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CHEMICAL & RADIATION EXPOSURE



Objectives



Not given (Yup, it is the norm!)

This lecture includes Dr. Tawfiq's notes regarding Inhaled Toxins; chemical exposure lecture and Inhaled Toxins covered the same topics.



NOTES EXTRA BOOK IMPORTANT GOLDEN NOTES

Chemical Exposure



Simple Asphyxiants VS Pulmonary Irritants:

Two mechanistically distinct groups of xenobiotics are capable of interfering with gas exchange:



Simple asphyxiants

It means there is no oxygen. There is another gas replacing the oxygen.

Pulmonary irritants

When there's replacing of oxygen gas by another gas causing damage to mucous membrane.



when you get exposed to a gas, what happens?
 If the gas is highly water soluble the symptoms will appear very fast and it will affect the mucous membrane and cause oral pain and rhinorrhea. The effect is faster and more to the proximal and superficial tissue, but the deep damage is less because it does not cross the mucous membrane.
 If it's a low molecule it will go down to the lung and you will not have these symptoms fast but later you will have acute lung injury.

Toxic Combustion Products

Asphyxiants

Simple Asphyxiants

-Carbon dioxide

Chemical Asphyxiants

-Carbon monoxide
 -Hydrogen Cyanide
 -Hydrogen Sulfide
 -Oxides of nitrogen (Methemoglobinemia)

Irritants

High water solubility
 (Upper airway injury)

-Acrolein
 -Sulfur dioxide

-Ammonia
 -Hydrogen chloride

Intermediate water solubility
 (Upper & Lower airway injury)

-Chlorine

-Isocyanates

Low water solubility
 (Pulmonary parenchymal injury)

-Oxides of nitrogen

-Phosgene

Simple Asphyxiants



Overview:



- Generally speaking, **have No pharmacological activity.**
- Displace oxygen from ambient air.
- $FiO_2 < 21\%$. (FiO_2 = Fraction of inspired Oxygen)(Simple Asphyxiants lower it)
- Decrease partial pressure of O_2 .
- Asphyxiation requires **high concentration** in a **closed space**.

Clinical Features:

Mainly symptoms of hypoxia. (Variation of symptoms correlate with the changes of FiO_2)

FiO_2 At sea level barometric pressure appropriate adjustments must be made for altitude and depth exposure

FiO_2	Signs/Symptoms
21	None
16 - 12	Tachypnea, hyperpnea, (resultant hypocapnia), tachycardia, reduced attention and alertness, euphoria, headache, mild incoordination The patient will be tachypneic and tachycardic because the body is trying to transport the oxygen to the tissues.
14 - 10	Altered judgment, incoordination, muscular fatigue, cyanosis
10 - 6	Nausea, vomiting, lethargy, air hunger, severe incoordination, coma
< 6	Gasping respiration, seizure, coma, death

Types of Simple Asphyxiants:

SA=Simple Asphyxiant

Noble Gases	Helium	SA
	Neon	SA
	Argon	SA
	Xenon	SA, Anesthetic properties
Short chain aliphatic hydrocarbon Gases	Methane (CH_4)	SA, Natural gas (fuel)
	Ethane (C_2H_6)	SA, Natural gas (fuel), refrigerant
	Propane (C_3H_8)	SA, Fuel, Solvent
	Butane (C_4H_{10})	SA, Fuel, Solvent
Nitrogen	SA, Nitrogen narcosis (Similar to anesthetic gases)	
Carbon dioxide	SA, but also causes systemic toxicity	

Treatment:

- **Immediate removal from exposure.** The first thing you need to do is remove the patient from the place before you resuscitate him.
- Ventilator assistance
- Supplemental oxygen
- Supportive care as needed



Pulmonary Irritants

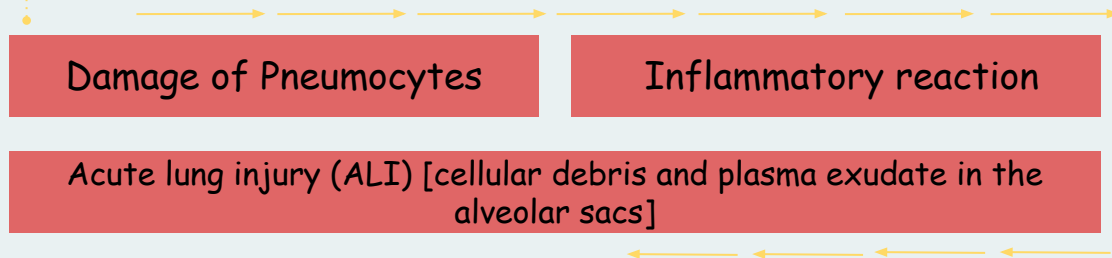


Overview:



The irritant gases are a heterogeneous group of chemicals that produce toxic effects via a final common pathway: the destruction of the integrity of the mucosal barrier of the respiratory tract

Follow the arrows to reveal the sequence of pathophysiology in irritants!



