Lethal Gases and Emergency Services on the Battlefield

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CONFLICT OF INTEREST

None to declare

Outline

- Overview of the Emergency Services on the Battlefield
- Discuss personal protection measures and decontamination procedures
- List some lethal gases, clinical manifestation and management

Chemical Weapons
History and Epidemiology

- Large-scale chemical warfare began in **World War I** (1.3 million casualties and 90,000 deaths)
- The Germans first used sulfur mustard in 1917
- Germany began producing **nerve agents** just before **World War II**.
- Sarin was synthesized in 1938

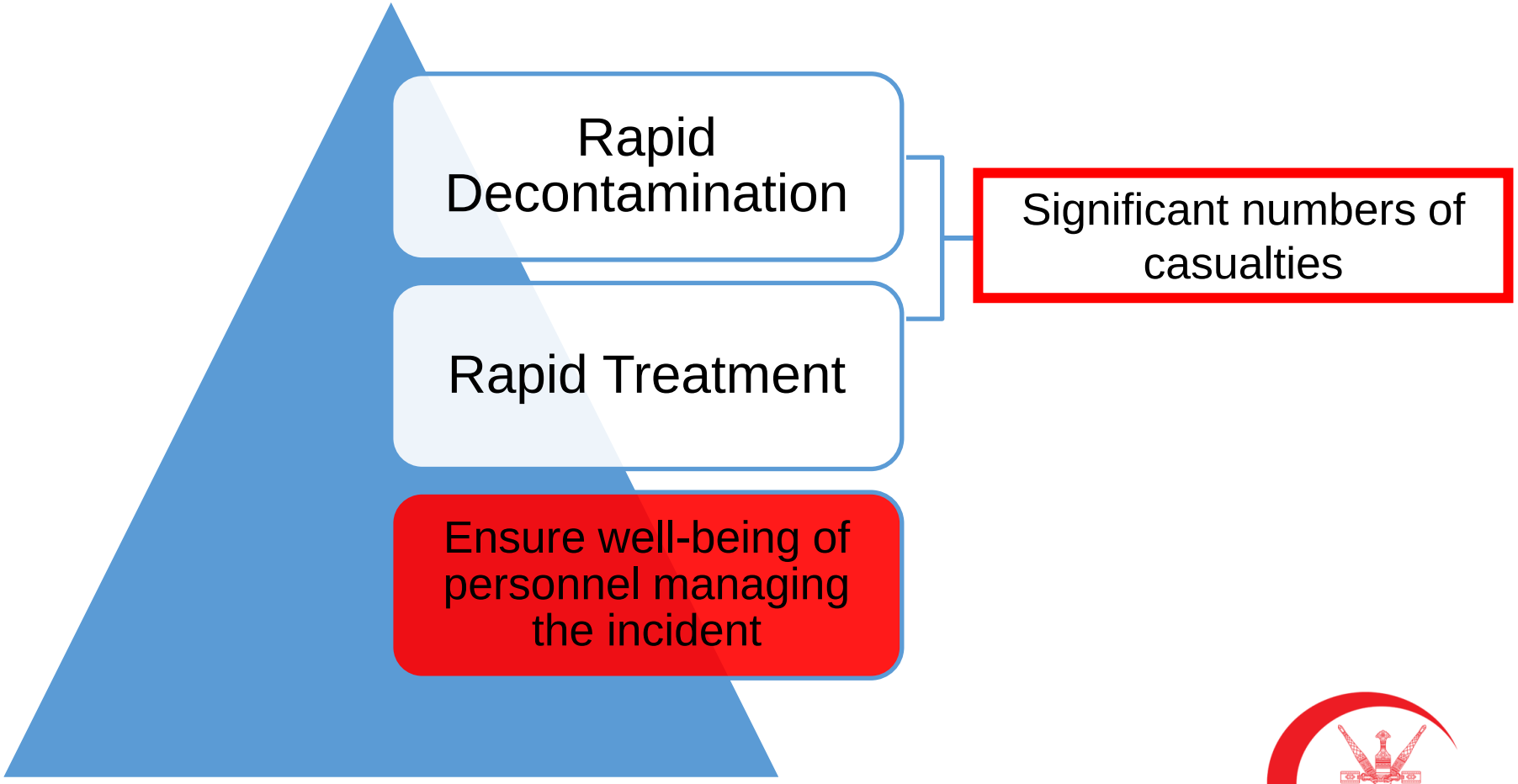
Why Chemical Terrorism?

