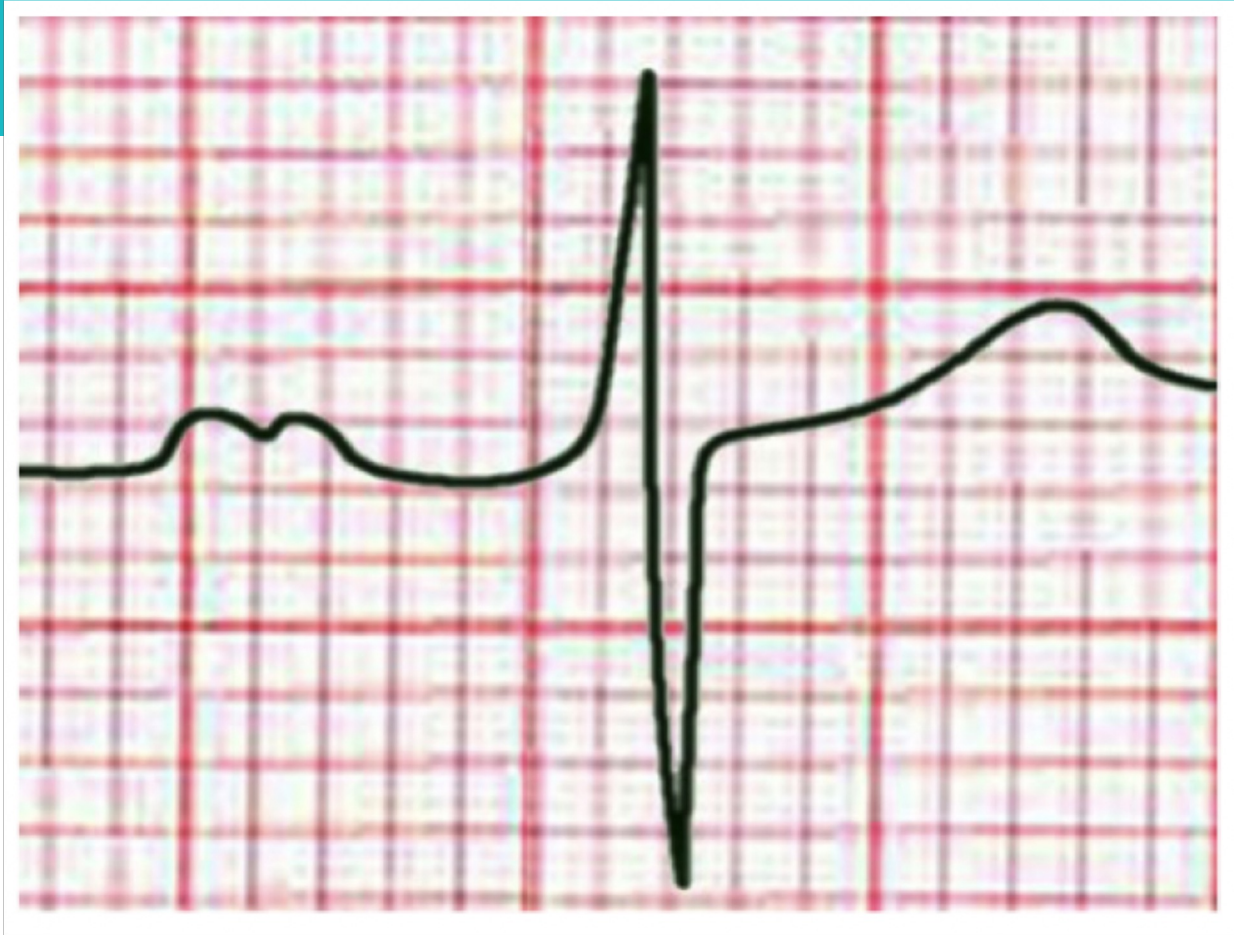
- A. Amiodarone
- B. Bretylium
- C. Bupropion
- D. Quinine
- E. Verapamil



Question 9 - Answer

Take a look at the image. The ECG abnormality, assuming not related to an underlying valvular disease, may be seen with which of the following medications?

- A. Amiodarone
- B. Bretylium
- C. Bupropion
- D. Quinine**
- E. Verapamil



EXPLANATION: A notched p wave is seen with quinine.

Question 10

Digoxin is a medication with a large volume of distribution following a two compartment model. How long after medication ingestion does digoxin reach a steady state?

- A. 30 - 90 minutes
- B. 3 - 4 hours
- C. 6 - 8 hours
- D. 1 - 2 days
- E. 4 days - 1 week

Question 10 - Answer

Digoxin is a medication with a large volume of distribution following a two compartment model. How long after medication ingestion does digoxin reach a steady state?

A. 30 - 90 minutes

B. 3 - 4 hours

C. 6 - 8 hours →

EXPLANATION: Digoxin reaches a steady state about 6 - 8 hours after ingestion.

D. 1 - 2 days

E. 4 days - 1 week

FEEDBACK SURVEY

Before you leave, please fill out the feedback survey.

This survey should appear in your browser when the meeting ends.

Let us know how we can improve the next interactive cram session!

COMING UP!

Interactive Cram Session #4
July 26, 2024



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CRAM SESSION TOPICS | FRI. JULY 26, 2024

Classic Toxicology

Antipsychotics

Psychotropics

Endocrine

TBA

SOCIAL MEDIA
@acmtmedtox



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#ACMTBoardPrep

ACMT BOARD REVIEW COURSE

Hydrocarbons Very Shortened....

Kurt Kleinschmidt, MD

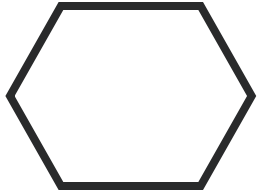
Professor of Emergency Medicine

Division Chief, Medical Toxicology

UT Southwestern Medical Center

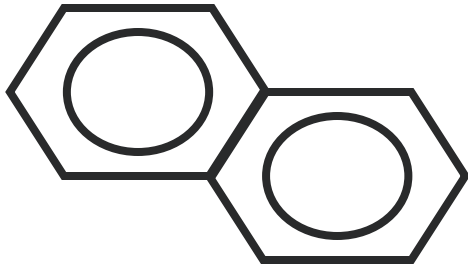


THE NAPHTHS



Acts like an aliphatic
Charcoal Lighter Fluid

Cycloparaffins (Naphthene) (Cyclohexane)



Naphthalene

Old Moth Ball item...mostly replaced
Pure white with noxious odor
#1 component of coal tar
Metabolized → active alpha-naphthol →
delayed clinical