Types of Pulmonary Irritants:

High water solubility	Intermediate water solubility	Poor water solubility
Ammonia, hydrogen florise, sulfur dioxide.	Chlorine, hydrogen sulfide (rotten eggs smell).	Phosgene (used in WWI), oxides of nitrogen, ozone.
-Affect upper A/W "airway" (oral, nasal, pharyngeal, conjunctival mucosa) -Irritation within seconds.	-Upper and lower respiratory symptoms -Symptoms develop after several hours -H2S inhibits mitochondrial respiration (similar to cyanide)	-Affect lower A/W "airway" more (although in large concentrations they produce significant upper A/W (airway) symptoms) -Delayed onset of symptoms.

Management:

<ul style="list-style-type: none"> A,B,C,D Protect the A/W (airway) Limit secretions Oxygen supplementation, bronchodilators ?Corticosteroids: may improve oxygenation, no specific benefit, small risk of harm. If all the previous steps didn't work then we give them steroids 	For Acute lung injury (ALI):		Neutralization Therapy: nebulized 2% sodium bicarbonate may be beneficial in patients poisoned by acid-forming irritant gases
	PEEP (Positive End-Expiratory Pressure)	Prone ventilation	Antioxidants: Ascorbic acid, NAC "N-acetylcysteine" (negligible benefit)
	Inverse ratio ventilation	Low tidal volume	Perfluorocarbon Partial Liquid Ventilation: improve oxygenation and may have anti-inflammatory effect
			Exogenous Surfactant: no benefit in RCTs "Randomized Controlled Trials"

It is good to remember this part also for Forensics (;



Carbon Monoxide (CO)



- The leading cause of poisoning morbidity and mortality in the United States. *Any fire can lead to CO intoxication even if the fire didn't contain smoke*
- Hemoglobin has 200-250 times greater affinity to CO than O₂.
- CO molecular weight = 28.01 daltons / gas density = 0.968 (air = 1.0)
- Blood carboxyhemoglobin level:

Nonsmokers	1-2%	Smokers	5-10%
Action level	>10%		



CO Poisoning:

Half-life	Pathophysiology	Clinical Manifestation	Sources
-Room air → 300 minutes (5 hours) -100% O ₂ → 60 minutes -HBO ¹ → 30 minutes <small>The half life here is based on room air. Why do we care about half life? Because based on the half life we need to reduce the CO and replace it with O as soon as we can.</small>	-CO binds HGB ² rendering it incapable of delivering O ₂ to the cells. Leftward shift of the oxyhemoglobin dissociation curve decreases offloading of O ₂ to tissues (decrease 2,3- BPG ³) -Interferes with cellular respiration by binding to mitochondrial cytochrome oxidase -Lipid peroxidation, particularly in the hippocampus and corpus striatum	-CVS symptoms: Cardiac dysrhythmias, Chest pain, Myocardial ischemia -Respiratory Symptoms: Dyspnea, Tachypnea -CNS symptoms: Ataxia, confusion, dizziness, headache, syncope -Others: Nausea, Visual blurring, vomiting, weakness	-Camp stoves & lanterns -Charcoal grills -Natural gas combustion furnaces (water heaters, ranges and ovens) -Methylene Bromide (it will get metabolized into CO) -Fire is the most common source Dr talked about these three only (for the full list click here)

1-Hyperbaric Oxygen
2-Hemoglobin
3-2,3-Bisphosphoglyceric acid



Investigations:

COHb level (Most useful)

ECG, monitor

Because hypoxia can cause MI

VBG, Lactate, CK, Troponin, BNP, +/- Echo. We check Lactate and CK here to evaluate the muscle not the heart.

Neurologic exam and mini mental state exam

CT as needed

We do a CT if there is CNS manifestation because CO can cause ischemia

[\(CT image\)](#)



Treatment:

1 **Removal from site of exposure**

2 **ABCD**

3 **O₂ (100% ASAP) Mainstay of treatment (antidote)**

4 **Supportive care (IV fluid for hypotension, standard ACLS PRN)**

5 **Hyperbaric Oxygen (HBO)**

Suggested indications for HBO:

-Syncope (Loss of consciousness)
-Coma
-Seizure

-Altered mental status (GCS <15) or confusion
-Carboxyhemoglobin >25%
-Abnormal Cerebellar function




-Age ≥ 36 years
-Prolonged Co exposure (≥24 hr)
-Fetal distress in pregnancy

It is good to remember this part also for Forensics (:

{ Cyanide (CN) }

ppm = parts per million. PPM is a term used in chemistry to denote a very, very low concentration of a solution.

