

Poisonous Cargo, Catastrophic Chemistry, and Toxic Terrorism:



Why Should Respiratory Therapists Be Concerned?



Bio and Disclosures

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-No financial or other conflicts to report .

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Course Objectives

- The participant-learner will be able to :
- Identify common hazardous materials and CBRN agents ,their classifications ,mechanisms of action,and the physiochemical properties which influence toxicity,exposure risk,respiratory injury and public health consequences.
- Recognize and explain fundamental toxicology principles ,concepts and terminology . Identify routes of exposure and environmental pathways of exposure .
- Describe appropriate levels of PPE, personal and facility protective countermeasures ,and decontamination procedures.
- Recognize clinical toxidromes of the chemically injured /ill patient and the respiratory manifestations exhibited by hazardous materials and CBRN exposures and outline/describe appropriate antidotal therapy ,respiratory support ,therapeutic and critical care management .
- Explain the importance of triage ,pulmonary diagnostics and pulmonary rehabilitation

My Inspiration My Heroes

Paracelsus- “Father of Toxicology “
“ The dose maketh the poison “



B. Ramazzini- Occupational Medicine
Textbook: De Morbis Artificum Diatriba



The Problem: Why Should WE Be Concerned?

- Hazardous materials are ubiquitous and accidental and intentional releases can occur which would generate adverse health effects such as acute lung damage and chronic pulmonary and/or systemic sequelae .
- Mass chemical exposures in a community will overwhelm local healthcare resources by creating a high influx medical surge and the ability to handle contaminated casualties .
- For respiratory therapy departments, EDs and critical care units ,this would mean overwhelming capacity and capabilities.
- “ Staff,stuff and space “ limits can lead to scarce resource allocation .

The Problem :Why Should WE Be Concerned ? (continued)

- . Toxic Terrorism -The on going threat of toxic industrial chemicals and materials (TICs/TIMs) being used as “ Weapons of Opportunity and Convenience”
- .The possibility that civilian populations may be attacked using military grade CBRN agents ,ie. nerve agents (sarin) ,vesicants (sulfur mustard) or radiological agents (radionuclides) ,RDDs/”dirty bomb” or other methods of dispersal (REMs-radiological emission device)



The Most Dangerous 2 Miles In America

US Army Threat Assessment ,FBI,DHS

New Jersey Turnpike area-Critical infrastructure -highly vulnerable

Potential estimated casualties -2.5 million -densely populated area



SCENARIO I- “ Something’s in the Air.”.

THE PROBLEM: Reports of multiple casualties at City Hall and South Mall .

Many complaints of coughing ,difficulty breathing ,” burning eyes and throat” and bleeding from nose. Caller:” Hey! Hello!! Send help to City Hall! Can’t breathe!”

Simultaneous calls for medical assistance at South Mall food court entrance . Caller:“ Many people gasping for air and having fits !! Lots of ‘em look dead!! Please help us!!”



Toxic Warfare –Chemo-terrorism or.. Meth Lab?



Scenario 2- “ We interrupt this broadcast “....

THE PROBLEM -You are a respiratory therapist on the 7pm-7am shift at a Community Hospital .

As you are assessing a child in the ED for an acute exacerbation of his asthma, you learn that the Polygon Plastics and Chemical Corporation facility located by the highway ,approximately 13 miles from the hospital, has experienced a “catastrophic event” involving an exothermic reaction and explosion with several workers injured and experiencing “severe symptoms “ .

The county’s Mass Casualty Plan has been activated ,and the first EMS units begin arriving at your facility with patients ,including first responders exhibiting S&S of acute respiratory distress .

Scenario 2-(cont'd)



A Sentinel Event: Donora Pennsylvania ,1948



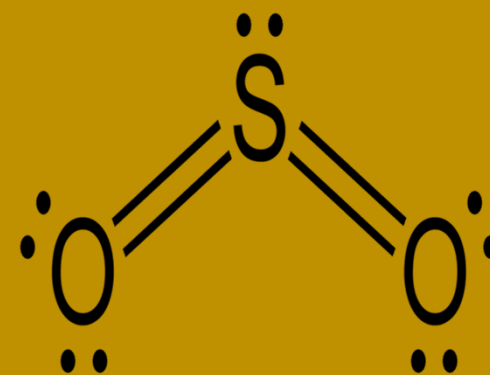
The Sentinel Event

Date: 27 October 1948- pop. 14,000

- Severe air pollution disaster caused by a temperature inversion ,toxic industrial emissions,and a stagnant weather front . H_2SO_4 , HF aq, NO_2 , heavy metal PM
- 20 people were killed immediately ,7,000 residents with severe dyspnea,bronchospasm, pulmonary edema, chest pains ,and severe coughing .



KILLER SMOG

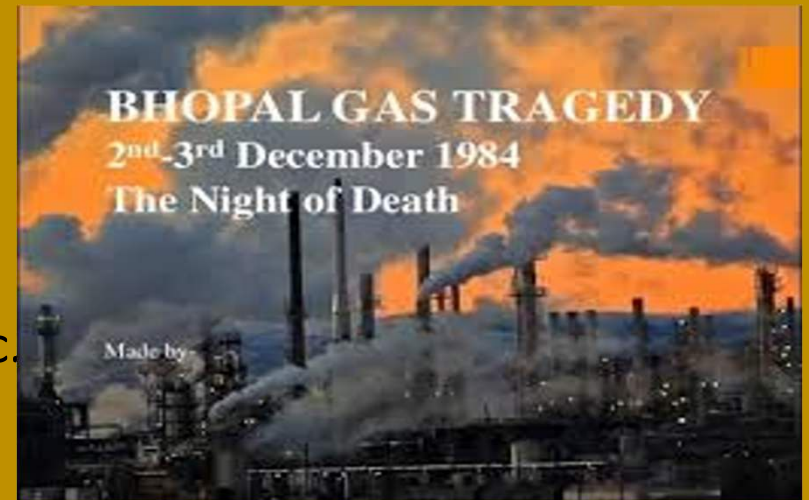


2 -3 DECEMBER 1984: CATASTROPHIC INDUSTRIAL CHEMICAL RELEASE,BHOPAL

- “ Union Carbide carbaryl insecticide (Sevin) plant
- “Sentinel event for chemical- sector safety and security reviews and policy-making/ emergency planning for HAZMAT events.(Emergency Planning & “Right to Know”)
- “Release occurred late night ,many residents asleep, warning siren silenced by plant operators to “ avoid panic”. Many crucial safety systems failed.
- “ Emergency planning and preparedness for major chemical incident non-existent.
- “Over 40 metric tons of product released into atmosphere resulting from a runaway exothermic reaction caused by water incursion into a plant reaction v
- “ Over 40 metric tons of product released into atmosphere resulting from a runaway exothermic reaction caused by water incursion into a plant reaction vessel . One consultant report indicated sabotage by disgruntled employee .
- “Methyl isocyanate and phosgene gas were released ,both potent pulmonary toxicants induce pulmonary edema.

Pulmonary Agents: Case Study

- “Bhopal ,India Methyl Isocyanate Release (cont'd)
- “Resulted in over 3,000 immediate deaths
- “~500,000 injuries and toxicant- induced illnesses
- “Mass casualties from respiratory distress, acute respiratory failure, toxicant-induced ocular injury, etc.
- “ Mass psychological impact, PTSD
- “After 35 years post-event : chronic health effects , including many congenital defects, cerebral palsy, chronic respiratory respiratory dysfunction and many disabilities
- “Data may be applied to model intentional toxic industrial chemical releases and impacts



Pulmonary Agents: Case Study

..5 January 2005, 0240, EST – Graniteville, South Carolina chlorine tanker release

- ..Released approximately 60 tons of chlorine
 - 851 treated for chlorine exposure
- ..72 people hospitalized
- ..15 admitted to area ICUs
- ..5,400 residents evacuated, 7,000 told to shelter in place
- ..9 deaths
- ..Delayed identification of chlorine for almost one hour
- ..Many in community left with chronic health problems, including PTSD, to present day
- ..Some authorities suspected sabotage/terrorism
- ..***Plausible scenario for a chemo-terrorism event***
- ..

Reference: *Railroad Accident Report RAR-05-04-NTSB*
(www.nts.gov)



Case Study :“The Cyanide Canary”

THE INCIDENT

27 August ,1996 at a fertilizer and waste-reprocessing facility in Soda Springs ,Idaho.

THE VICTIM

Twenty -year-old worker Scott Dominguez was ordered to clean a 250-gallon storage tank.