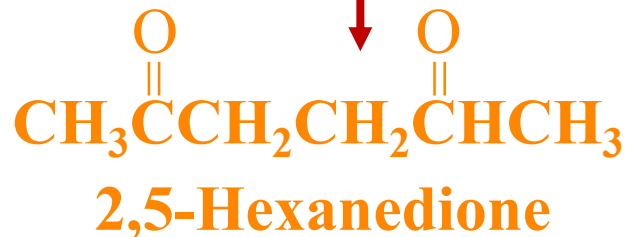
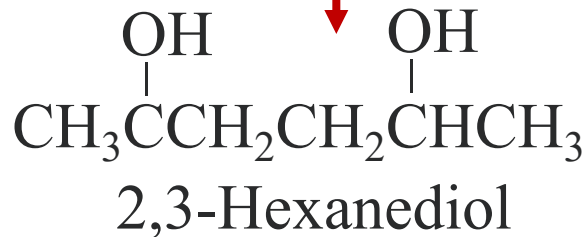
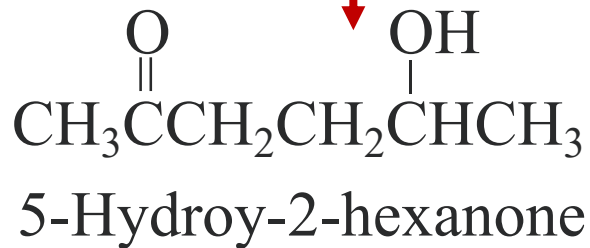
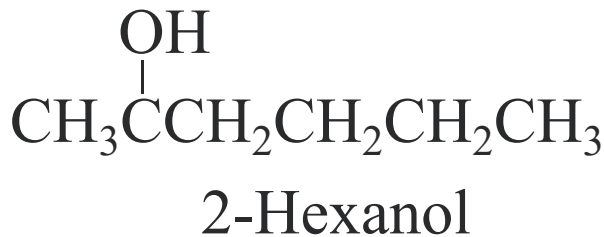
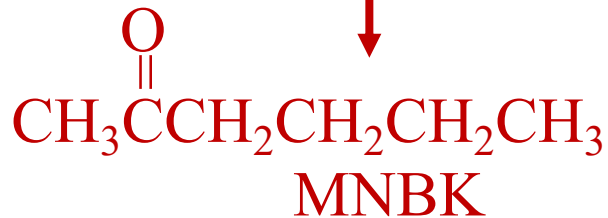
Clinical

Hemolysis #1 (esp G6PD deficient)
Methemoglobinemia



N-HEXANE

METHYL-N-BUTYL KETONE



γ-diketone

HYDROCARBON: NEUROPATHY

Peripheral

- Classic axonopathy:

N-Hexane

Methyl-n-butyl ketone

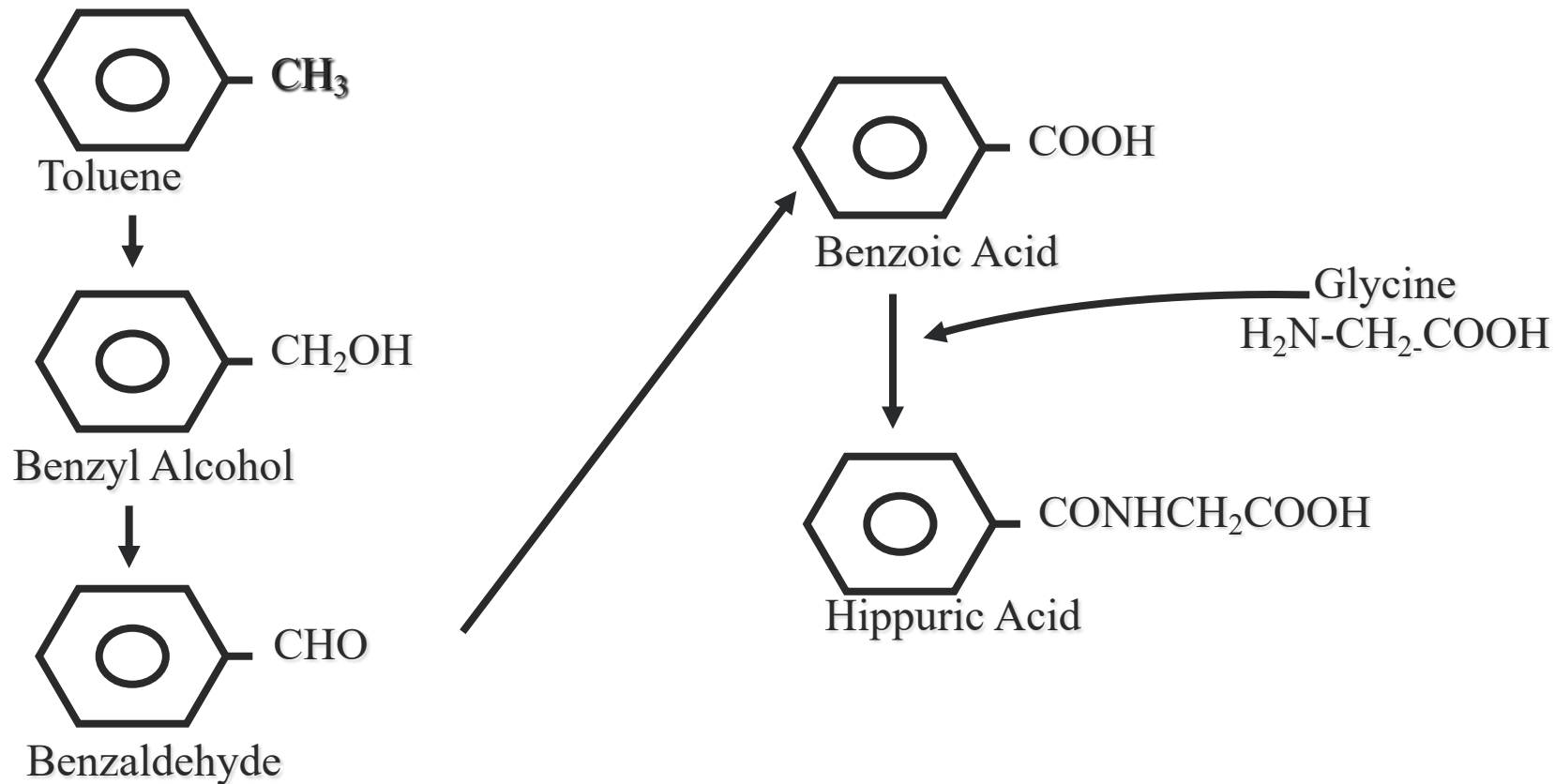
2,5 Hexanedione

- Also:

- CS₂
- Acrylamide
- Ethylene Oxide
- Styrene

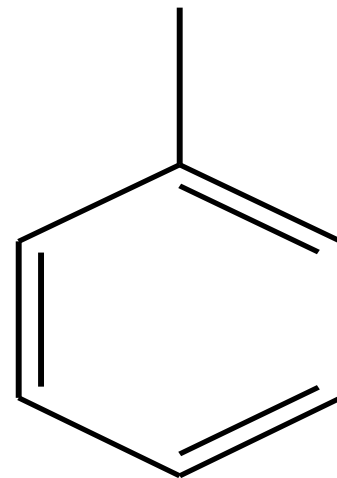
Distal axonopathy
Optic nerve (resembles optic neuritis)
CV (HTN, CAD)
Repro (SAB, prematurity, decreased libido)
Disulfiram Reaction