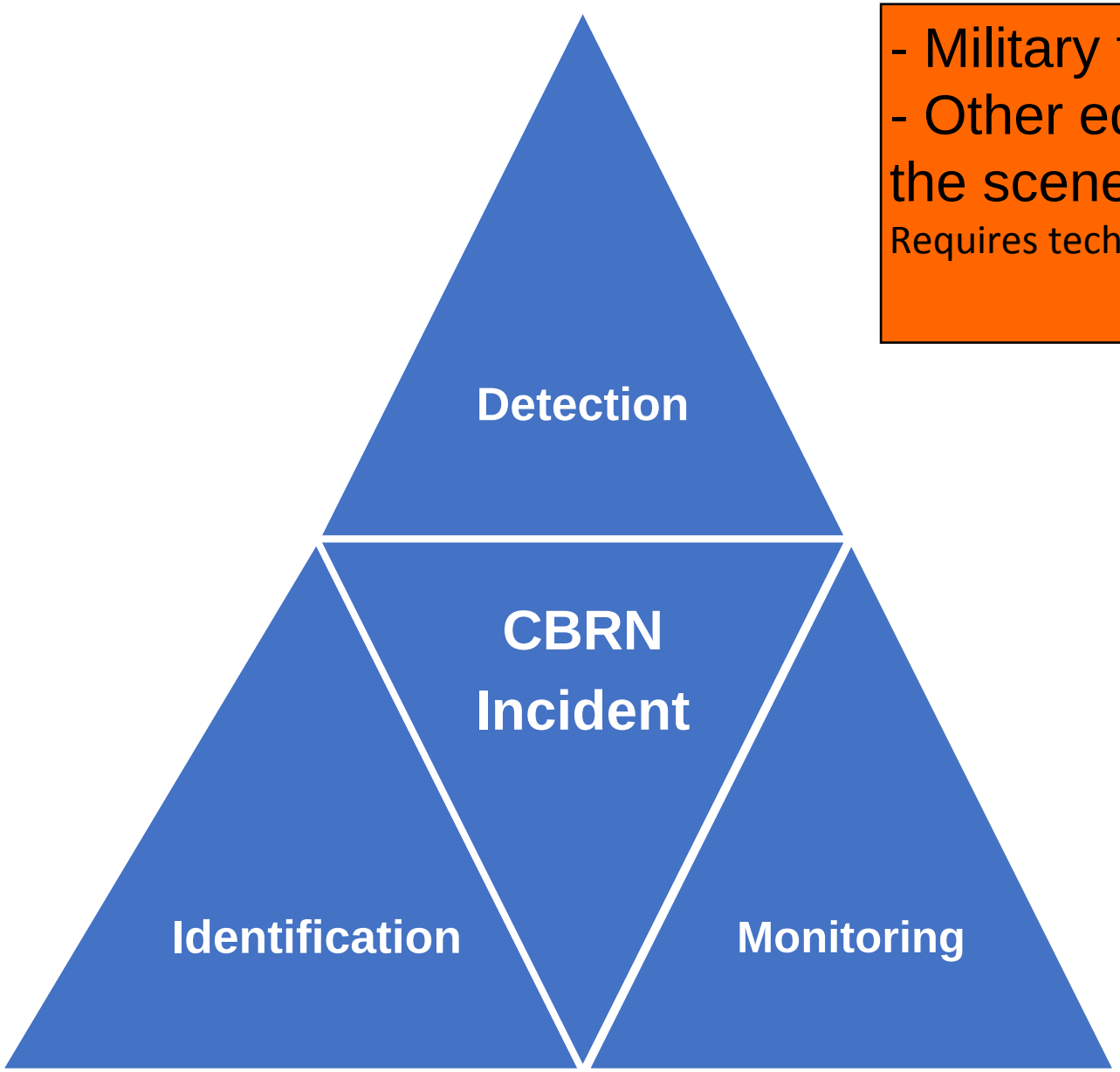
- Agents are available and relatively easy to manufacture
- Large amount not needed in closed space
- Chemical incidents may be difficult to recognize
- Easy to cover a large area
- Associated significant psychological impact
- Can overwhelm existing resources

Chemical Agents

- Nerve agents
- Blister agents
- Incapacitating agents
- Riot control agents
- Pulmonary agents
- Cyanides (Chemical asphyxiants)

Chemical, Biological, Radiological or Nuclear Challenge





- Military test paper to test liquid samples
- Other equipment that samples the air on the scene of the release

Requires technical expertise and training

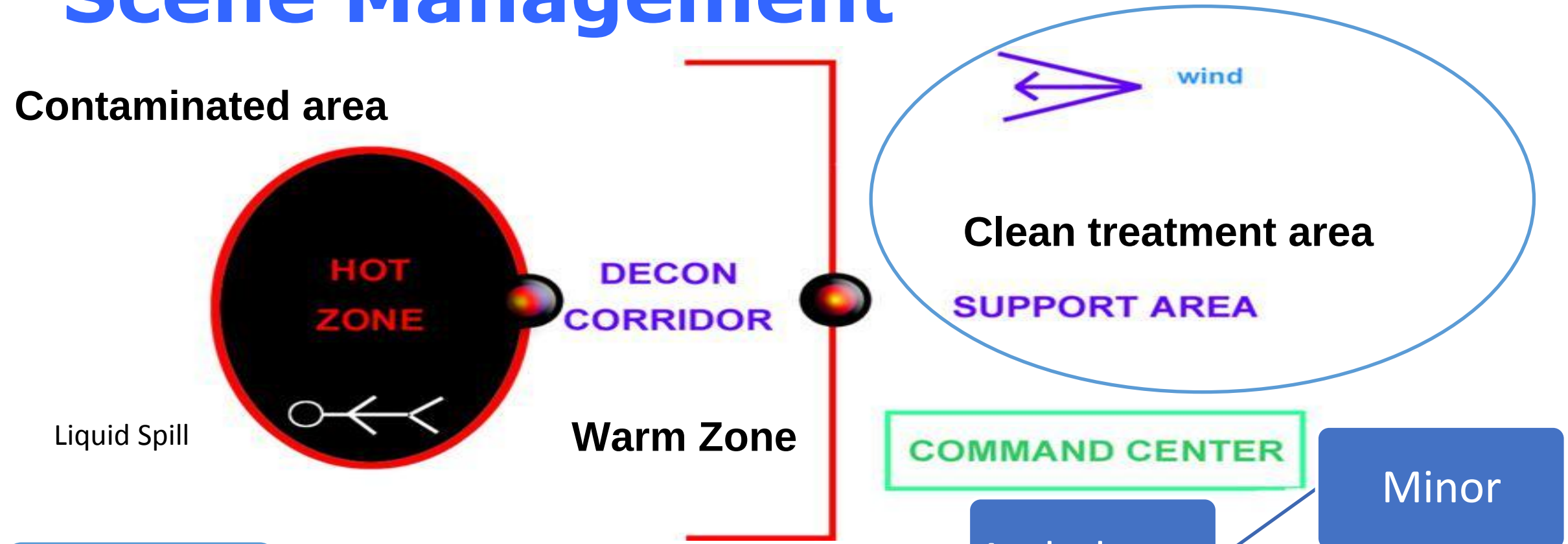
Ramesh, Aruna C., and S. Kumar. “Triage, Monitoring, and Treatment of Mass Casualty Events Involving Chemical, Biological, Radiological, or Nuclear Agents.” *Journal of Pharmacy and Bioallied Sciences* 2.3 (2010): 239–247. *PMC*. Web. 19 May 2017.