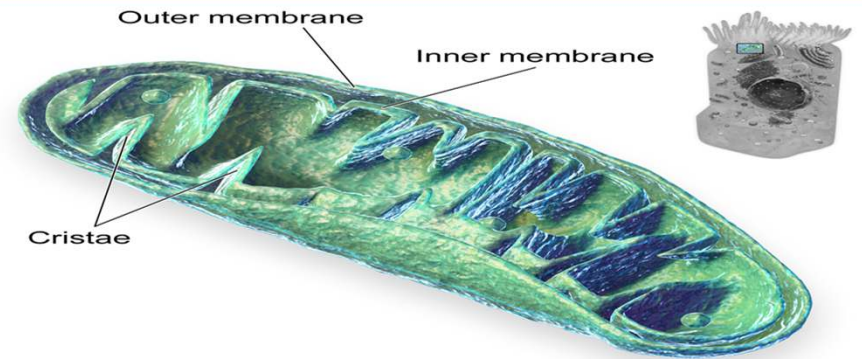
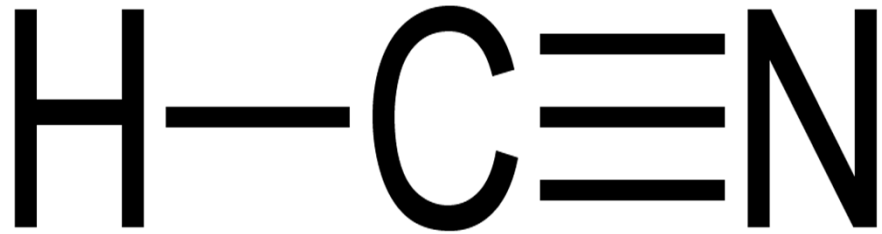
THE DANGER

Plant owner Allan Elias told workers the tank contained only mud and water and required no safety equipment .In reality ,it was laced with lethal cyanide.

Case Study-"The Cyanide Canary "-(cont'd)

THE RESULT

Dominguez was overcome by toxic vapors (HCN) suffered toxicant induced injury by disruption of cellular oxygen utilization caused by inhibition of mitochondria enzyme (cytochrome oxidase) activity and ATP energy production (*histotoxic hypoxia*) leading to cerebral anoxia and catastrophic ,permanent brain injury.



Mitochondria



Tokyo Subway Attack

- **March 19, 1995 --Monday morning rush hour, in Tokyo, has disintegrated into chaos, according to Japanese news and government sources. Reports of toxic fumes in a subway station began at approximately 08:17 a.m. (Tokyo time) and quickly escalated into a major mass casualty incident. According to the Tokyo Metropolitan Police, as many as five-hundred sixty-five (565) people were hospitalized and five people were confirmed dead.**
- **The exact nature of the chemical involved remained a mystery at the time of this original report:**
 - A. **Initial reports, attributed to the Tokyo Fire Department, said that the poisonous substance was acetonitrile, an extremely toxic substance used as a fumigant and in metal treatment and photo processing.**
 - B. **The next report, from the police department, said that there two boxes that were believed to contain Methyl Cyanide, also a very toxic chemical, that can cause rapid injury and death.**
 - C. **The latest, unconfirmed, report speculates that a nerve gas called Sarin may be the cause of the tragic occurrence.**

Nerve Agent -Case Study

20 MARCH,1995-TOKYO SUBWAY SARIN ATTACK- A CLASSIC CASE STUDY

“A 30% pure formulation of sarin was transported onto subway trains within the Tokyo subway system in plastic bags during AM rush hour.

“The bags were punctured using sharpened umbrella tips by a team comprised of members of the apocalyptic religious cult ,”Aum Shinrikyo”, i.e. ”Supreme Truth” (now known as “Aleph”).

Since sarin is the most volatile (high vapor pressure) of all of the nerve agents, the vapor was able to permeate the confined space of the subway system creating a highly toxic inhalation /vapor hazard.

Resulted in mass casualty event with contaminated casualties and responders
Within 1st hour 500 casualties ,including “worried well” presented at St. Luke’s International Hospital ED. ~ ***5,500 presented to area HCFs within 24 hours.***



Nerve Agent -Case Study (Cont'd)

TOKYO SUBWAY SARIN ATTACK CASE STUDY (CONT'D)

- 13 individuals died, 6300 taken ill (included psychogenic effects ,anxiety reactions, the “worried well”)
- Public safety and healthcare resources overwhelmed
- Cross-exposures, contamination and toxic effects to several public safety, transit workers and healthcare providers
- Multi-focal ,coordinated attack on five subway cars ,three separate train lines
- Preceded by a smaller attack in Matsumoto in 1994
- Planning, logistical and operational errors by authorities



Nerve Agent-Case Study

“ CLOSE ENCOUNTERS OF A DEADLY KIND”

“The Skirpal “Novichok” poisoning case –March 2018

“Sergei and Yulia Skirpal –father and daughter in
Salisbury ,England 2018

-Former KGB Colonel

-Novichok (“Newcomer”) nerve agent was sprayed on door
knob of home

-The Skirpals survived after weeks of
intensive care

-Attack resulted in cross-contamination
and death of British citizen and exposure of others



East Palestine, Ohio Train Derailment

3 February, 2023 - Norfolk Southern Railway Company freight train hauling vinyl chloride and several other TICs derailed adjacent to several residences and businesses.

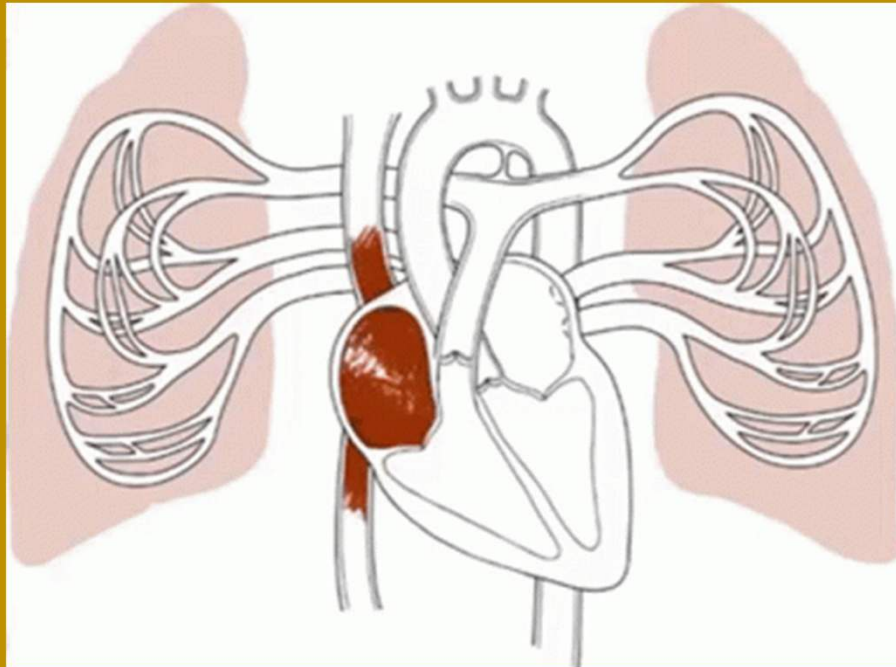
Resulted in the release of several TICs into the environment and a major chemical fire .

Vinyl chloride ,a potent respiratory and ocular irritant-pneumotoxificant and carcinogen was released creating serious health effects .

HAZMAT personnel from the railroad company along with local emergency responders decided to rupture tank cars and drain products into a trench and then ignited the toxic and flammable products generating a toxic cloud that affected three states . This was done to avert a catastrophic “BLEVE”.I

Respiratory Anatomy

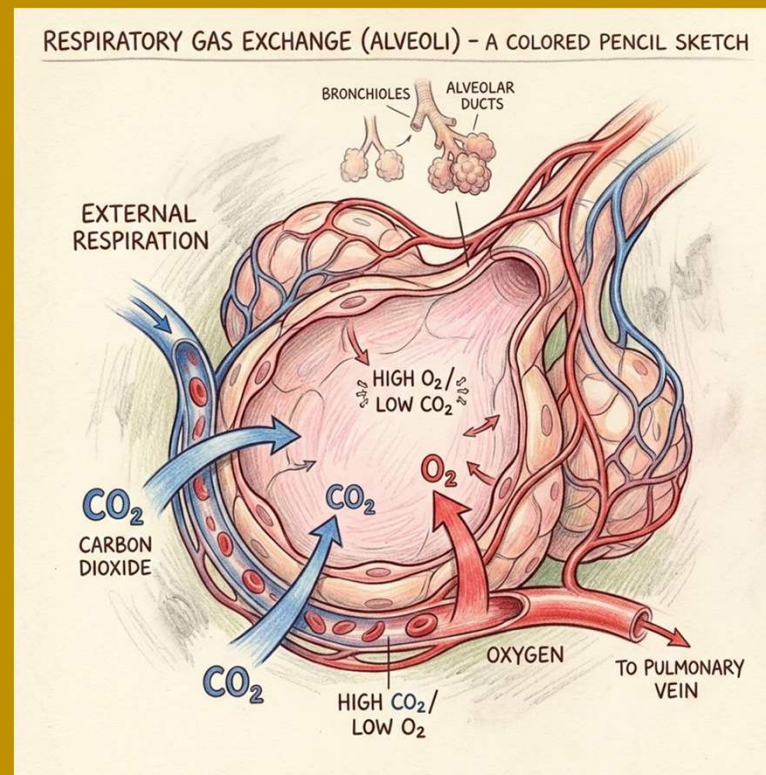
- The respiratory system consists of the upper airways, trachea, bronchi, bronchioles, and alveoli.
- • Upper airways
- • Lower airways
- • Alveoli: site of gas exchange



Respiratory Physiology

Gas exchange: oxygen in, carbon dioxide out.