TOLUENE METABOLISM



AROMATIC HYDROCARBONS: TOLUENE

- Colorless; Benzene-like odor; Flammable
- Natural from crude oil and in the “tolu” tree.
- Uses: **Big solvent!** Has replaced benzene as the top solvent. Paints, Thinners, Glues, etc. Some printing and leather tanning processes.
- Inhalation: Rapid intoxication
- Chronic Use
 - **Distal RTA**
 - **Metabolic acidosis (Hippuric acid)**
 - **Hypokalemia and associated symptoms**
 - **Leukoencephalopathy**



HALOGENATED HCs THAT HAVE 1 OR 2 CARBONS

CCl_4 Carbon Tetrachloride

CHCl_3 Chloroform

CH_2Cl_2 Methylene Chloride

$\text{Cl}_2\text{H}-\text{C}-\text{C}-\text{HCl}_2$ Tetrachloroethane

$\text{ClH}_2-\text{C}-\text{C}-\text{HCl}_2$ Trichloroethane

$\text{Cl}_2-\text{C}=\text{C}-\text{Cl}_2$ Tetrachloroethylene

$\text{ClH}-\text{C}=\text{C}-\text{Cl}_2$ Trichloroethylene

$\text{ClH}-\text{C}=\text{C}-\text{H}_2$ Monochloroethylene

$\text{Cl}_3\text{C}-\text{C}(\text{OH})_2\text{H}$ Chloral Hydrate

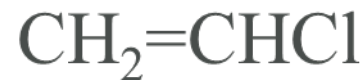
BONUS!!!

The only
IARC 1 among the
halogenated HCs

(PERC)
(TCE)
(Vinyl Chloride)

HALOGENATED HYDROCARBONS

VINYL CHLORIDE



- Chloroethene; chloroethylene; ethylene monochloride; Monochloroethene
- Gas; Flammable
- Uses: Make Polyvinyl chloride (PVC)
- **Hepatic angiosarcoma** - clear association (The only IARC 1 Halog. HC!)
- **Acrosclerosis**...Classic Rheum Problem
 - **Acroosteolysis...osteolysis** in terminal phalanges of some fingers
 - **Raynauds**
 - **Scleroderma...thickening** of the skin or raised nodules on the arms

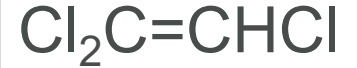
HYDROCARBON: NEUROPATHY

Cranial

- **Classic: Trichloroethylene (TCE) →**
 - **Trigeminal neuralgia** & other facial neuropathies
- Reported:
 - Trichloroethanol → Peripheral & Cranial neuropathies
 - Perchloroethylene (PERC) ... likely due to TCE being a contaminant in a PERC preparation

HALOGENATED HYDROCARBONS

TRICHLOROETHYLENE (TCE)



- Uses: Solvent for metal parts, paint removers, typewriter correction fluids, and spot removers.
- Was replaced by tetrachloroethene (PERC) b/c
TCE is more hepatotoxic than PERC
- Metabolism: → **Epoxide** (like PERC) ... but ... this is its 1° metabolic route → Trichloroacetic acid & Trichloroethanol
- Hepatic injury (DUE TO Epoxide intermediate)

HALOGENATED HYDROCARBONS

TRICHLOROETHYLENE (TCE)

- **Degreaser's Flush**
 - TCE #1, but other solvents associated i.e. xylene
 - Only need to be exposed to the vapor
 - Disulfiram-like reaction; Altered EtOH metabolism
 - Mechanism is unclear
 - Starts w/i 30 min of alcohol exposure;
Peaks within 30-60 min.; Clears w/i 1-hour.
 - Starts: Nose and cheeks: May → upper torso
- **Trigeminal Neuropathy** is classic...also...
other cranial nerve neuropathies

HYDROCARBON: LIVER

- **Chlorinated HCs** are #1 bad boys
- # of halogens ↑ & atomic weight of halogens ↑
→ Toxicity ↑
- **CCl₄** >> Benzene, Trichloroethylene
- Less toxic: trichloroethylene, tetrachloroethylene, and 1,1,1-trichloroethane
- AST/ALT ↑ & Hepatomegaly; Reversible
- Phase I activation → Reactive intermediate
→ Lipid Peroxidation, Free Radicals
→ Direct Membrane Injury
- Also
 - **Vinyl chloride** → **Liver carcinogen (angiosarcoma)**
 - **Aromatic HCs**



ACMT BOARD REVIEW COURSE

Alcohol & Glycols Very Very Shortened...

Kurt Kleinschmidt, MD

Division Chief, Medical Toxicology

UT Southwestern Medical Center

Dallas, Texas



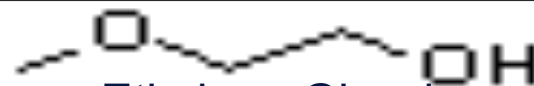
GLYCOL ETHERS

- Two groups:
 - Ethylene glycol ethers
 - Propylene glycol ethers
- Ethylene Glycol Ethers
 - Many exist
 - 2 examples

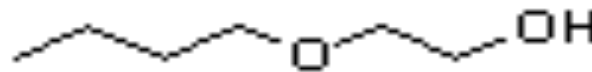
Ethers: R_1-O-R_2

- Propylene Glycol Ethers
 - Many
 - Example

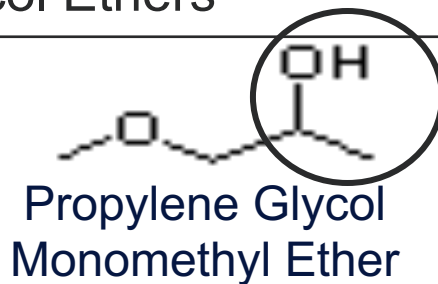
EG Monoalkyl Ethers base:
 $R_1OCH_2CH_2OR_2$
 R_1 =Alkyl gp; R_2 =H or Acetate



Ethylene Glycol
Methyl Ether (EGME)



Ethylene Glycol
Butyl Ether (EGBE)



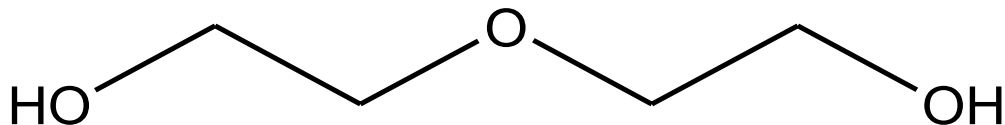
Propylene Glycol
Monomethyl Ether

Is a 2° alcohol
(On the 2nd Carbon)

DIETHYLENE GLYCOL – A GLYCOL ETHER

- Description:
 - Clear, Colorless
 - Syrupy liquid
 - Water soluble
- Uses:
 - Solvent
 - Antifreeze
 - Humectant
 - Plasticizer
- History: DEG substituted for propylene glycol or glycerin in oral elixirs has been a classic and too common problem:
 - **Elixir Sulfanilamide Disaster of 1937:**
 - DEG used as diluent in this elixir by the Massengil company → 105 Deaths
 - Resulted in the **1938 Food, Drug, and Cosmetic Act** - regulated the formulation & safety of medications
 - Liquid paracetamol 1996 (Haiti)