Concentration of Cyanide is important because it will determine the outcome; if you are exposed to a concentration of 270 of CN, you will just die once you inhale it (:

-  Molecular weight = 26.02 daltons
-  Whole blood < 1 microgram/mL (38.5 micromol/L)
-  Concentrations: airborne

Immediately fatal

270 ppm

Life threatening

110 ppm
(>30 mins)



CN Poisoning:

Pathophysiology	Exposure	Clinical Manifestations (image)
<p>-Cyanide is an inhibitor of multiple enzymes, including succinic acid dehydrogenase, superoxide dismutase, carbonic anhydrase, and cytochrome oxidase (Yuupp, same as CO! Just remember this, CN and CO are twins and no one can separate them! Any patient with history of smoke exposure, must be checked for these two agents)</p> <p>-Doctor explained this illustration but said it is for your information but focus on the two red boxes (: CLICK</p> <p>-In cellular respiration there is many enzymes that are essential for generating energy, some of the toxins act on these enzyme to disturb this process therefore we will not be able to either get the ATP so switch to anaerobic pathway (lactic acid; acidosis) or won't be able to use Oxygen (hypoxia)</p>	<ul style="list-style-type: none"> -Fire in a closed space (Plastic burn) -Chemistry labs -Plants (Cassava, apricot seeds) -Iatrogenic (Nitroprusside) -Photographic industries -Jewelry manufacturing 	<ul style="list-style-type: none"> -CNS: Altered level of consciousness, Seizure, Anxiety, Headache, Agitation -CVS: Hypotension, Tachycardia, Arrhythmias. (May have transient hypertension and tachycardia) -Respiratory: SOB, Tachypnea. (Later hypoventilation and pulmonary edema) -Metabolic: Lactic acidosis (classically, lactate more than 10). (Needs VBG with Carboxyhemoglobin) -Most characteristic to CN toxicity: (always look for them in mcq!) Unconscious, hypotensive, and high lactate

Treatment:

Almond tree ->
CN toxicity causes bitter almond odor

ABCDE

Antidote (almost always empirical)

Supportive care

-Hydroxocobalamin (5 g IV during 15 minutes for adults and 70 mg/kg IV for children, up to an adult dose)
-Cyanide + hydroxocobalamin = Cyanocobalamin

Cyanide antidote kit [see here:](#)
-Amyl nitrite
-Sodium nitrite
-Sodium thiosulfate

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Others

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For all the chemicals instead of metals
the treatment is water, water water!

Chemical	Clinical Manifestations	Treatment
<p>Hydrofluoric Acid (Etching glass, removing rust, and cleaning cement and bricks)</p>	<p>Severe painful burn, decrease Ca, Mg, arrhythmias (image for symptoms)</p>	<p>Irrigation with water at least 15-30 min, remove blisters, Calcium gluconate (2.5%) gel, SC Ca, IV /IA Ca, regional anesthesia</p>
<p>Formic acid (used in industry and agriculture)</p>	<p>coagulative necrosis, acidosis, hemolysis and hemoglobinuria.</p>	<p>Copious wound lavage, sodium bicarbonate for $\text{pH} < 7.30$, exchange transfusion, HD (hemodialysis)</p>
<p>Anhydrous ammonia (Fertilizers, manufacture of explosives, petroleum, plastics, and synthetic fibers), Meth dry cook</p>	<p>Can freeze any tissue, chemical burns by liquefaction necrosis</p>	<p>Prompt irrigation of the eyes and skin with water and management of inhalation injury</p>
<p>Cement</p>	<p>Three types of cement burns; The most common is a chemically abrasive form, heat-related or blast burns can occur</p>	<p>Copious irrigation after all clothes have been removed. Early excision and grafting are often necessary</p>
<p>Phenol and derivatives (widely used in the agricultural, cosmetic, and medical fields)</p>	<p>Coagulative necrosis, stimulation, lethargy, seizures, or coma. Tachycardia/bradycardia</p>	<p>Irrigated with large volumes of water delivered under low pressure. ACLS (advanced cardiac life support) PRN (as needed)</p>
<p>White Phosphorus (munitions manufacturing, in fireworks, methamphetamine production, fertilizers, rodenticide)</p>	<p>profound thermal injury, hypocalcemia and hyperphosphatemia, ECG changes -Three stages: GI, latent, multi-organ failure</p>	<p>Copious water irrigation, decontamination, treatment of electrolyte disturbances, management of the skin burns is as with any other burn wound</p>
<p>Hydrocarbons (fuels, solvents, paints, paint and spot removers, dry cleaning solutions, lamp oil, rubber cement, lubricants)</p>	<p>Aspiration (e.g., coughing, gagging, vomiting, wheezing, tachypnea, or hypoxia). Inhalation (headache, dizziness, nausea, or wheezing). Cardiac sensitization</p>	<p>Removal from exposure site. Supportive care (O_2, Bagonists)</p>

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Others

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For all the chemicals instead of metals the treatment is water, water water!

Chemical	Clinical Manifestations	Treatment
Nitrate and Nitrites	<p>Symptoms depend on methemoglobin conc:</p> <ul style="list-style-type: none"> -20%: headache, anxiety, dyspnea, and tachycardia -40-50%: Confusion, lethargy, and acidosis. -70%: SZ, hypotension, dysrhythmias and death 	Supportive care, Methylene blue
Tar	Burn	Immediate cooling with cold water then removal in the ED using Solvents. Sunflower oil can be used. Antibiotic ointment if in cornea
Elemental metals (lithium, sodium, and potassium)	harmless unless they come in contact with water when violent exothermic reaction occurs	Use mineral oil if available before water. If using water use large amount to limit the reaction

Chemical Warfare Agents (CWAs) [\(image\)](#)