- Ventilation
- Diffusion
- Perfusion



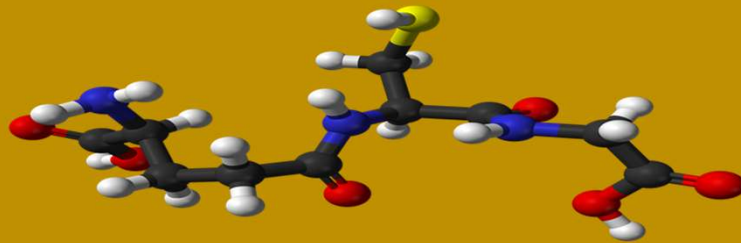
Respiratory Biochemistry

Antioxidant protection -Glutathione (GSH) stores in the lung -present in epithelial lining fluid

- .Ascorbic acid (Vitamin C)
- .Tocopherols (Vitamin E)
- .Uric acid

Enzymatic scavengers : Intracellular enzymes such as superoxide dismutase (SOD), catalase, and glutathione peroxidase convert toxic superoxides/reactive oxygen species (ROS) into water and oxygen

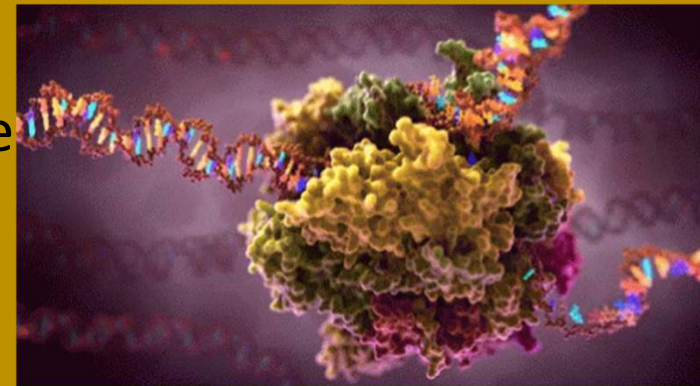
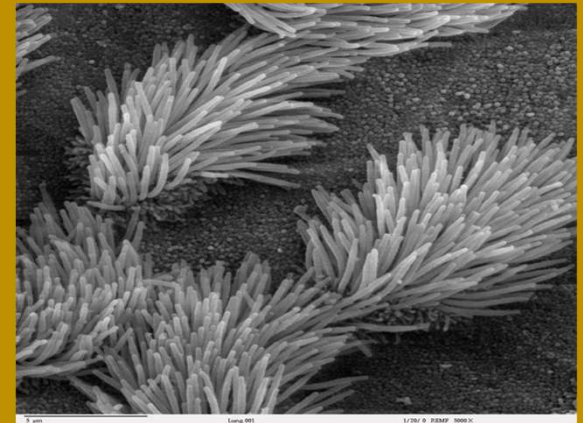
Nrf2 Pathway : The master regulatory transcription factor which activates cytoprotective and antioxidant genes in response to cellular stress.



Respiratory Cytology/Histology

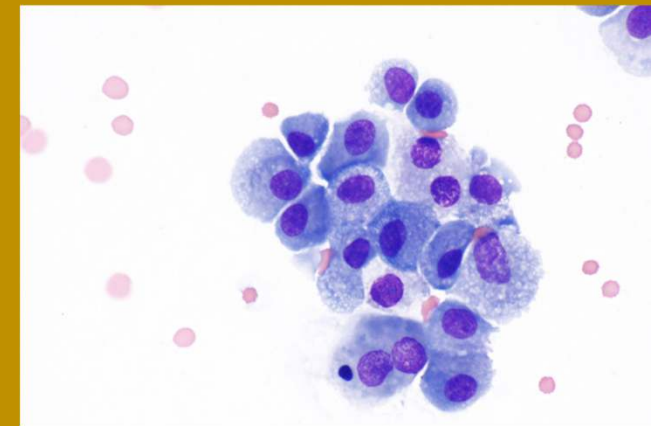
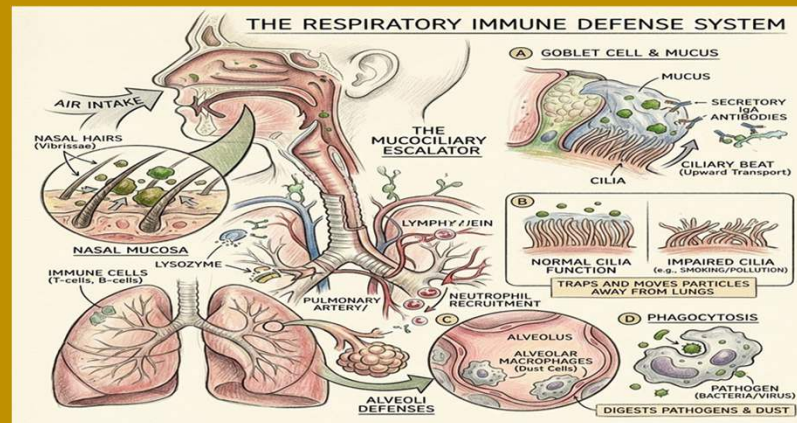
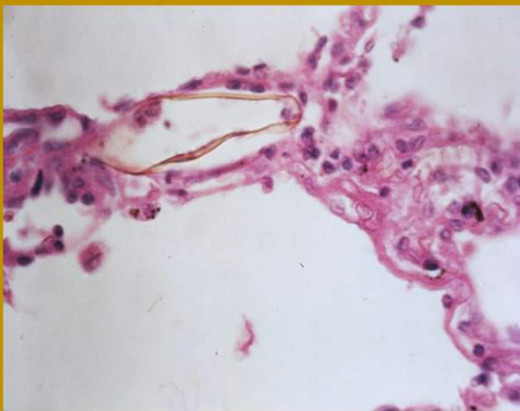
Respiratory epithelium structure.

- **Ciliated cells**-motile,hair-like projections
Coordinated ,whip-like movements that sweep mucus,trapped dust,and pathogens out of lungs and airways
- **Goblet cells**-secretory (mucus)
- **Basal cells**- basement foundation
- **Type I pneumocytes**-facilitate gas exchange, maintain ion /fluid balance ,communicate w/Type II to secrete surfactant in response to stretch
- **Type II pneumocytes**- production/secretion of surfactant to reduce surface tension



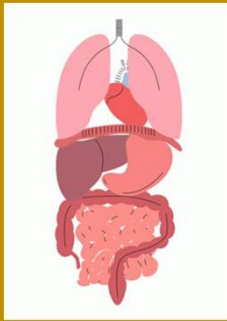
Respiratory Immunology

- Epithelial/physical, innate and adaptive respiratory immune defenses.
- **Physical barriers** ,eg. Vibrissae (nasal hairs),mucus,cilia
- **Innate**-Pulmonary/alveolar macrophages-phagocytosis (engulfing)
- dendritic cells-” messengers”,capture antigens,transport to lymph nodes,present antigens to T-cells to initiate adaptive immune response
- **Adaptive**-Pathogen -specific defense network