→ Renal ↓↓;
85 kids died



DIETHYLENE GLYCOL

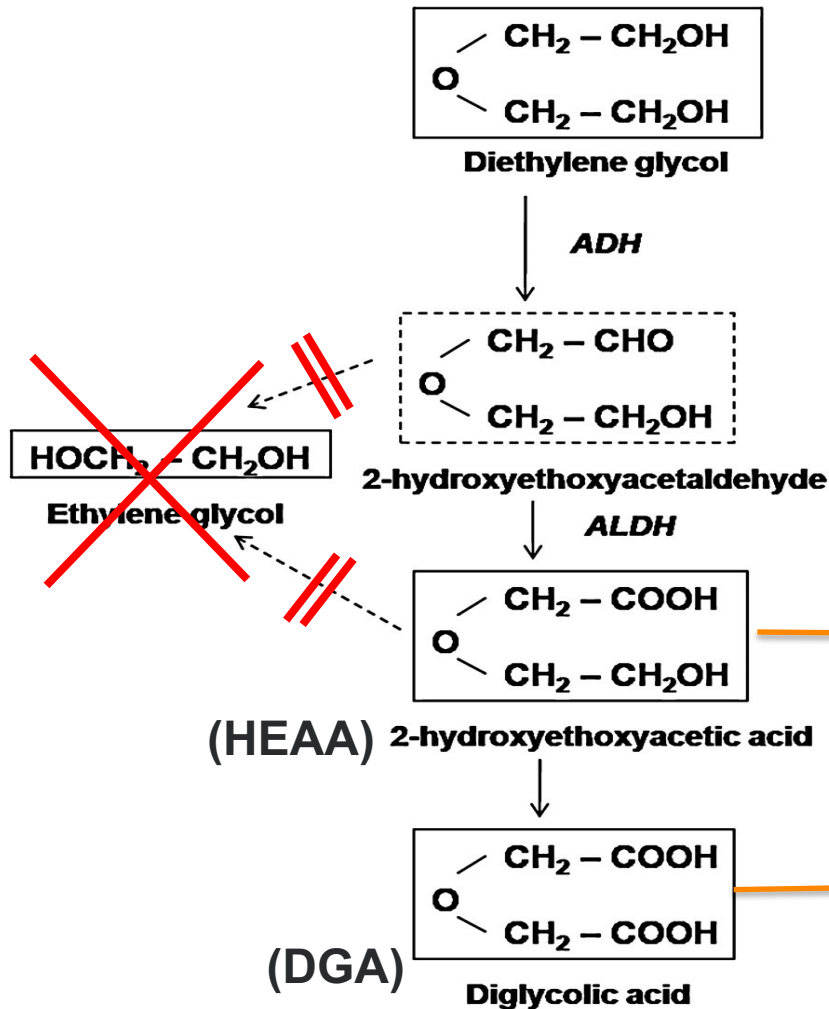
CLINICAL

- Stage 1
 - GI sxs (NV + abd pain), intoxication, and acidosis
 - Onset is delayed 1-2 days!!!
- Stage 2 (ARF)
 - Renal failure #1 consistent problem after 2-6 days.
 - Acidosis is worse now. Severe acidosis uncommon until ARF present (Unlike EG or methanol)
- Stage 3 (Neuro)
 - If pt survives the ARF, then Neuro - CNS ↓, CN VII neuropathy, extremity weakness

TREATMENT

- Hemodialysis recommended for symptomatic pts
- ADH inhibitors seem to make sense (?) but patients have done poorly despite their use

DEG METABOLISM



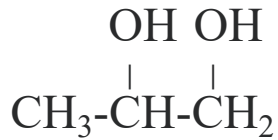
Mammals don't have the enzymes to break an ether bond So don't end up with ethylene glycol

Renal Elimination

HEAA correlates with the **acidosis** (Not DGA)

Enters Renal Cells → accumulates → **Kidney badness** (Little is renally eliminated)

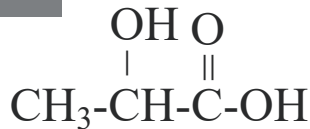
PROPYLENE GLYCOL



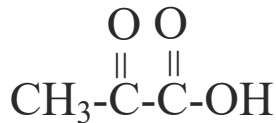
Propylene Glycol



Alcohol &
Aldehyde
Dehydrogenase



Lactic Acid



Pyruvic Acid



NAD⁺
NADH



Krebs Cycle

Found:

Med Diluent

Silver sulfadiazine

Most Common

Mild **lactic acidosis**

Hyperosmolarity

Rare Serious

Hypotension (acute)

Renal (chronic)

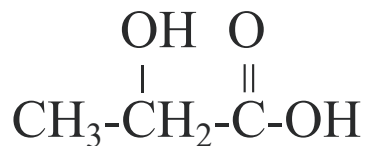
Cardiac conduction changes
(Wide QRS; ↑ wave)