CWA type	Chemical Agents	Method of exposure	Clinical symptoms	Treatment
Nerve Agents	<ul style="list-style-type: none"> -G agents (Sarin, cyclosarin, tabun, soman) -V agents (VE, VG, VM, VR, VX) *VX is the most lethal agent 	Inhalation	SLUDGE: miotic pupils, bradycardia, bronchospasm, bronchorrhea, muscle spasm/fasciculations, weakness, flaccid paralysis, tachycardia, Seizures, respiratory failure	<ul style="list-style-type: none"> -Atropine -Pralidoxime
Blistering Agents	Nitrogen mustard and sulfur mustard (mustard gas)	Inhalation	<ul style="list-style-type: none"> -Acute: Skin, eye and lung damage (pulmonary edema and hemorrhage), erythematous rash, skin blistering -Chronic: Lung damage (COPD, asthma, bronchiolitis obliterans), neutropenia, pancytopenia 	<ul style="list-style-type: none"> -Hydrotherapy -Moist dressing on blisters -Supportive care
Asphyxiants	Carbon monoxide, chlorine, phosgene, Hydrogen sulfide gases	Inhalation	Upper airway distress, skin and eye irritation, fatal pulmonary edema and acute respiratory distress syndrome	Supportive care

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Others

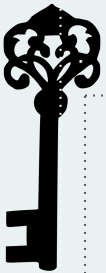
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Chemical Warfare Agents (CWAs) (image)

CWA type	Chemical Agents	Method of exposure	Clinical symptoms	Treatment
Blood Agents	Cyanide	Skin absorption, inhalation and ingestion	Severe distress, tachycardia, cyanosis, hypotension, severe metabolic acidosis, Seizures, cardiac arrest	Cyanide kit: -Amyl nitrite -Sodium nitrite -Sodium thiosulfate Hydroxocobalamin
Hydrofluoric acid	-	Skin absorption, inhalation and ingestion	Severe pain in exposed area, GI distress, vomiting, cardiac arrhythmias, hypocalcemia, hyperkalemia	Irrigation with water, Ca, regional anesthesia and removing blisters



Key Concepts:



- For chemical injury, the degree of skin destruction is determined mainly by the properties of the toxic agent, its concentration, and the duration of contact.
- Chemical injuries are commonly encountered after exposure to acids and alkalis.
- Hazmats are substances that can cause physical injury and can damage the environment if improperly handled.
- In dealing with hazmat incidents, two distinct goals must be achieved: (1) The hazmat must be contained, fire and explosions should eventually be extinguished, and the site must eventually be cleaned, and (2) people exposed to the hazmat must be treated.
- Alkali burns tend to penetrate deeper than acidic burns; as a result, alkali burns tend to be associated with greater morbidity.
- HF burns can be associated with significant hypocalcemia.
- Exposure to various toxic gases can occur from routine industrial settings, and knowledge of these agents is necessary for proper treatment by the emergency physician.
- Unconventional chemical weapons may be categorized into four major classifications; nerve agents, vesicants, choking agents, and cyanide agents.

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Dr. Tawfiq Notes

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General Talk

- Example of cutaneous toxin: organophosphate
- Example of ingested toxins (can be liquid or solid): drug of abuse, منظفات البيت زي الكلوركس والصابون
- Inhaled toxins: any substance goes to the lung and injures the lung with different mechanisms they're the worst, difficult to manage, fatal.
- The respiratory tract is divided to upper and lower which start from the nasopharynx going down to trachea (oropharynx, vocal cords), then divides into right and left, and then bronchioles, then alveoli.
- Toxins cause injury to every mechanism, e.g. when ciliar mechanism that wash gets injured it shut down. Mucus mechanism which get secreted by goblet cells
- (I'll ask you about the basic pathophysiology) (picture): a lot of inflammation edema, mucus production, very bad bronchitis and alveolitis the symptoms he's going to have are severe dyspnea, cough.. ect
- X-ray: Fire victim, typical (ARDS) adult respiratory distress syndrome
- What's the difference between smoke and smog? Smoke is a product of **combustion?** (الاحتراق) while smog is associated with vapor (البخار)
- The smaller the particle (of toxins) the deeper it'll go until it reaches the alveoli

Simple Asphyxiants

- When a gas replaces the oxygen, either by 1. Liquified gas 2. An Apparatus like divers 3. Working in confined place.
- the oxygen percentage in the room now is 21% if it drops down we'll have symptoms. (MCQs) At 15% > cardiac, autonomic, cerebral hypoxia (manifest by confusion, dizziness, incoordination, and seizure sometimes)
- Patient die from hypoxia not hypercarbia
- It's really important to understand that hypoxemia is the main principle of causing mortality
- If O2 sat falls below 10% patient will have cerebral edema (MCQs)
- If O2 sat falls below 6% patient will have immediate cardiac arrest
- The identification of the gas is not important because the treatment is supportive
- High risk patients (elderly, seizure, cardiac) are kept for observation (MCQs)