Scene Management

- Control movement of contaminated victims
- Provide safe working environment for responders
- Contain the release of the substance

Control Access

Scene Management



Source Medscape

A summary of physical characteristics of nerve agent exposure in a triage procedure

Category	Status of Casualties
Immediate – RED	Causality walking, talking and being capable of self aid Miosis and rhinorrhea Mild to moderate respiratory distress
Delayed - YELLOW	Recovering with antidotes
Minor - GREEN	Causality walking, talking and being capable of self aid Miosis and rhinorrhea
Expectant - BLACK	Not talking (unconscious) Circulation failed (no heart rate)

Ramesh, Aruna C., and S. Kumar. “Triage, Monitoring, and Treatment of Mass Casualty Events Involving Chemical, Biological, Radiological, or Nuclear Agents.” *Journal of Pharmacy and Bioallied Sciences* 2.3 (2010): 239–247. *PMC*. Web. 19 May 2017.

Decontamination Consideration

The principle of decontamination are:

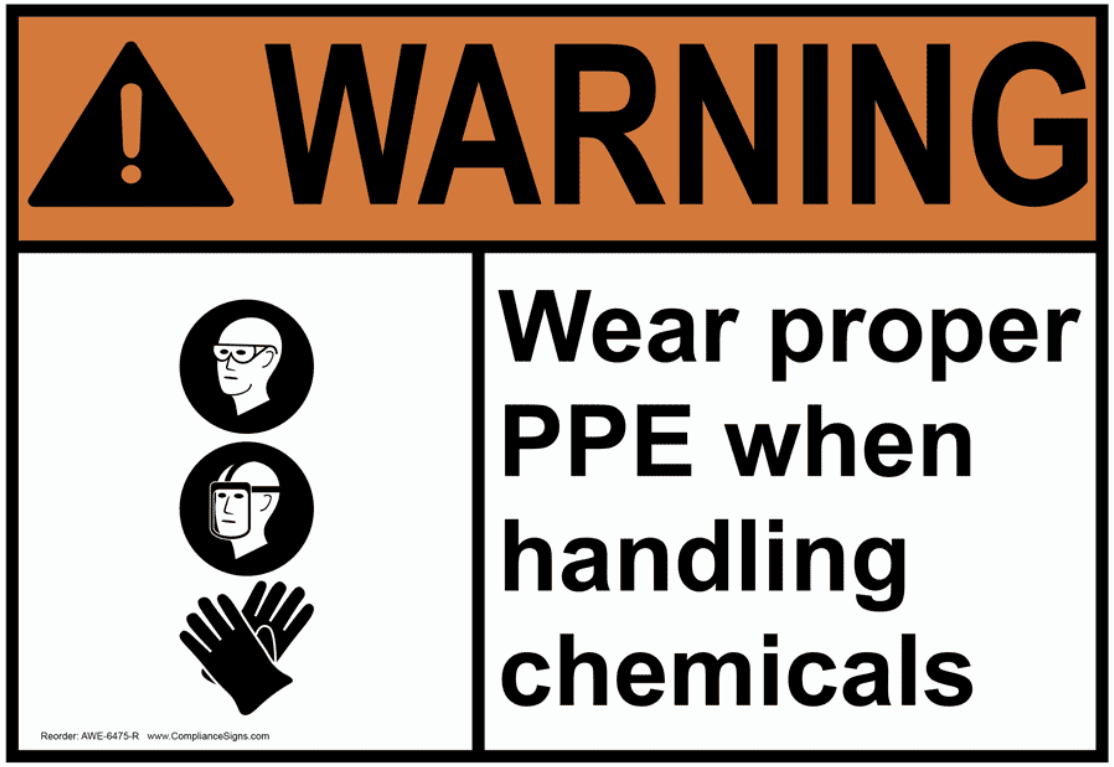
- Decontaminate as soon as possible
- Decontaminate by priority
- Decontaminate only what is necessary
- Decontaminate as far forward as possible