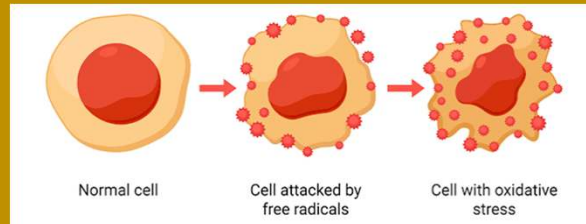


Hazardous Materials and Respiratory Diseases: Pathogenesis

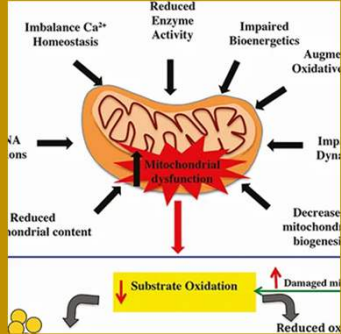
Inflammation



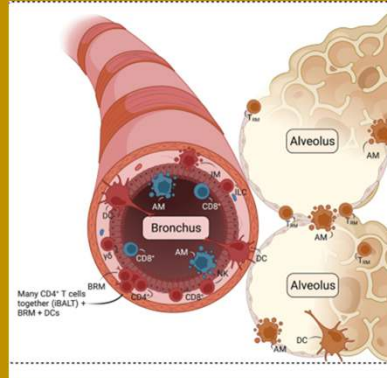
Oxidative Stress



Mitochondrial Injury

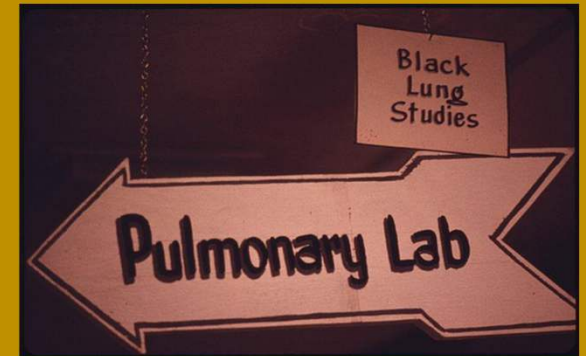


Impaired Defenses



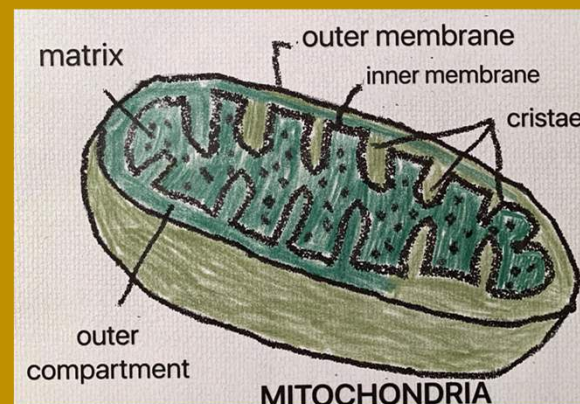
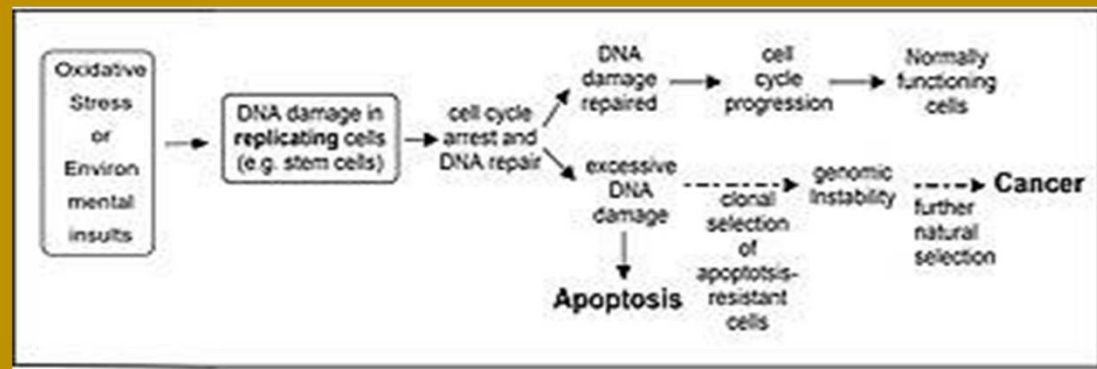
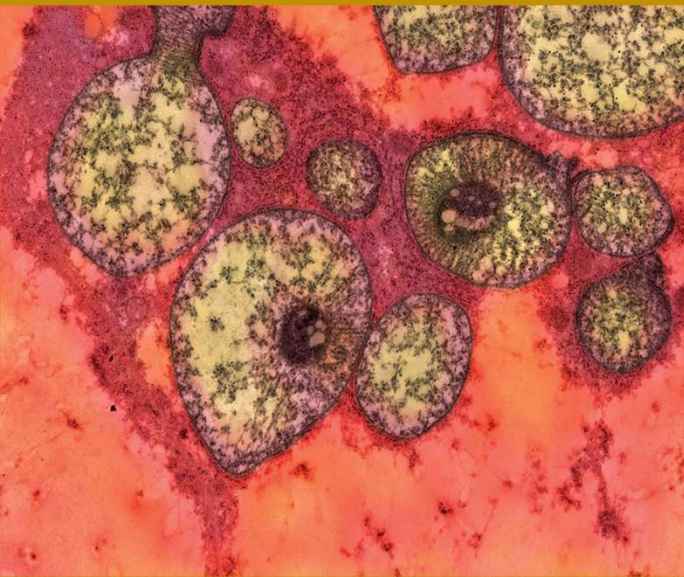
Categories of Respiratory Diseases and Dysfunction

- Obstructive, Restrictive, Other, Occupational and Environmental diseases
- Toxicant-induced respiratory injury can induce and exacerbate respiratory disease .Acute and chronic airway and lung pathology.
- Asthma ,reactive airways dysfunction
- COPD/COLD
- Bronchiectasis
- Pneumoconioses - asbestosis ,silicosis,beryllosis ,coal dust,Interstitial lung disease
- Pulmonary fibrosis , chemical pneumonitis ,ARDS



Mitochondrial Injury and Dysfunction

- Oxidative stress, energy disruption, cell death pathways.



HAZMAT: Physiochemical Properties

CHEMICAL AGENT CHARACTERISTICS

Physicochemical Properties Relate to Toxicity!

•Solid?

•Liquid?

•Gas/Vapor?

•Aerosol? Dust? Powder? Fumes?

•Volatility? (vapor pressure,vapor density)

•Viscosity? Boiling point? Freezing point? Flash point?

• Solubility? (degree of H₂O solubility),Miscibility?

•Reactivity/Corrosivity? (explosive, incompatibilities,extreme pH)

•Carrier substances present? (e.g., respirable particles)

These factors are also important for PPE selection

Toxicology and Pathophysiology

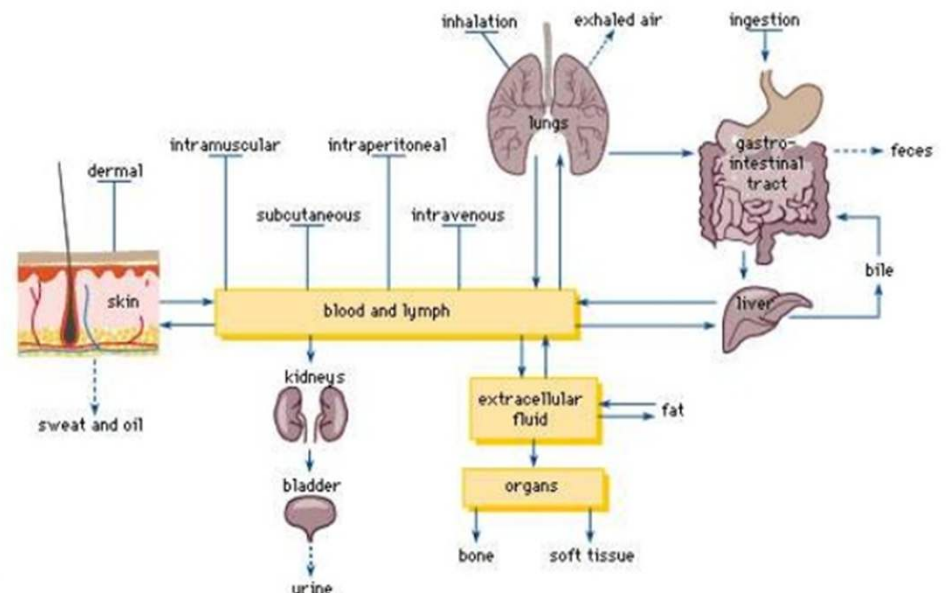
ROUTES OF EXPOSURE:

- Inhalation (most common resulting from airborne toxicants and bioagents)
- Dermal (skin absorption)
- Mucous membranes
- Ingestion
- Injection (less common in CBRN scenarios)

EXPOSURE PATHWAYS: ENVIRONMENTAL

- Air/Atmospheric transport
- Soil
- Water, Food

HOW CHEMICALS ENTER THE BODY IS DEPENDENT ON THE PROPERTIES OF THE CHEMICAL AND THE BIOLOGY OF THE ORGANISM.



Toxicology and Pathophysiology

•TOX-TERMS AND FACTORS

•The severity of a toxicological emergency involving CBRN or HAZMAT agents is determined by physicochemical properties and biological factors but also by factors involving :

•Exposure rates and time

$$\underline{DOSE = Exposure\ time\ x\ concentration}$$

•Concentration of substance

•Confined space vs. open air dispersal or release

•Environmental stability and persistence, particle size

Meteorological –e.g. wind direction& speed ,precipitation, humidity, UV light index(sunlight,topography

Toxicology and Pathophysiology

TOX-TERMS (Cont'd)

• **TDlo- *Toxic Dose,low*** (National Institute for Occupational Safety & Health (NIOSH)

The lowest dose of a substance introduced by any route ,other than inhalation, reported to produce any toxic effect in humans OR to produce tumors or reproductive effects in animals.

• **TClo-*Toxic Concentration ,low*** (NIOSH)-Same as above,except this deals with inhaled air concentrations

• **LDlo- *Lethal Dose,low*** (NIOSH)-The lowest dose of a substance introduced by any route ,other than inhalation ,which has been reported to cause death in humans or animals

• **LClo-*Lethal Concentration ,low*** (NIOSH)-Same as above, except this value deals with inhaled air concentrations.