NADH:NAD⁺ RATIO AND ALCOHOLS

Alcohol Metabolism → **NADH**

Ethylene Glycol 4 Oxidative steps

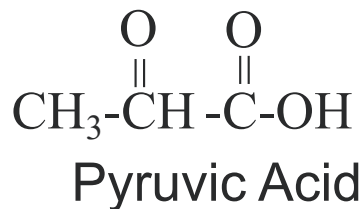
Isopropanol 1 Oxidative step



Lactic Acid



NAD⁺
(LDH)
NADH



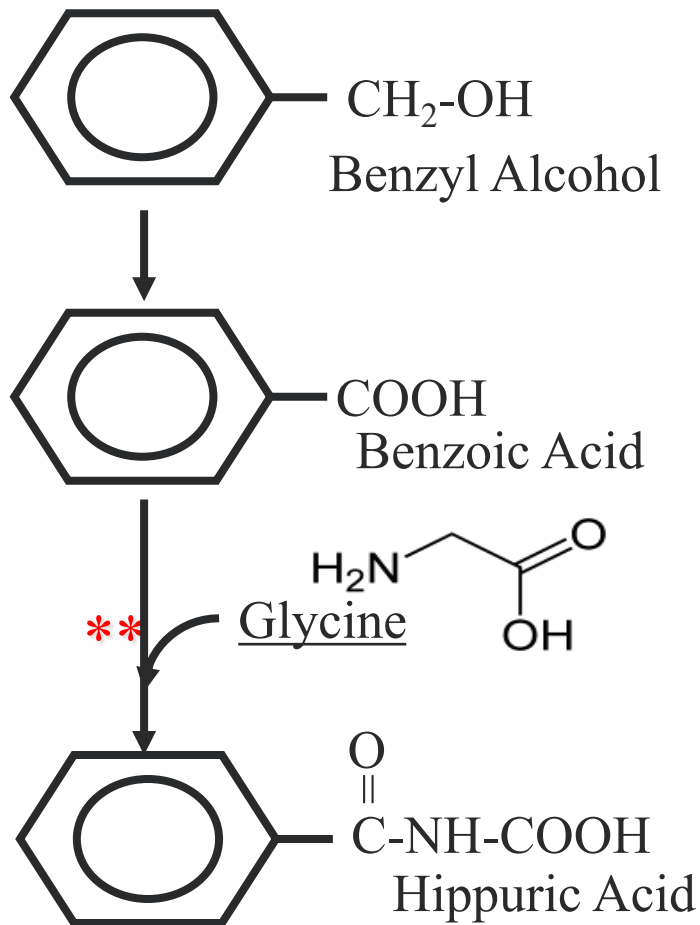
Glycolysis



Krebs Cycle



BENZYL ALCOHOL

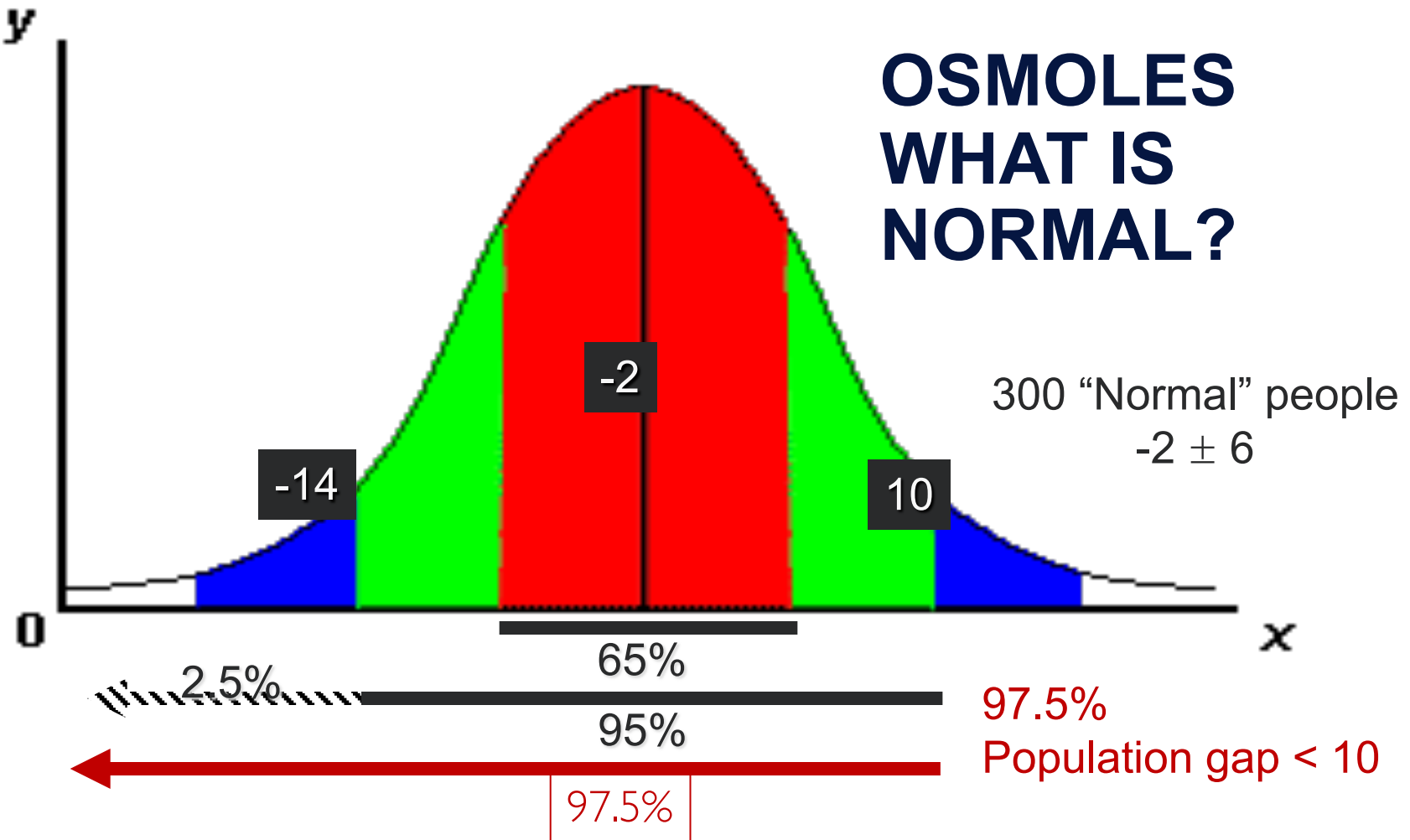


- Common IV preservative at 0.0-2.0%
- Toxicity usually due to repeated dosing
- Saline / Heparin use in pre-term neonates

**** Premature infants have decreased ability to make Hippurate**

“Gasping” syndrome

OSMOLES WHAT IS NORMAL?



DIAGNOSIS OSMOL GAP

- Ethylene glycol at 50 mg/dL will add only ~ 8 mOsm
- Not surprising: Case of a patient with a gap of 7.2 who eventually needed dialysis

- A normal gap does not rule-out a toxic ingestion
- A really big gap (> 40) likely is a toxic alcohol
- An osmole gap is a substitute for a toxic alcohol level.

Do NOT use a gap if you can get timely levels

ETHYLENE GLYCOL PATHOPHYSIOLOGY AND CLINICAL

If severe...

- Cardiac Dysrhythmias and Depression 2° to
Metabolic acidosis & ↓ Ca^{++}
- $\text{Ca}^{++} \downarrow \rightarrow$ (1) myoclonus, tetany, seizures
(2) ↑ QT interval
- Multisystem organ failure, ARDS

Late and unusual:

- Cranial nerve palsies
- Bone Marrow suppression

Causes **False elevation of lactate**
In some analyzers

ETHYLENE GLYCOL PATHOPHYSIOLOGY & CLINICAL

- CNS Depression
- Renal Damage (ATN)
 - Due to:
 1. Direct toxicity
 2. Oxalate crystals in tubules
 - Early as 16 hours; Failure by 48 hours
(Fast compared to the EG ethers!!!)
- Metabolic acidosis due to:
 1. Glycolate (#1) & Oxalate & other metabs
 2. EG oxidation \rightarrow NADH / NAD⁺ ratio $\uparrow \rightarrow$
 1. \uparrow Lactate / Pyruvate Ratio &
 2. \downarrow TCA Cycle activity

POLYETHYLENE GLYCOLS

- Various compounds with wide range molecular weights
- The “number” is the avg. MW (Range 200-40,000)
- **< 600** are clear, viscous liquids; Absorbable, can be toxic
 - Partially metabolized (ADH) → hydroxyacid and diacid metabolites (excreted unchanged in urine)
 - diethylene and triethylene glycol
 - In: Lorazepam, Decadron, ophth ointments
 - Toxicity
 - **Nephrotoxicity (ATN)**
 - **Hyperosmolarity and metabolic acidosis**
- **> 1000** are solid (pastes to flakes) but soluble
 - Not absorbed and are Non-Toxic
 - Ex - PEG 3350 - PEG-ELS (WBI agent)