Decontamination is more important in liquid agents exposure than gases agents

Dermal Contamination with Liquid Versus Vapor Exposure

- Skin contamination with liquid agent can have delayed onset of signs and symptoms (up to 18 hours depending on factors like the amount of liquid agent).
- Exposure to vapor will cause more signs and symptoms more rapidly.
  - Eye complaints may be common
  - Respiratory complaints are common

Personal Protective Equipment Levels (PPE)



Level A

Self Contained Breathing Apparatus

Encapsulating vapor tight suit

Gases, vapors, aerosols, oxygen-deficient atmospheres

Level B

Self Contained Breathing Apparatus

Lesser skin protection

Chemical resistant suits

Liquids & Solids

Level B Ensemble

Level C

Air Purifying Respirator

Hooded, splash-protective chemical resistant suit

Liquids & Solids

Level D

NO Respiratory Equipment

NO Skin Protection

Personal Protection

- Butyl rubber or nitrile gloves. Latex not protective

Latex

Nitrile

Nerve Agents

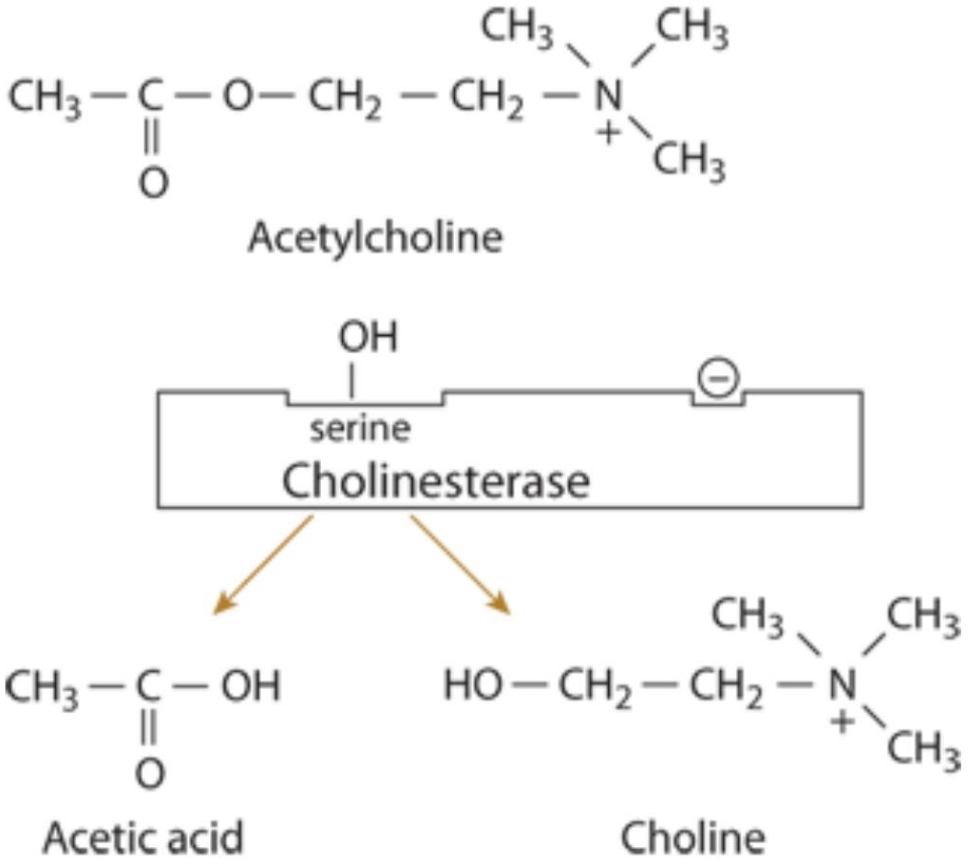
Nerve Agents

- Extremely potent organic phosphorus compound cholinesterase inhibitors
- Pure nerve agents are clear and odorless
- 1930’s- Nazi’s synthesize “G” agent during WWII
- 1940”s - Soviet Union begins production after capturing German munitions
- 1950”s - USA begin production