TOX-TERMS AND FACTORS

The severity of a toxicological emergency involving CBRN or HAZMAT agents is determined by physicochemical properties and biological factors but also by factors involving :

Exposure rates and time **$DOSE = Exposure\ time \times concentration$**

Concentration of substance

Confined space vs. open air dispersal or release

Environmental stability and persistence, particle size

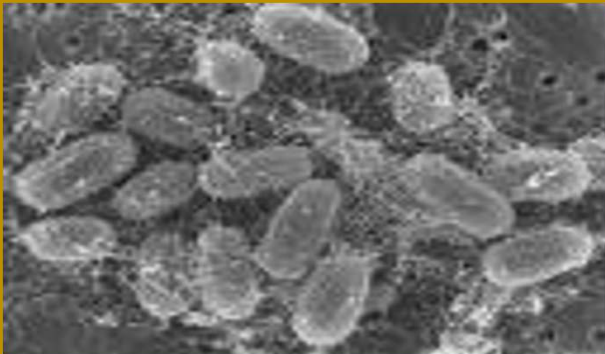
Meteorological –e.g. wind direction& speed ,precipitation, humidity, UV light index(sunlight)

Toxicology and Pathophysiology

Toxin vs. Toxicant

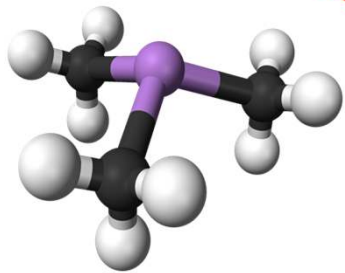
Toxin-derived or produced via a biological organism

Toxicant- Synthetic chemicals ,particulate matter ,radionuclides ,heavy metals (mercury ,lead, cadmium, beryllium)



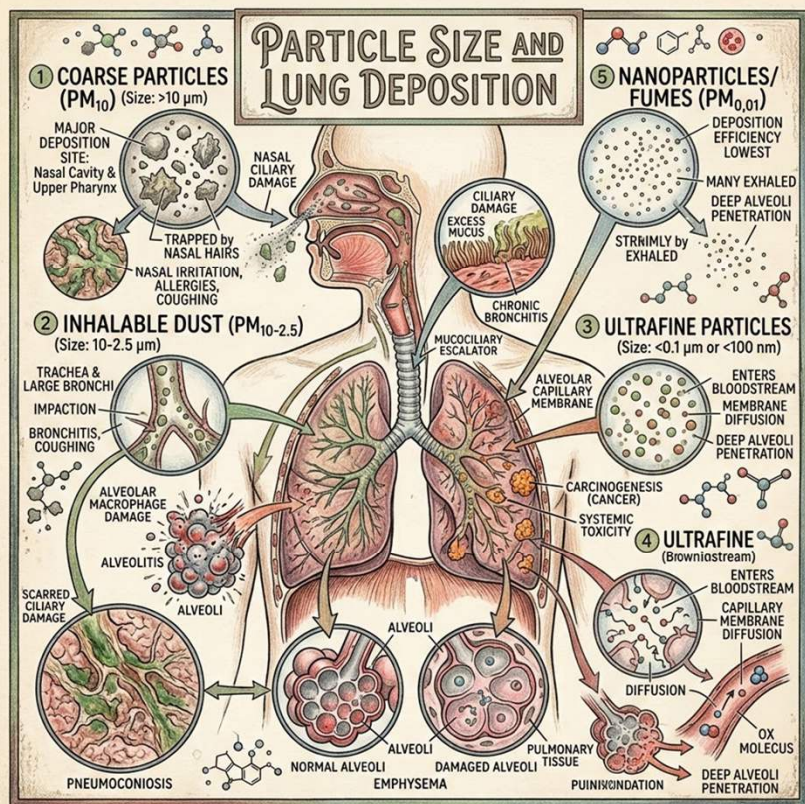


Toxicology and Pathophysiology

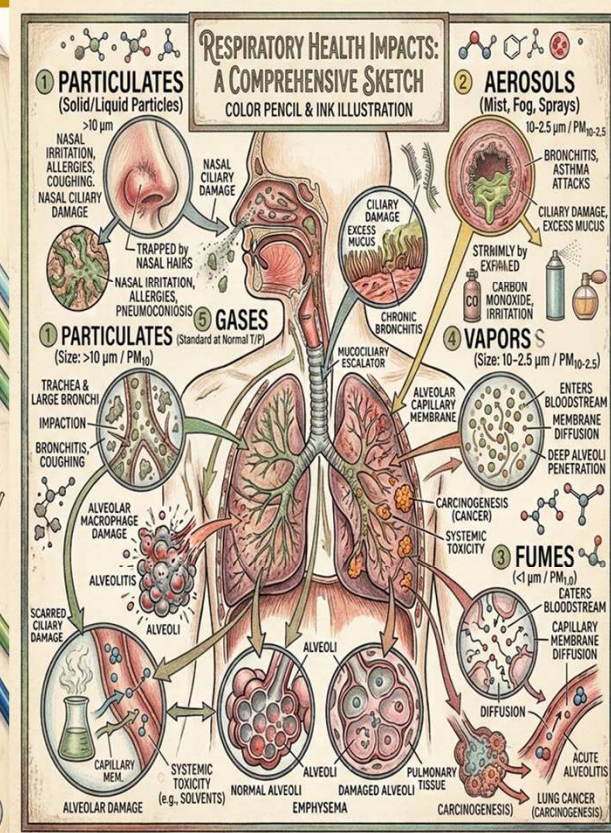


Particulates, Aerosols, Fumes, Vapors, and Gases

Particle Deposition



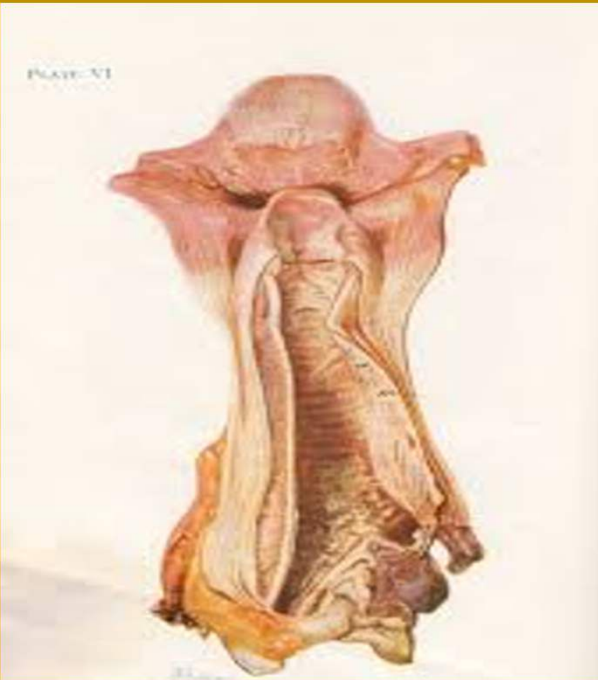
Respiratory Impacts



Mechanisms of Harm

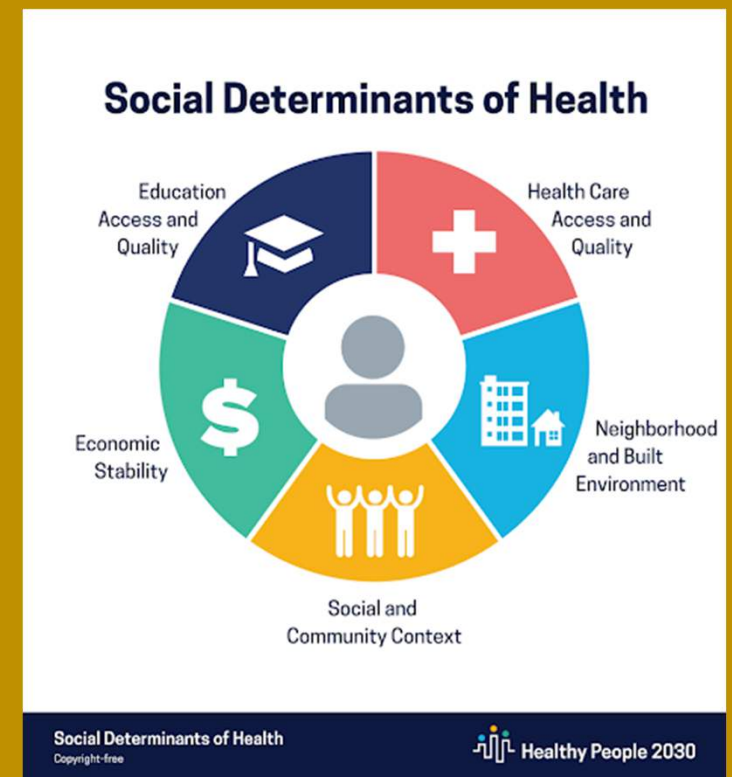
- “*Thermal*- Incendiary devices, e.g.,” Molotov cocktail”
- “*Etiologic*- Infectious diseases ,e.g. Anthrax {Etiology= cause or origin of a disease
- “*Asphyxiating*- Simple asphyxiating gases that displace oxygen/oxygen deficiency
- “*Mechanical*- Explosive blast, structural collapse
- “*Chemical*- Chemical exposures and effects
- “*Psychological*- Panic , Anxiety disorder, traumatic stress/PTSD
- “*Radiological*- Radiological Dispersal Device (RDD) ,” dirty bomb”, stationary radiological source or other terrorist use of radioactive materials (food supply contamination)
- “***Remember*** : “TEAM CPR” ***OR*** “CPR TEAM “

Mechanisms of Harm (cont'd)



Pre-disposing Factors to Exposures and Disease

- Age
- Genetics
- Occupation
- Immunity
- Pre-existing disease/medical history
- Socioeconomic factors
- Diet
- Lifestyle
- Environment
- Prior exposures



What is a Toxidrome?