ACMT BOARD REVIEW COURSE

Aquatic Toxicity- CRAM SESSION

Ann Arens, MD

Emergency Medicine, Ochsner Medical Center, New Orleans, LA

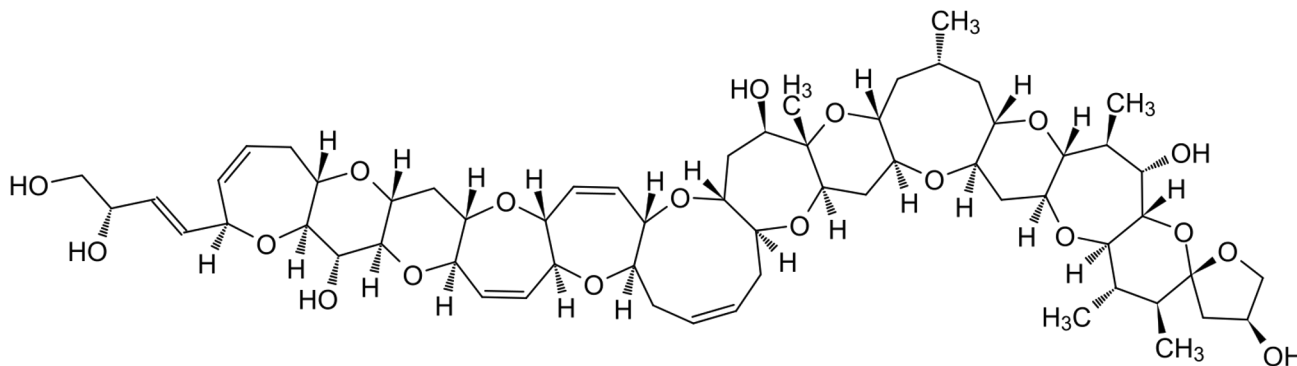
Medical Toxicology, Ochsner Medical Center, New Orleans, LA



TOXICITY

CIGUATOXIN

- Most prominent symptoms are neurologic
 - Dysesthesias (i.e. hot/cold reversal)
 - “Dental pain” (i.e. tooth are loose or “itch”)
- Small percentage with bradycardia / hypotension



Basnis, AM J Trop Med Hyg, 1979



SCOMBROID



Fugu

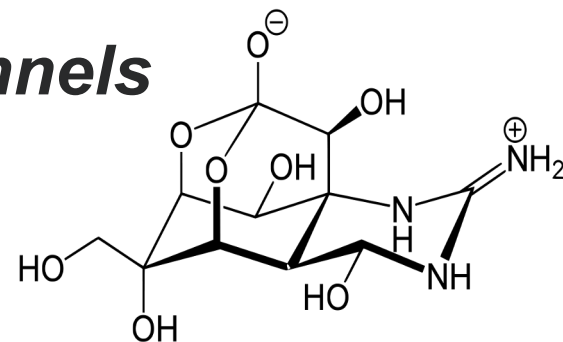


*Hapalochlaena
maculosa*

TOXICITY

TETRODOTOXIN

- Highest concentrations in skin, liver, intestine, ovary
 - Female more toxic than male
 - **Heat stable** (unless $>212^{\circ}\text{F}$ IN ACID), water soluble
- ***Blockade of voltage-gated Na^+ channels***
 - Blocks axonal neurotransmission
 - PNS, CNS, autonomic, cardiac



SYMPTOMS

TETRODOTOXIN

- **GI within 3 hours**
- **Neurologic minutes to hours**
 - Progressive paresthesias of lips, tongue, mouth, face, fingers, and toes
 - Progressive weakness (bulbar), ataxia
- ***Ascending paralysis, respiratory paralysis***
 - Preserved mental status
 - Death reported within 17 minutes!
 - Survival beyond 24 hours excellent prognosis

Shellfish poisoning	Toxin	Mechanism	Symptoms
Paralytic	saxitoxin	blocks Na ⁺ -channels	rapid neurologic
Neurotoxic	brevetoxin	stimulate Na ⁺ -channels	GI / neuro simult No paralysis
Amnestic	domoic acid	glutamate agonist	rapid onset memory loss
Diarrhetic	okadoic acid	phosphatase α1 + α2 inhibitor	secretory diarrhea

I know it sucks but...

Learn the
Scientific name

STINGRAY / *DASYATIS SPP*

- Most common stinging fish
- Atlantic / Mediterranean / Indian Ocean
- Typically stay on sand
- Venom apparatus; whip-like spine on dorsal surface
 - Lacerates and envenomates
 - A sheath surrounds the spine and may become embedded in wound



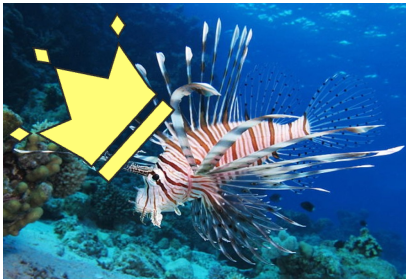
SCORPAENIDAE

- 2nd most common fish envenomations
- Includes (in order of least to most severe)
 - Lionfish (*Pterois spp.*)
 - Scorpionfish (*Scorpaena spp.*)
 - Stonefish (*Synanceja spp.*)



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SEA SNAKES

Common Sea Snake

Enhydrina schistosa



SEA SNAKES

- Small front fangs, dry bites common
- Typically minimal pain at bite, but painful rigidity can occur
 - Symptoms develop in minutes up to 6 hours
- **Results in neurotoxicity, myotoxicity, hemolysis**
- **Hallmark: Painful muscular rigidity, myoglobinuria followed by ascending paralysis**
 - NMJ blockade from pre- and post-synaptic ACh receptor effects
 - Other symptoms: dysphagia, ptosis, n/v, fasciculations, seizure, coma

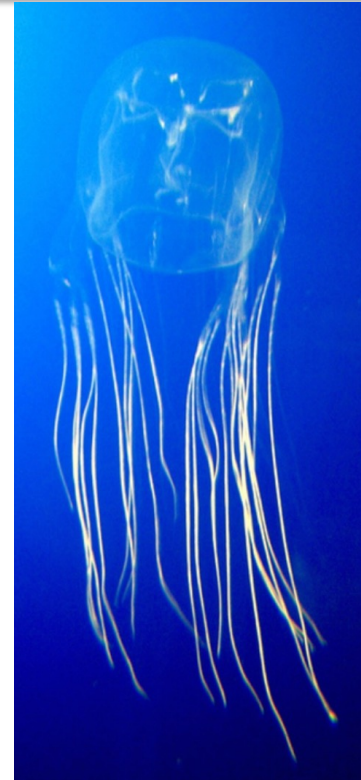
CNIDARIA: TREATMENT

- Most respond well to warm water or vinegar
- **World-wide: Vinegar**
 - *Chironex fleckeri* (Box jellyfish)
 - *Carukia barnesi* (Irukandji)
- **N. America: Hot water/sea water**
 - *Physalia physalis* (Portuguese man of war)
- Antihistamines/steroids prn
- No prophylactic ABX



BOX JELLYFISH / *Chironex fleckeri*

- Most victims with severe pain only
- Wounds may become necrotic
- May develop acute and/or delayed hypersensitivity
- Severe: Hypotension, cardiac arrhythmias, respiratory failure, anaphylaxis
 - Cardiotoxin: pore-forming toxin with increased Na⁺ permeability in cardiac tissue
- Death more common in children, occurs fast
- **Sheep derived whole IgG AV in Australia**



Things with Antivenom

1. *Syncaceja spp.* (stonefish)
2. *Enhydrina schistosa* (common sea snake)
3. *Chrionex fleckeri* (box jellyfish)

IRUKANDJI JELLYFISH / *Carukia barnesi*

- Peanut-sized, translucent jellyfish
 - Australia's north coast, Pacific, Florida
 - Relative of the box jellyfish
- Catecholamine surge, with cardiac and pulmonary effects, death may occur
 - Massive vasopressor response
- No antivenom available



SEA BATHER'S ERUPTION (AKA "SEA LICE")

- Most prevalent between March-June in SE Florida
- Larvae of Thimble Jellyfish *Linuche unguiculata*
- Pruritic, discrete, closely spaced papules, pustules, vesicles, urticaria in areas covered by bathing suit
 - New lesions continue to appear over 72 hours
- Symptoms resolve spontaneously hours to days, up to 2 weeks

BLUE RINGED OCTOPUS / *Hapalochlaena maculosa*

- Mostly in Indo-Pacific region, especially off Australia
- Toxin is **tetrodotoxin**
 - Also found in puffer fish, porcupine fish, sunfish, rough skinned newts, some frogs, and some salamanders
 - Blocks Na^+ conductance resulting in paralysis
- Paralysis, hypotension common



Questions?