Nerve Agents

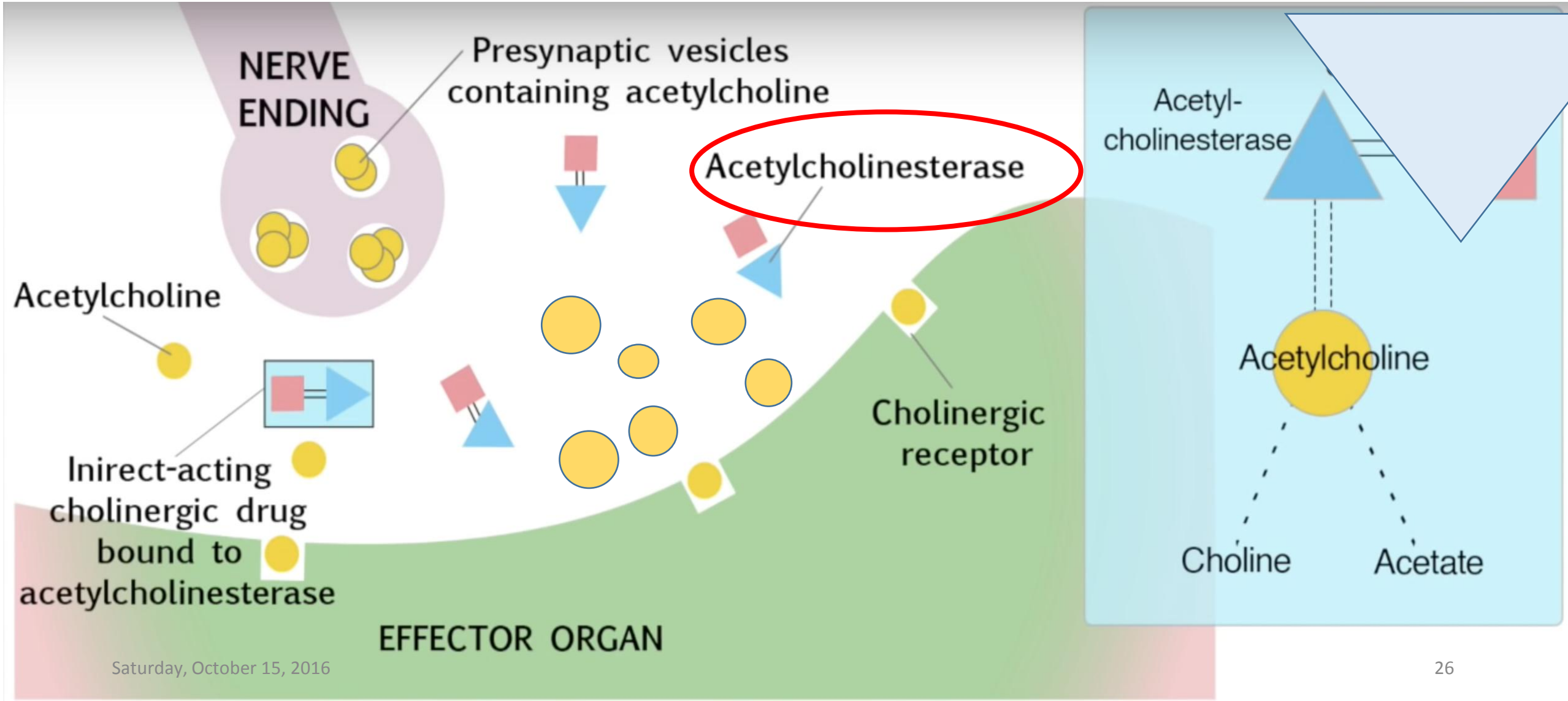
- Two classes
  - G agents
    - Tabun (GA)
    - Sarin (GB)
    - Soman (GD)
  - V agents
    - VX
- Cholines**terase** inhibitors - similar to organophosphate

Nerve Agents
Mechanism of Action



Suchard JR. Chemical Weapons. In: Hoffman RS, Howland M, Lewin NA, Nelson LS, Goldfrank LR. eds. *Goldfrank's Toxicologic Emergencies, 10e.* New York, NY: McGraw-Hill; 2015

Cholinergic Toxidrome



Nerve Agents
Mechanism of Action

- Organophosphates bind **irreversibly** to cholinesterase, unless pralidoxime is given prior to dealkylation “aging.”

- “Aging” is the average time for irreversible binding between organophosphates & cholinesterase

Nerve Agents
Aging Half-Times

Name of the Nerve Agent	Synonym	Aging Half-Time
Sarin	GB	Around 5 hours
Soman	GD	Around 2 minutes
Tabun	GA	>14 hours
VX	None	Around 48 hours

Nerve Agents
Clinical Effects

- The resultant **cholinergic toxidrome**
  - Peripheral **Muscarinic**
  - Peripheral **Nicotinic**
  - **Central effects** (loss of consciousness, seizures, respiratory depression)
- Aerosol or vapor exposure initially affects the eyes, nose, and respiratory tract. Miosis is common - 1995 Tokyo subway sarin incident

- **Long-term effects:** psychologic sequelae

Nerve Agents

Peripheral Nervous System- Muscarinic

❖ **DUMBBBEL**

- **D**iarrhea
- **U**rination
- **M**iosis, Muscle fasiculation
- **B**radycardia
- **B**ronchorrhea
- **B**ronchospasm
- **E**mesis
- **L**acrimation