Taken from the words *toxic and syndrome*.

A specific constellation clinical signs and symptoms caused by an overdose or dangerous exposure to a particular drug, chemical or poison

Types of toxidromes :

1. Irritant Gas -chlorine, phosgene, methyl isocyanate, ammonia
2. Corrosive - strong acids, bases, military vesicants (“ blister agents”)
3. Cholinergic - organophosphate pesticides, military nerve agents ,eg. sarin
4. Simple asphyxiants -CO₂, any substance which can cause oxygen displacement in the ambient environment ,eg inert gases such as argon
5. Systemic asphyxiants - CO ,HCN, nitrites, nitrates, nitrobenzene
6. Etiological- pathogens 7. Radiological-Ionizing radiation

Chemical Agent Toxicology and Pathophysiology

Pulmonary Agents

IRRITANT GAS TOXIDROME

- Directly affect the upper, central and /or the peripheral airways
- Effects are related to water solubility in the airways
- Example, chlorine gas will form hydrochloric and hypochlorous acids in respiratory tract. Chlorine affects the upper and central airways and is moderately water soluble, while phosgene is insoluble, has poor warning properties with delayed-onset pulmonary edema affecting the peripheral airways and alveolar-capillary membrane.
- Signs, symptoms and effects include coughing, dyspnea, inspiratory stridor, eye and conjunctival irritation, abnormal lung sounds (wheezes, rales, rhonchi), nose bleeds, hemoptysis, aphonia, reactive airways, bronchoconstriction, pulmonary edema, ARDS, respiratory failure
- Acute, delayed-onset and chronic health effects from inhalation
- Examples: anhydrous ammonia, chlorine, phosgene, methyl isocyanate

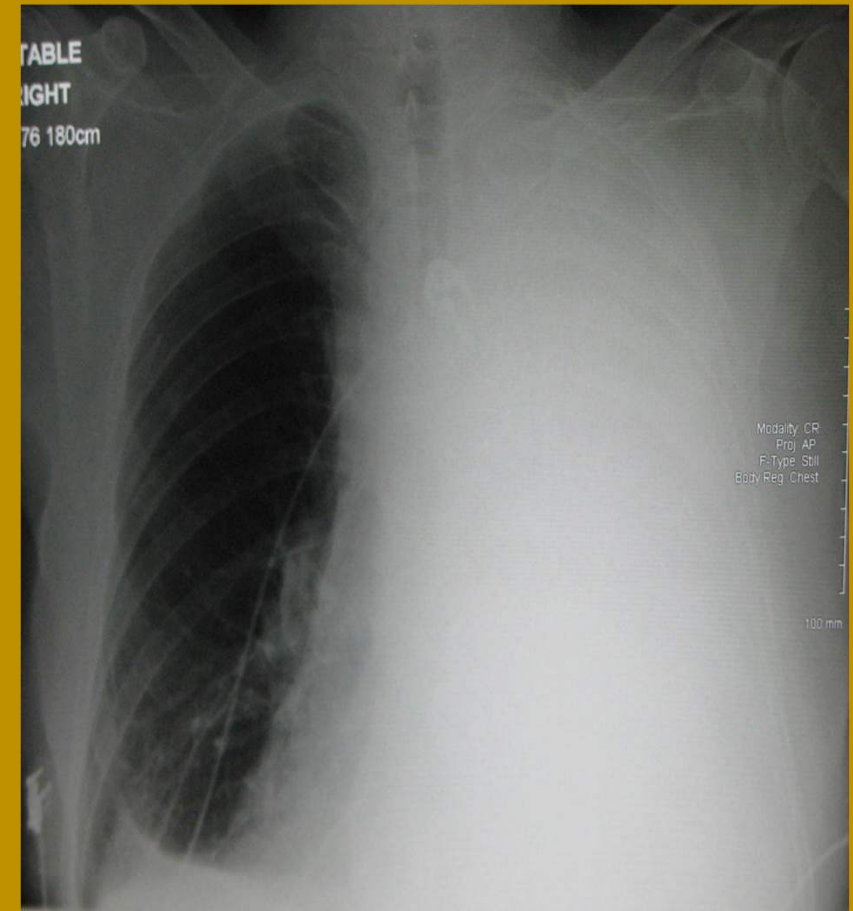
Pulmonary Agents: Treatment

- Remove patient from contaminated/toxic environment
- Decontamination of skin is usually not required for vapor/gas exposures
- Removal of clothing for any vapor trapping or “off-gassing” will normally suffice.
- Airway management and support
- High flow oxygen (100%)
- Aerosolized bronchodilators to reverse bronchospasm
- Possibly steroids, nebulized sodium bicarbonate (NaHCO_3) for acid gases
- Early intubation before glottic edema ensues, may require surgical airway
- Treat for non-cardiogenic pulmonary edema –CPAP,BI-PAP,MV



CXR OF PULMONARY /IRRITANT GAS VICTIM

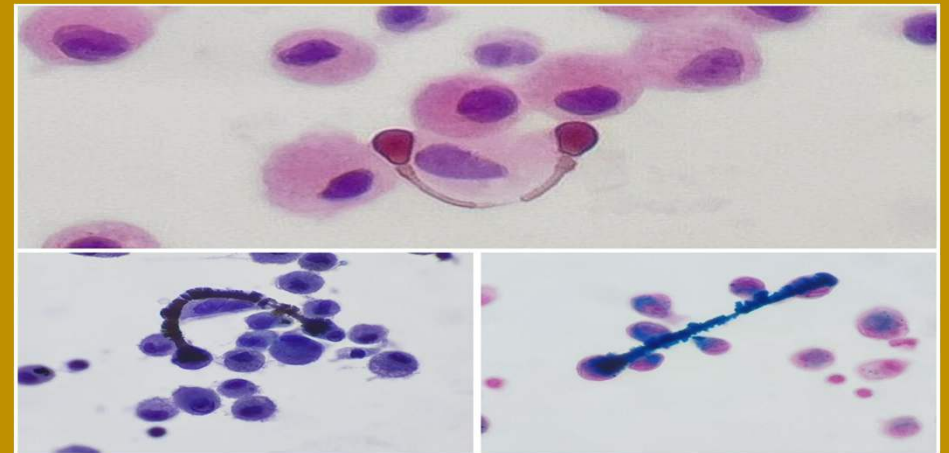
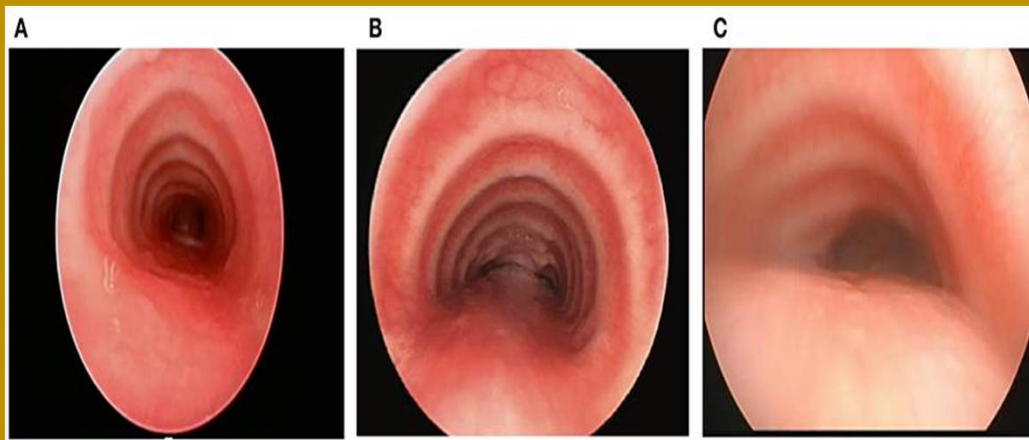
Diffuse ,bilateral infiltrates



BRONCHOSCOPY -BAL

Bronchoscopy and bronchoalveolar lavage are excellent tools for the clinical assessment of toxic inhalations and toxicant induced lung injury

Very important methods for pulmonary research studies /inhalation toxicology in animal models .