ACMT BOARD REVIEW COURSE

Radiation Emergencies & Radionuclides

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Assistant Medical Director, Georgia Poison Center

Director, Grady Occupational and Environmental Toxicology Outpatient Clinic

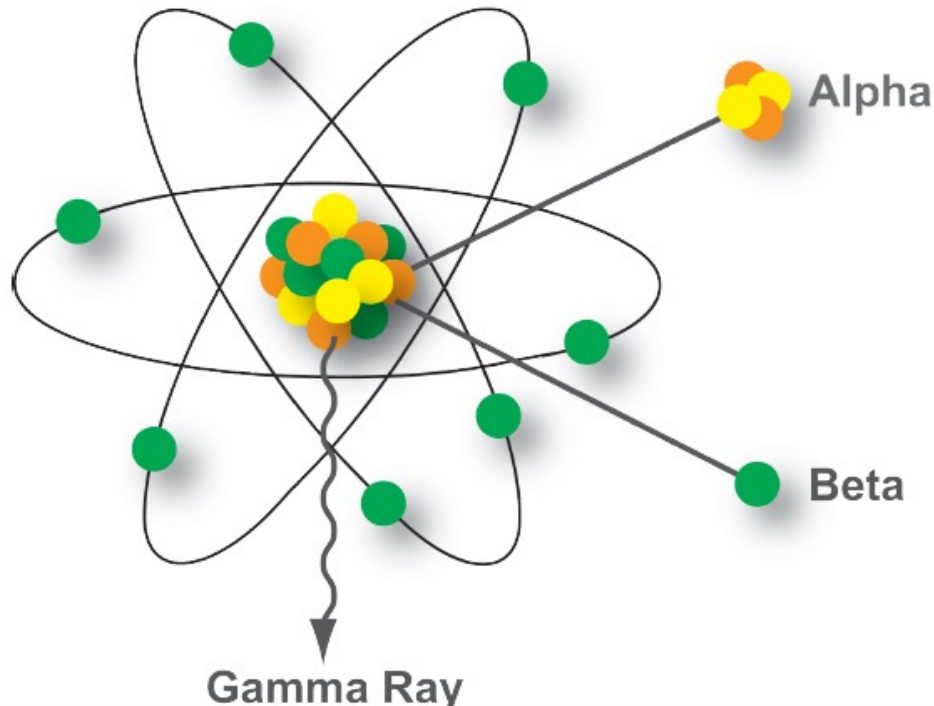
Associate Medical Director, Southern Regional Disaster Response System

Immediate Past-President, MENATOX

Secretary Treasurer, ACMT



Why do Certain Atoms Emit Radiation and are Radioactive?



- Radioactive atoms are unstable
- They release energy to become stable
- The energy can be in the form of gamma rays or particles (alpha or beta)
- This is radioactive decay
- Neutrons are released during a fission reaction

What is a Radiation Source Activity?

- Activity reflects how radioactive a source is
- How many disintegrations or decays are occurring every second
- As time passes, a radioactive source is no longer radioactive
- The amount of time needed depends on the source radiological half life
- Biological half-life: what the body does to the radionuclide
- Effective half-life combines the effects of radioactive decay with biological excretion or elimination of the radionuclide

Deterministic versus Stochastic

- Deterministic
 - Threshold dose
 - The higher the dose received the more severe the disease, the more rapid the onset of manifestations and the more compressed the time course
 - Examples: Local radiation injury or Acute radiation syndrome
- Stochastic
 - Random
 - No threshold
 - The higher the dose received the more likely the disease. The disease is not more severe, however
 - Example: Oncogenesis

4.1.5 Radiation exposure management

- Time
 - Dose received is proportional to the duration of exposure to radiation
- Distance
 - Dose received is inversely proportional to the square of distance
- Shielding
 - Concrete cement or steel
 - Lead aprons not effective against gamma rays. Effective against X-rays
- Quantity
 - Place contaminated materials like clothing away from work areas and limit access to the area. Place warning signs
- ALARA: As Low As Reasonably Achievable

4.1.5.2 Decontamination

- Does not precede life-saving interventions
- Soap and water
- Out to in, meticulous and guided by radiation survey
- Start with contaminated areas that can lead to internalization (i.e., wounds and face)
- In a mass casualty incident, victims may need to self decontaminate by showering

Available Therapies for Internal Contamination

Radionuclide	Medication
Iodine	Potassium Iodide (KI)
Transuranics such as Plutonium & Americium	Zn-DTPA Ca-DTPA
Uranium	Bicarbonate
Cesium Rubidium Thallium	Prussian Blue* [Ferrihexacyano- Ferrate (II)]
Tritium	Water

Acute Radiation Syndrome (ARS) is a Deterministic Effect of Radiation

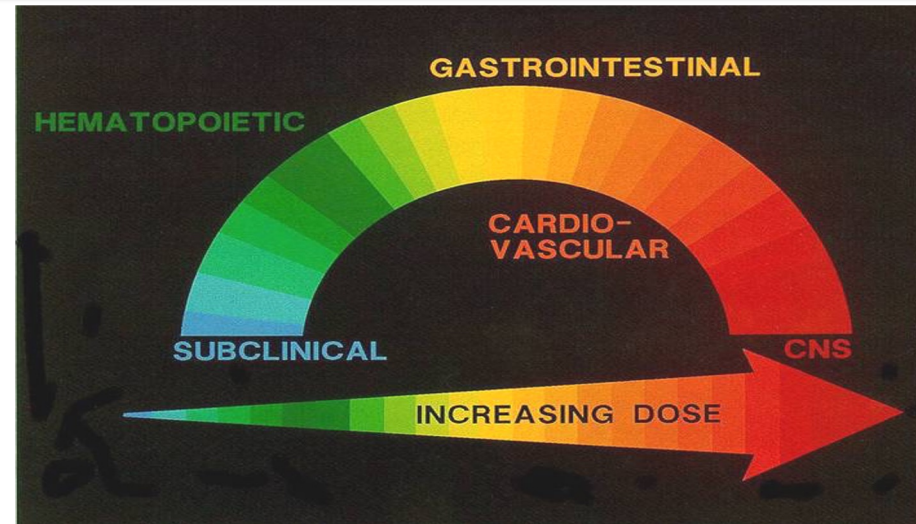
The Rule of 4s:

4 stages

- Prodrome
- Latent
- Manifest
- Recovery or death

4 Conditions

- Exposure to penetrating radiation like gamma rays
- Whole body exposure or near whole body
- Dose ≥ 2 Gray
- Dose received over a short period of time (minutes to few hours)



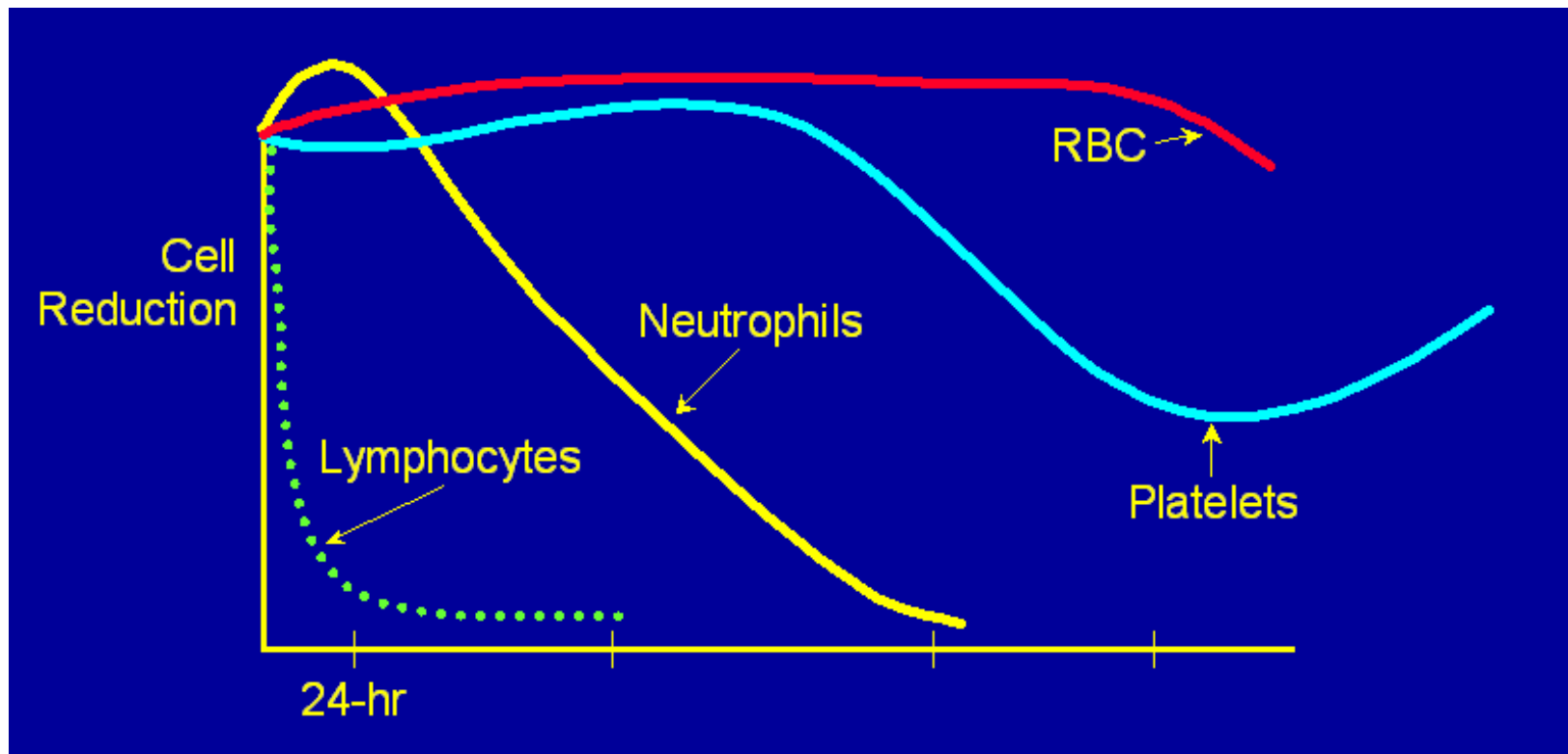
4 Potential Subsyndromes

- Hematopoietic subsyndrome
- Gastrointestinal subsyndrome
- Cerebrovascular subsyndrome
- Cutaneous subsyndrome

Prodromal Manifestations and Dose Estimation

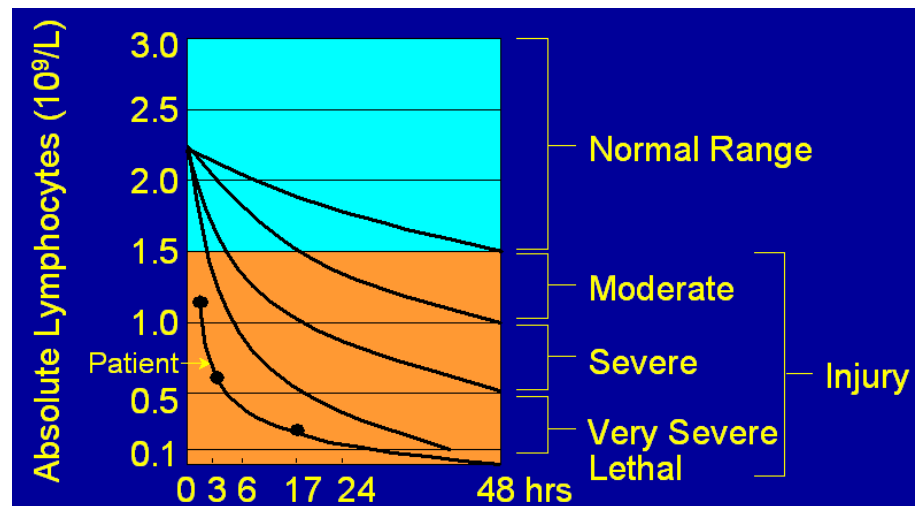
- Vague manifestations: nausea, vomiting, headache, fatigue, diarrhea, fever
- Help predict the dose: the higher the absorbed dose the earlier and the more frequent the manifestations occur
- Time to onset of vomiting can be used to estimate radiation dose
 - The earlier the onset of vomiting the higher the dose received
 - Patients experiencing a time to vomiting less than 4 hours after their exposure should receive immediate medical care, and those that vomit in less than 1 hour often die.
 - Patients who vomit after 4 hours will require less urgent care.
- Diarrhea onset within 1 hour of exposure is a bad prognostic factor

Hematopoietic Syndrome (2-6 Gy)



Lymphocyte Depletion Kinetics

- Lymphocyte depletion kinetics (Andrew's nomogram) helps estimate the dose of radiation.
- WBC with differential every 6 hrs for first 24-48 hours.



Cutaneous Radiation Injury

- Delayed onset
- May be accompanied with systemic manifestations of ARS and other subsyndromes
- The higher the dose received the more severe are the clinical manifestations and the earlier the onset
- May be divided into several types:
 - Erythema
 - Epilation
 - Dry desquamation
 - Wet desquamation
 - Necrosis
- Protracted treatment course (antimicrobials, analgesia, surgical debridement) in addition to the management of other complications like the hematopoietic subsyndrome