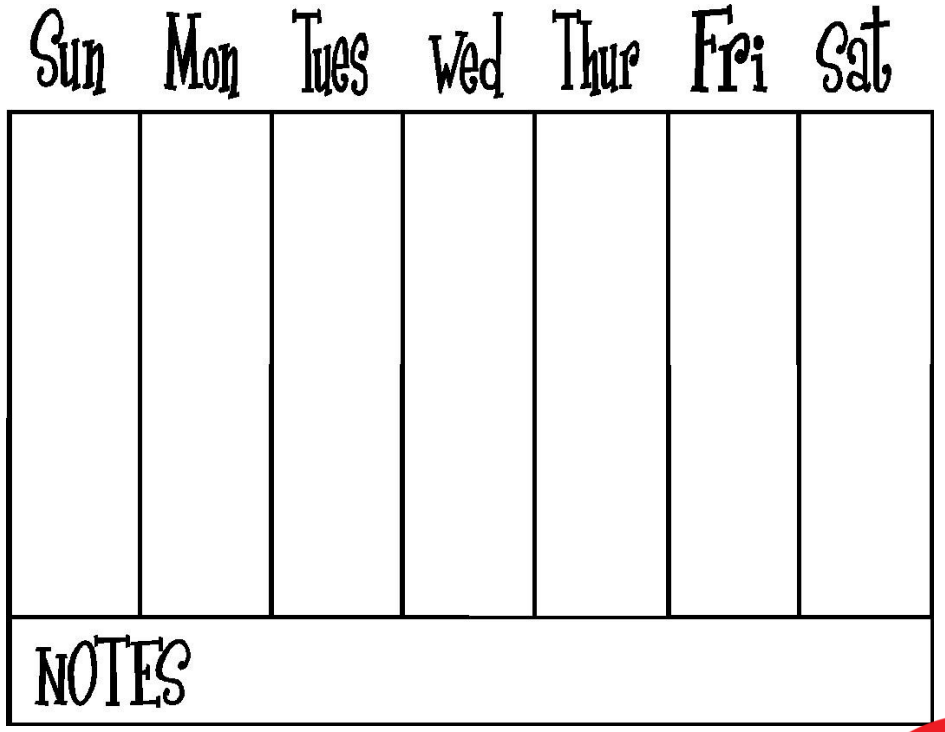
❖ **SLUDGE**

- **S**alivation
- **L**acrimation
- **U**rination
- **D**iarrhea
- **G**I complaint
- **E**mesis

Nerve Agents

Peripheral Nervous System- Nicotinic

- **M**- Mydriasis
- **T** - Tachycardia
- **W** - Weakness
- **(t) H** - Hypertension
- **F** -Fasiculations



Nerve Agents
Decontamination

- Two functions:
  1. Prevent further absorption
  2. Prevent spread to other persons
- Alkaline solutions
  - Diluted sodium hypochlorite solution (takes 15-20 minutes to inactivate chemical agent)
- Soap and Water

Nerve Agents
Treatment

- **Atropine** - combat excess muscarinic Acetylcholine
  - Goal: dry respiratory secretions
  - Dose: adults: 2 mg; children: 0.05-0.1mg/kg.
    - Repeat doses are given every 2- 5 minutes until resolution of muscarinic signs

Nerve Agents
Treatment

- **Diazepam** - combat excess nicotinic Acetylcholine
- **Oximes (Pralidoxime)** - combat "aging" process
  - Should be given in conjunction with atropine (can't reverse muscarinic effects when given alone)
  - **Mark I Kits:**
    - Pralidoxime autoinjector
      - 600mg IM
    - Atropine Autoinjector
      - 2mg IM

Nerve Agents
Pre-treatment

- **Pyridostigmine**
  - Carbamate acetylcholinesterase inhibitor (reversible)
  - Occupy cholinesterase > blocking access of nerve agent to the active site
  - Protect a small %of AchE from irreversible OP aging

Pulmonary Irritants

Pulmonary Irritants

- Chlorine (Cl)
- Phosgene (CG)
- Diphosgene
- Nitrogen oxides (NOx)
- Various organohalides

- **Induce delayed ARDS from increased alveolar-capillary membrane permeability**

Pulmonary Irritants

Agent	Color	Physical state	Odor	Water Solubility	Timing of effects
Chlorine	Yellow-green	**Gas**, with pressure and cooling can be liquid	Strong bleach	Intermediate	Immediate irritation, pulmonary edema 2-24hrs later
Phosgene	Colorless or white to pale-yellow cloud	**Gas**, with pressure and cooling can be liquid	Freshly mown hay, green corn	Poor	Delayed up to 48hrs (usually 2-6 hrs)
Diphosgene	Colorless	Gas	Freshly mown hay, green corn	Poor	Delayed up to 48hrs (usually 2-6 hrs)

Effects depend on water solubility

DANGER

CHLORINE

CAUSE BURNS
SEVERE EYE HAZARD
MAY BE FATAL IF INHALED

Traffic or rail accidents, spills, or other disasters

Short-term + high-level exposures

http://www.newschannel5.com/news/local-news/shelter-in-place-in-effect-after-spill-on-i-24

U.S.

nytimes.com

Cloud Rising From Train Wreck, Then Death and a Ghost Town

By ARIEL HART and MATTHEW L. WALD JAN. 8, 2005

GRANITEVILLE, South Carolina

- **Nine people died**
- **72 were hospitalized in nine hospitals**
- **525 were examined as outpatients**
- **Fifty-one people (8%) had a severe medical outcome**

Wenck MA, Van Sickle D, Drociuk D, et al. Rapid Assessment of Exposure to Chlorine Released from a Train Derailment and Resulting Health Impact. *Public Health Reports*. 2007;122(6):784-792.

60 tons of chlorine

Environ Res. 2002 Feb;88(2):89-93.

Acute accidental exposure to chlorine gas in the Southeast of Turkey: a study of 106 cases.

Güloğlu C[1], Kara IH, Erten PG.

22 April 1915, North of Ypres, Belgium

The Daily Mirror

CERTIFIED CIRCULATION LARGER THAN ANY OTHER PICTURE PAPER IN THE WORLD

No. 3,611. FRIDAY, MAY 21, 1915 One Halfpenny.

"DEVILRY, THY NAME IS GERMANY!": SOLDIERS, TRAPPED BY A GAS CLOUD, LIE UNCONSCIOUS IN THE TRENCHES.