Chemical Agent Toxicology & Pathophysiology

CORROSIVE TOXIDROME

• **Vesicants** (aka “Blister Agents”)/ Corrosives

• **Sulfur mustard ,nitrogen mustards,mixed vesicants-** cause severe dermal,ocular and mucous membrane injury ,vapor inhalation severe toxic chemical injury to tracheobronchial tree and other pulmonary structures. **Radiomimetic**, as it alkylates and damages DNA similar to ionizing radiation. Mutagen,carcinogen and teratogen (causes birth deformities).

• Mustard agents can induce severe bone marrow suppression,as it” mimics “ radiation effects. **Effects are delayed –onset** . Nitrogen mustard-still used in Ca chemotherapy.

• **Lewisite- Has immediate onset and effects ,including pain.**

• **Phosgene Oxime** (“ nettle agent”,not a “true”vesicant)-strongly corrosive

• **“ Toxic caustics”** (Hydrofluoric acid-HF)-causes severe burns and hypocalcemia

• **Strong acids & bases /alkalai** ,e.g.,Sulfuric acid (H₂SO₄),nitric acid,sodium hydroxide (lye). Acids especially have been used regularly for targeted attacks in London which create serious chemical burns ,disfigurement ,disability and even death .

• **T-2 Mycotoxins** (fungal toxins) can cause vesicant tissue damage and induce radiomimetic effects . Biochemical toxins that are dermally active

Chemical Agent Toxicology and Pathophysiology

•VESICANTS (cont'd)

•S&S/Effects:

- Delayed blistering, irritation ,redness(erythema) ,wheals, necrosis
- Hoarseness
- Rhinorrhea ,nasal bleeding
- Cough, laryngitis, aphonia, inspiratory stridor, dyspnea, pneumonitis ,airway necrosis with tissue sloughing of large airways, pulmonary edema ARDS, respiratory failure
- Ocular-Severe conjunctivitis , lacrimation, blepharospasm and lid edema
- Bone marrow suppression , sepsis

Chemical Agent Toxicology and Pathophysiology

VESICANT /CORROSIVES EFFECTS-Chemical burns ,radiomimetic (mustard agents and T-2mycotoxins) ,infection,immune/bone marrow suppression,sepsis

Inhalational injury-Airway sloughing , pseudomembrane formation,bronchial casts,airway obstruction

VESICANT EFFECTS



Iran/Iraq War: 90-95% burns, pulmonary injury, bone marrow suppression, sepsis, and eventually died.

CORROSIVE /VESICANTS TREATMENT

•VESICANTS –TREATMENT

- No antidotes available
- Decontamination
- Airway management
- Ventilatory support
- Judicious fluid therapy
- Irrigate eyes
- Utilize burn protocols
- DO NOT*** RUPTURE BLISTERS!
- Will create entry for pathogens
- BLISTERS ***DO NOT*** CONTAIN AGENT



Chemical Agent Toxicology and Pathophysiology

Cholinergic Toxidrome -Nerve Agent/Organophosphates Signs& Symptoms

*-Due to the S&S overstimulation of parasympathetic nervous system the following mnemonic can be used: **D-U-M-B-E-L-S***

*-**D**iarrhea,Diaphoresis*

*-**U**rination*

*-**M**iosis (pinpoint pupils)*

*-**B**ronchorrhea, Bronchoconstriction,Bradycardia ("**KILLER B's**")*

*-**E**mesis*

*-**L**acrimation*

*-**S**alivation (sialorrhea),Sweating (diaphoresis),Secretions*

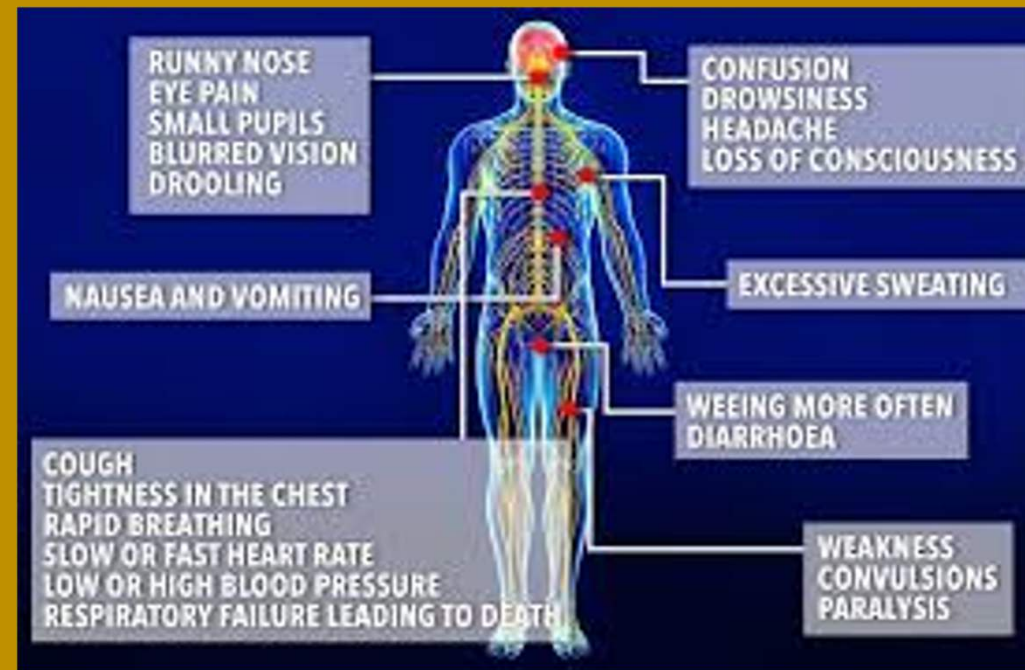
Muscarinic S&S -Glands

Nicotinic S&S - Muscles



Chemical Agent Toxicology and pathophysiology

NERVE AGENTS The “**BAG & PUDDLES**” MNEMONIC - Cholinergic Toxidrome



Chemical Agent Toxicology and Pathophysiology

Nerve agents: Inhibit **acetylcholinesterase(AChE)**-enzyme that breaks down neurotransmitter responsible for nerve transmission

AchE “eats” Ach

N.A.s BIND to AchE, inhibiting its action

When nerve agents or OP insecticides bind to AchE ,an “aging” process occurs ,which results in irreversible bond, If not treated.

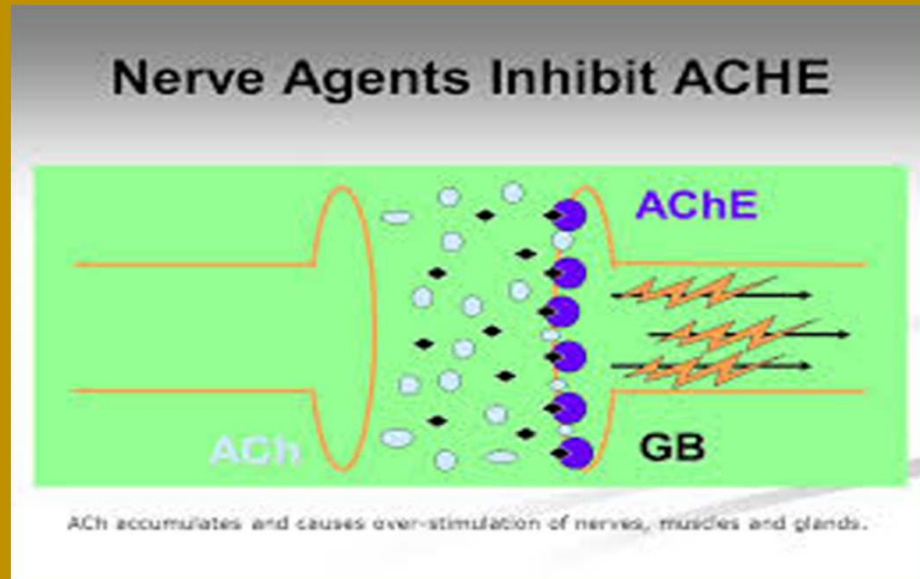
The aging time varies depending upon the nerve agent ,but requires administration of antidote promptly.

Acetylcholine builds up at the **nerve synapse and**, nerve impulses continue to stimulate muscles and glands uncontrollably and will create **clonic and tonic seizures** causing excessive production of fluids & **bronchospasm (constriction of bronchial tubes) .**

Bradycardia will also result in an excessively slow heart rate .

Bronchorrhea ,bronchospasm, bradycardia= (“KILLER B’s”) – CHOLINERGIC CRISIS/ TOXIDROME resulting from overstimulation of PARASYMPATHETIC NERVOUS SYSTEM (PNS).

Chemical Agent Toxicology and Pathophysiology



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NERVE AGENTS : TREATMENT

Interventions : FOLLOW YOUR DEPARTMENTAL PROTOCOLS & PROCEDURES

“Decontamination – Clothing removal appropriate for both vapor & liquid exposures This removes ~ 85-90% of contaminant, followed by GENTLE warm water and soap wash. Wipes and foams are also available. DO NOT USE BLEACH ON SKIN !