Pulmonary Irritants

- You inhale them, they go to the lung, react and form new compounds e.g.
1. **chlorine**: (used in swimming pools) if aspired in large amounts, it forms hydrochloric acid in lungs, which is very corrosive to the respiratory tract.
 2. **ammonia**: it forms ammonium hydroxide (base)
 3. **sulfur dioxide**: forms sulfurous acid which reacts and form acid or alkaline which are very irritating and cause a lot of damage
 4. **Phosgene**: a gas used in chemical wars, very very injurious
- You don't have to go in details just example and mechanism of injury
- Pulmonary irritants mechanism:**
1. Dissolve in the mucus of bronchial tree
 2. Induce very intense inflammation
 3. Form new acid or alkaline
 4. Form new oxygen radicals. All these mechanisms cause severe bronchospasm and bronchial inflammation

Weapons of War (Poison Gas)

- Management:**
- Removal from the scene
 - Supportive treatment (oxygenation)
 - Observation
- Until you reach the hospital

- Signs of respiratory distress:**
1. Cyanosis
 2. **Tachypnea**
 3. **Tachycardia**
 4. Pulsus paradoxus
 5. Using accessory muscles
 6. **CNS manifestations**
 7. Coma

Smoke Inhalation

- **How does it kill victims?**
 - 1. thermal
 - 2. Particulate in the soot (very high temperature, 4000 higher than air temperature)
 - 3. Trauma
 - 4. Chemical compounds association e.g. cyanide
 - A picture of a patient with stridor (patient dies in half an hour):
 - 1. black carbonaceous material around the vocal cords
 - 2. Laryngeal edema
 - 3. oropharyngeal edema
- Very difficult patients, mortality is high, usually the only vein preserved for cannulation is axillary vein.
- Management:**
1. Removal from exposure
 2. Supportive (ventilation, bronchodilators)
 3. Sodium bicarbonate (MCQs) you have to suspect cyanide poisoning in every patient with metabolic acidosis, the most important signs are stridor, hoarseness, respiratory distress

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Radiation

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Decay: I want you to get the concept only!

Radioactive decay



Unstable nuclei decay or transform into more stable nuclei (daughters) via the emission of various particles or energy.

In nutshell; there is some SUPER active substances in our world and because they are too generous, they will lose some of their energy into the surrounding as RADIATION (Yes, you who thought physics and chemistry classes are not important since you are going for medicine >_<)



Emission of γ rays

Emission of α particles

Emission of β particles

Emission of positrons

Capture of an electron

These particles form ionizing radiation

5 mechanisms of radioactive decay

$\frac{1}{2}$

Half-life ($t_{1/2}$): the period of time it takes for a radioisotope to lose half of its radioactivity

I want you to get the concept only!

Penetration abilities of different types of radiation: [\(image\)](#) [\(image\)](#)

α

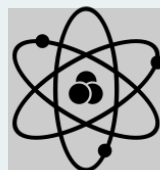
Alpha particles:
Stopped by a sheet of paper
least tissue penetrating ability

β

Beta particles
Stopped by a layer of clothing or less than an inch of a substance (e.g. plastic)

γ

Gamma rays
Stopped by inches to feet of concrete or less than an inch of lead



Neutrons
Stopped by a few feet of concrete

Alpha and Beta are the most important (:

α -particles

- Radioactive decay of an atom nucleus giving off 2 protons and 2 neutrons
- Travel only few centimeters in air.
- Unable to penetrate the outer layer of dead skin
- Causes serious damage when ingested (incorporated)**
- E.g. Polonium-210 [video](#)

β -particles

- Radioactive decay of an atom nucleus giving off an electron or a positron (positively charged electron)
- Able to travel a few meters in air due to small size
- Can be stopped by a piece of plastic or a stack of papers
- Can penetrate the skin a few centimeters
- Causes serious damage when ingested (incorporated)

Photons

- Massless particles that travel at the speed of light and mediate electromagnetic radiation.
- Depending on the energy of the particles, and, therefore, their wavelength
- The radiation has different names:
 - Radio waves: have the lowest energy and the longest wavelength
 - Microwaves: higher energy and shorter wavelength

γ and X-rays

- γ Rays and x-rays are the same and are only distinguishable by their source
- Emission of high-energy wave (photon of energy being emitted) Not a particle
- Since it has no mass or charge, it can travel much farther than α or β particles
- Can be stopped by thick material (lead is the most effective shield)
- An x-ray machine generates x rays by accelerating electrons through a large voltage and colliding them into a heavy metal target



Radiation



Ionizing VS Non-ionizing radiation [See image](#)

Doctor said it is not important >_<



I Ionizing

Any radiation with sufficient energy to disrupt an atom or molecule with which it impacts

Non Ionizing

consists of relatively low-energy photons and is used safely in cell phone and television signal transmission, radar, microwaves, and magnetic fields that emanate from high-voltage electricity and metal detectors.