Regime chlorine gas attack kills 100 in Syria's Idlib

aa.com.tr

UK, France call for emergency meeting of UN Security Council

home > todays headlines, middle east 04.04.2017 Ali H. M.Abo Rezeg

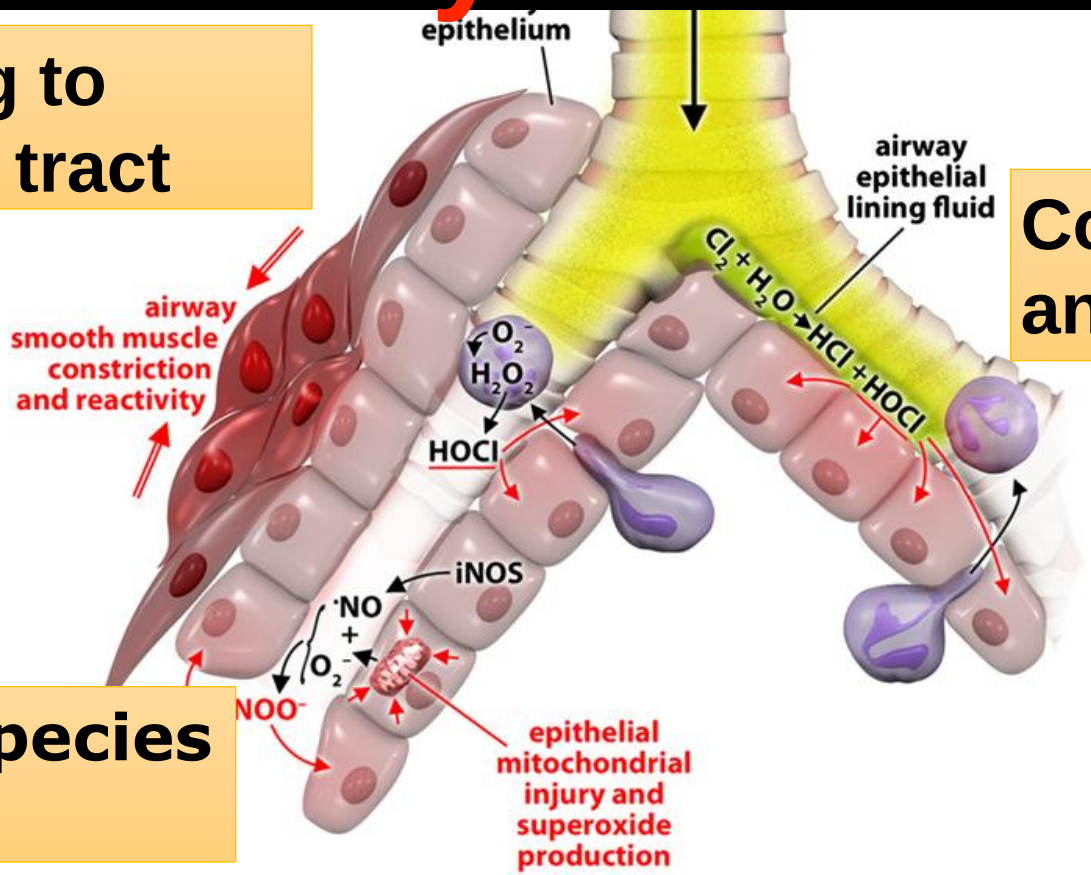
Workplace and public (swimming pools)

Log-term, low-level exposure

https://www.researchgate.net

Cough
bronchospasm
pulmonary edema

Moderately irritating to upper respiratory tract



Combines with water and forms HCl

Reactive oxygen species (ROS)