“Decontamination also serves as a therapeutic modality by reducing absorption of toxicants . Rapidly removing patients from contaminated areas also serves to minimize exposure.

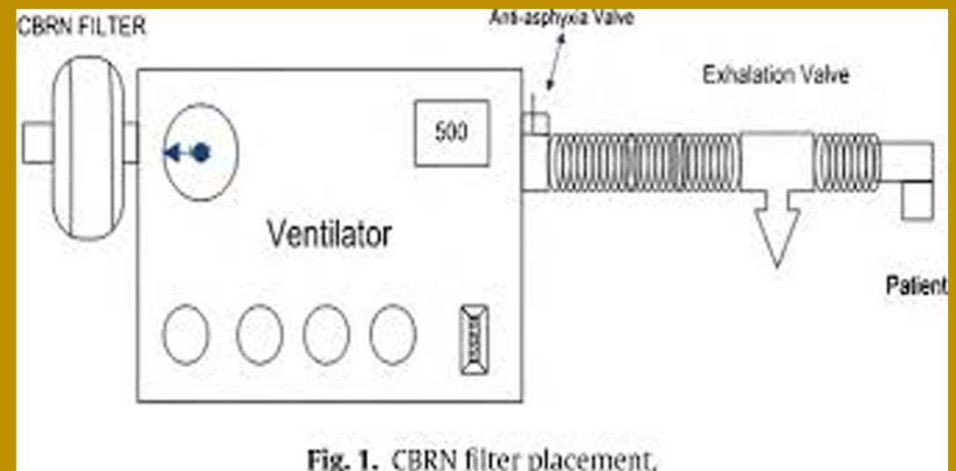
“Provisions for special needs populations must also be provided

Life –saving procedures must be incorporated

into the decontamination process (,hemorrhage control) airway management, needle decompression for tension pneumothorax ,antidote administration

NERVE AGENTS : AIRWAY & SUPPORTIVE THERAPY

- Airway management , oxygen ,ventilatory and cardiovascular support,monitoring in Warm Zone /Decontamination area
- Transport ventilators with CBRN filtration are available for use in both transport and decontamination settings



NERVE AGENTS : PHARMACOTHERAPY

Pharmacotherapy- therapy may begin in Hot Zone with autoinjectors

-Atropine - reverses cholinergic crisis, dries up secretions ,reverses bronchoconstriction llows for AchE to be regenerated to regulate acetylcholine and normalize nerve impulse transmission .

Available as **DuoDote** autoinjector-Atropine & 2-PAM Chloride
and bradycardia

-2-PAM chloride - an oxime antidote which “frees” the AchE from the nerve agent and a-
Benzodiazepines eg diazepam or midazolam to control seizures

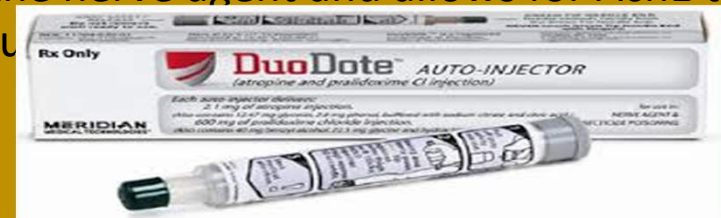
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Chemical Toxicology and Pathophysiology

SYSTEMIC ASPHYXIANTS

• Inhibit oxygen transport of hemoglobin OR inhibit mitochondrial enzyme systems responsible for oxidative phosphorylation and efficient production of ATP . This results in disruption of oxygen utilization and production of adequate cellular energy and tissue hypoxia (anaerobic metabolism).

• SIGNS & SYMPTOMS: Cherry-red skin (CO poisoning),altered mental status ,headaches, tachypnea ,
air hunger (especially cyanides),unconsciousness ,pallor or cyanosis ,combative behavior, confusion,
irritability,seizures

..

• EXAMPLES :

• Carbon Monoxide (CO) binds 250 x greater than O₂ to hemoglobin = poor **OXYGEN TRANSPORT** to cells resulting in tissue hypoxia/anoxia

Cyanide: Inhibits cytochrome a₃ system in mitochondria = poor **OXYGEN UTILIZATION**

• Azides, Arsine , (possibly,Phosphine), Hydrogen sulfide (H₂S) are all considered systemic asphyxiants .Arsine causes hemolysis resulting in poor oxygen transport tissue hypoxia/anoxia.

ASPHYXIAN TOXIDROME

Treatment

SYSTEMIC ASPHYXIANTS TREATMENT

- Remove victims from contaminated atmosphere
- Airway management & ventilatory support
- High –flow oxygen (100%)
- Hyperbaric oxygen
- Cyanide Antidote Kit (for cyanide toxicity)
- Includes Sodium Nitrite and & Sodium Thiosulfate
- Hydroxycobalamin (Vitamin B12 analogue)-newer and safer therapy
- -Converts cyanide to B12



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A yellow circle containing the black letter 'G'.A yellow circle containing the black letters 'VX'.

Decontamination

“Interventions : FOLLOW YOUR DEPARTMENTAL PROTOCOLS & PROCEDURES

“Decontamination – *Clothing removal appropriate for both vapor & liquid exposures This removes ~ 85-90% of contaminant, followed by GENTLE warm water and soap wash. Wipes and foams are also available. DO NOT USE BLEACH ON SKIN !*

“*Decontamination also serves as a therapeutic modality by reducing absorption of toxicants . Rapidly removing patients from contaminated areas also serves to minimize exposure.*

“*Provisions for special needs populations must also be provided*

Life –saving procedures must be incorporated into the decontamination process ie, hemorrhage control, airway management, needle decompression for tension pneumothorax ,antidote administration

Decontamination (Cont'd)

"HOT ZONE OR EXCLUSION ZONE" – ***This where the epicenter of the event and the immediate contaminated area are located*** .Unless the suspected/HAZMAT CBRN agent has been ascertained, concentrations are known ,and there are acceptable ambient oxygen levels, personnel entering this area should be wearing a fully encapsulated, vapor tight HAZMAT ensemble with self –contained breathing apparatus (SCBA). This is known as a Level A ensemble, and is used for **initial reconnaissance ,sampling and monitoring**, rescue and **recovery operations** and anytime there are unknown factors such as nature of a CBRN threat or an Immediately Dangerous to Life and Health (IDLH) atmosphere . The Health and Safety Officer at any emergency operation must be consulted as to selection of appropriate personal protective equipment (PPE) along with an on scene risk vs.benefit assessment.



Decontamination (Cont'd)

WARM ZONE OR CONTAMINATION REDUCTION ZONE – This is the area where the treatment of life threatening conditions ,such as airway compromise , severe hemorrhage , tension pneumothorax ,acute toxicity ,as well as decontamination occur simultaneously.

· **NOTE:** *Current thoughts and protocols call for rapid and initial treatment in the Hot Zone for severe hemorrhage (tourniquet application) and administration of nerve agent countermeasures. PPE here is a level lower than Level A (usually B ,or may be downgraded further as a result of on going sampling and monitoring and the recommendations of the Safety Officer).*



Decontamination (Cont'd)

"COLD, CLEAN or SUPPORT ZONE"- While this area is the "clean" and uncontaminated area, the situation in any HAZMAT or WMD-CBRNE event is fluid and dynamic and can change swiftly . This is the area where EMS teams will stage for **Triage, Treatment** HOT ZONE OR EXCLUSION ZONE – ***This where the epicenter of the event and the immediate contaminated area are located*** . Unless the suspected CBRN agent has been ascertained, concentrations are known ,and there are acceptable ambient oxygen levels, personnel entering this area should be wearing a fully encapsulated, vapor tight HAZMAT ensemble with self –contained breathing apparatus (SCBA). This is known as a Level A ensemble, and is used for **initial reconnaissance ,sampling and monitoring, rescue and recovery operations and anytime there are unknown factors such as nature of a CBRN threat or an Immediately Dangerous to Life and Health (IDLH) atmosphere** . The Health and Safety Officer at any emergency operation must be consulted as to selection of appropriate personal protective equipment (PPE) along with an on scene risk vs.benefit assessment



Summary/Key Takeaways

- Hazardous materials and their sources are ubiquitous in commerce, industry, healthcare, our homes, workplaces, schools, etc.
- Toxic, reactive, corrosive, flammable chemicals, heavy metals and radionuclides can be released intentionally as Weapons of Opportunity and Convenience to harm people and the environment.
- The use of WMD-CBRN agents is a major homeland and national security issue. The respiratory system is the major route of exposure and target organ system for airborne chemicals, gases, vapors, fumes, and particulate matter and can also induce profound systemic effects, as well as chronic disability.
- Respiratory therapeutics and critical care medical support are ESSENTIAL to manage chemically ill / injured victims.
- Respiratory therapists should be able to recognize toxidromes and be trained in the medical management of hazardous materials and CBRN affected patients. Decontamination and PPE are required in the medical

Q & A...????

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