Stochastic VS Deterministic Effects

Stochastic Effect

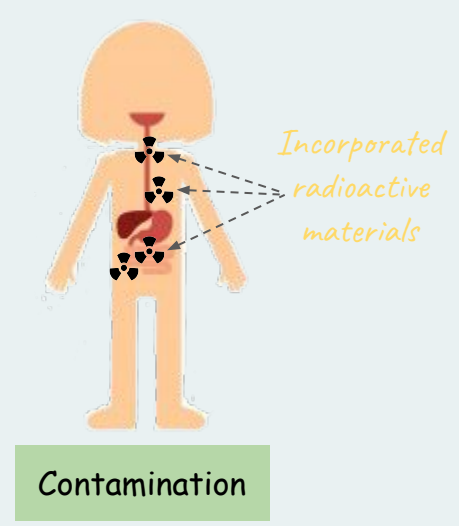
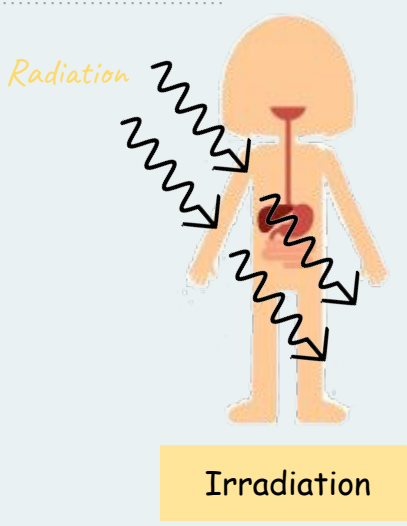
Injuries that do not require a threshold limit to be exceeded. E.g: mutagenic and carcinogenic changes to individual cells where DNA is ultimate target

Deterministic Effect

require a threshold limit to be exceeded. large number of cells of an organ system must be killed before an effect becomes clinically evident

Irradiation: You are the only one who will be harmed.
Contamination (external and internal): You become a potential source for harming others and irradiate them! (It is not only you who will be harmed)

Irradiation, Contamination and Incorporation



"External"

"Internal"



Radiation



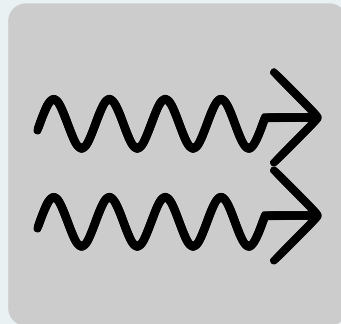
Definitions:

Know general idea about it and get the concept; focus on red (;



Amount of radioactivity (Ci/Bq)

The amount of radioactivity in a radionuclide can be described either by the number of disintegrations per second (the becquerel "Bq"), or by comparing the number of disintegration to that of radium (The curie "Ci").



Amount of radiation emitted (Roentgens)

Particles released during radioactive decay travel in all directions. When Gamma or X-rays ionize the air surrounding a source, an electrostatic charge is produced. This ionization is quantified by the roentgen "R", which is an indirect measure of the amount of radiation.



Amount absorbed by tissue (Rad/Gy)

Most of these particles pass through tissue without being absorbed. Only the fraction of particles that contacts and is absorbed by tissue can cause cellular damage. This fraction is measured in rads or gray (Gy)



Degree of damage (Rem/Sv)

For a given energy, larger particles cause more damage when absorbed by tissue than smaller particles. To predict the degree of damage that a given particle will cause, the dose in Gy or Rad is multiplied by the particle-specific biological effectiveness coefficient (Q) to calculate rem or Sv.



Management:

Decontamination:

- Remove all clothing
- Wash thoroughly with soap and water remove up to 95% of radioactive material
- Carefully scrub open wounds; to minimize the risk of internal contamination
- Use a portable dosimeter
- Collect all clothing and liquid used for decontamination and mark as RADIOACTIVE WASTE

ABCDE

Treat nausea and vomiting
Treat pain (APAP, or opioid)

Serial CBC (biodosimetry)
Electrolytes, Renal and hepatic function

Specific therapy:

- Colony- stimulating factor (3 Gy or greater, 2 Gy if <12 or > 60.
- Probiotics
- Ca-DTPA and Zn-DTPA (decontamination of plutonium, americium, curium, and soluble uranium salts)
- Prussian blue (thallium)
- KI (131I)







Radiation



Prognosis According to Lymphocyte Count within 48 hrs After Acute Exposure to Penetrating Whole-Body Radiation [\(image\)](#)

Minimal Lymphocyte Count (per mm ²)	Approximate Absorbed Dose (Gy)	Extent Of Injury	Prognosis
1400-3000 (Normal range)	0-0.4	No clinically significant injury	Excellent
1000-1499	0.5-1.9	Clinically significant but probably non lethal	Good
500-999	2-3.9	Severe	Fair
100-499	4-7.9	Very severe	Poor
100	8	More Severe	High incidence of death even with hematopoietic stimulation

Acute Radiation Syndrome

	Dose (Gy)	Symptoms and Consequences	Medical Management
 <p>Nausea, Vomiting, Diarrhea (NVD) Syndrome</p>	1-2	Nausea, vomiting, diarrhea, anorexia, giddiness, and loss of appetite.	Symptomatic treatment, antacid, sucralfate, antiemetics
 <p>Hematopoietic Syndrome</p>	2-6	Loss of cellularity in bone marrow, spleen, and thymus. The individual may die between 10 - 30 days without medical intervention.	Antibiotics, cytokines, bone marrow transplant, stem cell therapy .
 <p>Gastrointestinal (GI) Syndrome</p>	8-15	Damage to intestinal crypt cells, loss of absorption of nutrients, dehydration, loss of weight, severe electrolyte imbalance, and low blood pressure. Death occurs usually within 3 - 5 days without medical intervention.	Antibiotics, antiemetics, replacement of fluids and electrolytes, stem cell therapy, bone marrow transplant.
 <p>Central Nervous System (CNS) Syndrome</p>	>25	Irritability, hyper excitability response, epileptic type fits and coma. Symptoms are irreversible. Death usually occurs within 48 hours.	No treatment available.

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Radiation

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This is how they run an X-ray for a baby! (I know it is irrelevant but this is SUPER adorable)

- Three principal risks to a fetus following radiation exposure:
 1. Congenital abnormalities
 2. Mental retardation
 3. Later Development of neoplasm

However, the vast majority of routine diagnostic imaging procedures impart less than 0.05 Sv (degree of damage) to the fetus and so are considered a negligible risk.

Early on (0-2 weeks): risk of fatality

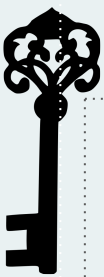
3-7 weeks: congenital anomalies and mental retardation

8-25 weeks: mental retardation

The risk generally reduces at the 16th week.



Key Concepts:



- Externally Contaminated patients are "radioactive"; irradiated patients are not.
- No danger to medical personnel from contaminated patients exists with proper precautions and decontamination procedures.
- Decontamination should not delay or impede the stabilization of the patient in radiation emergencies.
- Careful evaluation of initial symptoms and signs is the most reliable indicator of radiation dose received and patient's **prognosis**.
- Most therapy is supportive and symptomatic except for exposures involving the ingestion or inhalation of radioactive material, when specific therapy with blocking or chelating agents may be indicated.
- Detonation of a "dirty bomb" would cause psychological terror and little or no radiation injuries.
- Formal consultation is available 24 hours a day and should be obtained when any patient with radiation injuries is evaluated.

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Summary

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Simple Asphyxiants	Pulmonary Irritants
<ul style="list-style-type: none"> • Generally speaking, have No pharmacological activity. • Displace oxygen from ambient air. • $FiO_2 < 21\%$. ($FiO_2 = \text{Fraction of inspired Oxygen}$)(Simple Asphyxiants lower it) • Decrease partial pressure of O_2. • Asphyxiation requires high concentration in a closed space. 	<p>Causes destruction of the integrity of the mucosal barrier of the respiratory tract</p> <ul style="list-style-type: none"> • Damage of Pneumocytes • Inflammatory reaction • Acute lung injury (ALI) [cellular debris and plasma exudate in the alveolar sacs]
<p>Sign and symptoms Varies according to the levels of FiO_2</p>	<p>Signs and symptoms vary according to water solubility</p>
<ul style="list-style-type: none"> • Immediate removal from exposure. The first thing you need to do is remove the patient from the place before you resuscitate him. • Ventilator assistance • Supplemental oxygen • Supportive care as needed 	<ul style="list-style-type: none"> • A,B,C,D • Protect the A/W (airway) • Limit secretions • Oxygen supplementation, bronchodilators • ?Corticosteroids: may improve oxygenation, no specific benefit, small risk of harm. If all the previous steps didn't work then we give them steroids
Carbon Monoxide (CO)	Cyanide (CN)
<p>-CVS symptoms: Cardiac dysrhythmias, Chest pain, Myocardial ischemia</p> <p>-Respiratory Symptoms: Dyspnea, Tachypnea</p> <p>-CNS symptoms: Ataxia, confusion, dizziness, headache, syncope</p> <p>-Others: Nausea, Visual blurring, vomiting, weakness</p>	<p>-Unconscious -hypotensive -high lactate</p>
<p>Removal from site of exposure, then:</p> <ul style="list-style-type: none"> • ABCD • O_2 (100% ASAP) Mainstay of treatment (antidote) • Supportive care (IV fluid for hypotension, standard ACLS PRN) • Hyperbaric Oxygen (HBO) 	<ul style="list-style-type: none"> • ABCDE • Antidote (almost always empirical) <ul style="list-style-type: none"> ○ -Hydroxocobalamin (5 g IV during 15 minutes for adults and 70 mg/kg IV for children, up to an adult dose) ○ -Cyanide + hydroxocobalamin = Cyanocobalamin • Supportive care
Radiation and pregnancy	<ul style="list-style-type: none"> • Three principal risks to a fetus following radiation exposure: <ol style="list-style-type: none"> 1. Congenital abnormalities 2. Mental retardation 3. Later Development of neoplasm



How Toxic is Your Knowledge!



1-After radiation exposure injury, which of the following is likely to happen?

- A.Pancytopenia
- B.Thrombocytosis
- C.Hypertension
- D.Hypothermia

2-After radiation exposure, in "survival impossible group" of patients, which of the following is likely to happen?

- A.No symptoms and signs
- B.Severe diarrhea and vomiting
- C.Most of them survive the illness
- D.50% of patients survive the illness

3-Which of the following is the underlying mechanism of tissue injury after radiation exposure?

- A.Deposition of energy in the tissues
- B.Loss of heat from the tissues
- C.Malfunction of cytochrome P450
- D.Excess retention of salt and water in the tissues

4-Which one of the following is the treatment of acute radiation syndrome?

- A.Monitor neutrophils count
- B.Give growth factors to stimulate bone marrow
- C.Calcium chloride
- D.Isolate in a negative room pressure

5- Carbon Monoxide gas is considered to be what type of chemical agent

- A.Vesicant agent
- B.Systemic asphyxiant
- C.Biological agent
- D.Nerve gas

6-X- Ray is under which one of the following rays ?

- A.Alpha particles
- B.Beta particles
- C.Gamma ray
- D.Neutron



Cases from Doctor's slides:

Case 1:

- 25 year-old healthy male. Presented to the ED with history of syncope, H/A, SOB, cough and dizziness.
- 2 of his brothers have similar symptoms
- VS: HR=110 beat/min, RR=20, O2 sat= 100% in RA, temp= 37.0
- ECG: sinus tachycardia, Glucose= normal

This case is not clear and the symptoms can mimic a lot of differential diagnosis, but there is two hints can guide you to CO intoxication which is syncope and 2 of his brothers have the similar symptoms.

Case 2:

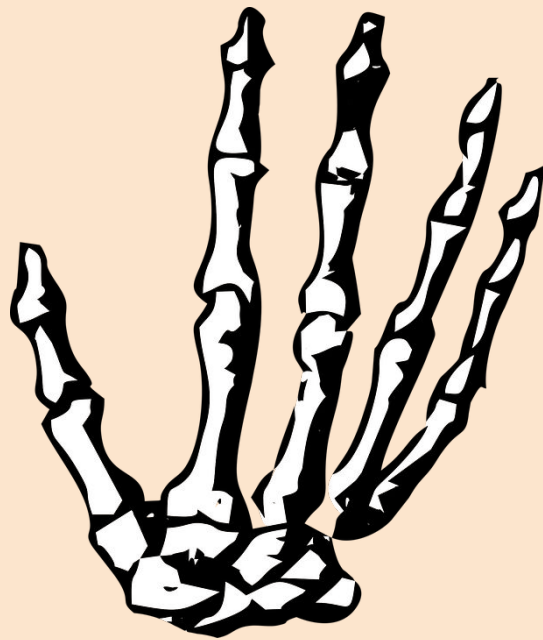
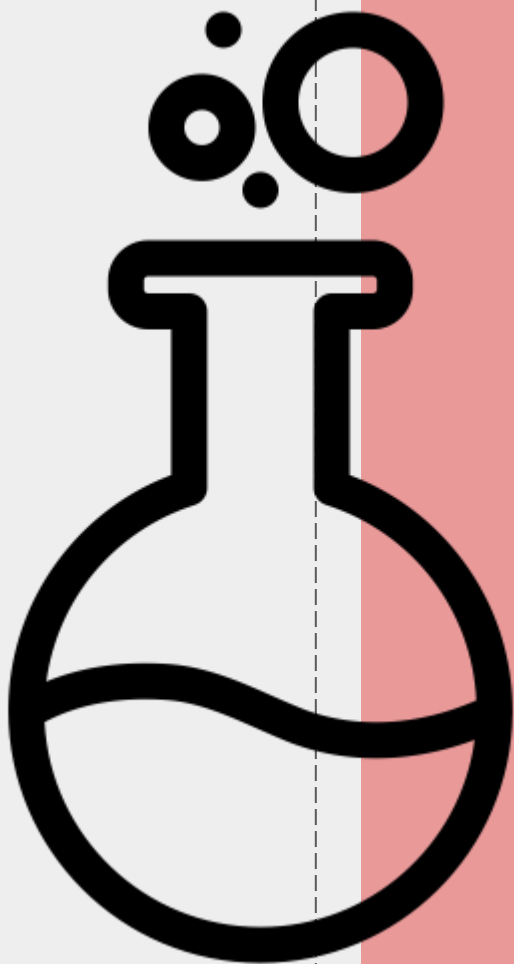
- 30-year-old male. Brought to the ED by EMS following a fire incident at a plastic factory. The patient is unresponsive. His BP= 80/40, HR= 130, RR= 30, O2 Sat is 90% on non-rebreather mask.
- He was intubated on arrival to the ED. Vital signs are unchanged.
- No obvious skin burn, Chest: few scattered wheezes CVS: S1+S2+ no added sounds. Abdomen: normal
- Investigations: VBG: PH= 7.01, PCO2= 30, PO2=60, HCO3- = 5, lactate = 15, CO= 10%

There is hypotension, sever lactate acidosis and the patient is unconscious these three guide you to cyanide intoxication.



- 1-A
- 2-B
- 3-A
- 4-B
- 5-B
- 6-C

THANK YOU AND GOOD LUCK!



VERY TOXIC BUT YOU ARE
GONNA DO IT!

A+ is yours (:

- Email us at:

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How well do you think we have done? We are waiting for your feedback!



Click here!

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