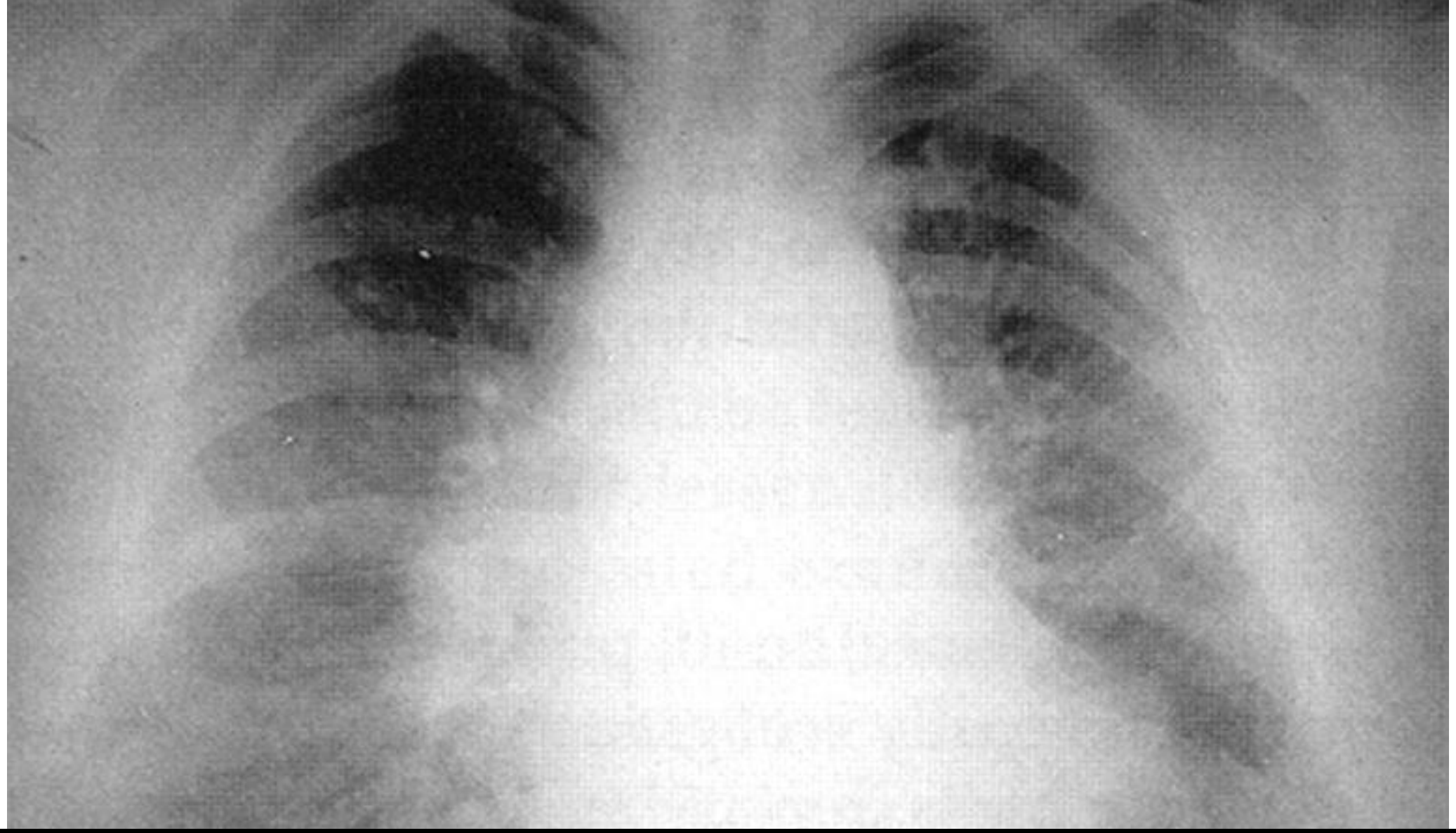
Low-level (3-5%, 1-15 ppm) acute exposure

Manifestations are as follows:

- Eye tearing, nose and throat irritation
- Sneezing
- Excess salivation
- General excitement or restlessness

High-level (20%, >30 ppm) acute exposure

- Dyspnea: Upper airway swelling and obstruction may occur
- Violent cough
- Nausea and vomiting (with the smell of chlorine in emesis)
- Lightheadedness/ Headache
- Chest pain or retrosternal burning
- Muscle weakness
- Abdominal discomfort
- Dermatitis (with liquid exposure): Corneal burns and ulcerations
- Esophageal perforation



Acute lung injury and delayed pulmonary edema

Pulmonary Irritants

Management

- Decontamination: not required unless concentrated liquid – **level B-C**
- Eye decontamination
- No Antidote
- Management:
  - Oxygen and supportive care
  - Nebulized sodium bicarbonate
  - Bronchodilators
  - Steroids
  - Antibiotics

Pulmonary Irritants

Phosgene (CG)

- Phosgene produces injury by hydrolysis in the lungs to hydrochloric acid
- Non-cardiogenic pulmonary edema
- Hypoxemia
- Respiratory failure
- Hypovolemic shock
- No antidote

Cyanides (Chemical Asphyxiants)

Chemical asphyxiants

- Asphyxiants
  - **A. Simple: affect the respiratory system alone**
    - Carbon Dioxide
  - **B. Systemic:**
    - Carbon monoxide, Cyanide, Hydrogen Sulfide, Azides

- Symptoms
  - Fast breathing, tachycardia
  - acidemia, hypoxia

Cyanides (Chemical Asphyxiants)

- Relatively ineffective because of rapid dispersion
- Extremely potent
- Routes of exposure: inhalation, ingestion, dermal, and parental
- Colorless gases
  - Hydrogen Cyanide (HCN)
  - Cyanogen chloride (CNCl)
- Inorganic cyanide salts: Na, K, and Ca CN

Cyanides (Chemical Asphyxiants)
Mechanism of Action

- Inhibit succinct acid dehydrogenase, carbonic anhydrase, and superoxide dismutase
- Inhibit cytochrome oxidase at cytochrome a3
  - Inability to utilize oxygen by the cell
  - No ATP formation
  - Hydrogen ion accumulation > acidemia (**lactic acidosis)**
- Inhibit glutamate decarboxylase > decrease brain GABA > **seizures**
- Activate NMDA receptors directly
- Activate voltage sensitive calcium channels: reactive oxygen species and nitrous oxide

Cyanides (Chemical Asphyxiants)

Clinical Manifestations

- Time of onset seconds
- Initial non-specific symptoms
  - **CNS (progressive hypoxia)**: headache, anxiety, agitation, confusion, coma
  - **CVS:** tachycardia followed by bradycardia and hypotension, shock
  - **Lungs:** pulmonary edema, apnea
- Salts can produce corrosive injuries
- Delayed neurologic sequelae of survivors (Parkinsonism)

Cyanides (Chemical Asphyxiants)

Decontamination

- Remove the patient from the area
- Remove the clothes
- Wash with soap and water
- Ensure adequate ventilation with 100% oxygen

CYANIDE ANTIDOTE KIT

Amyl Nitrite Pearls

Sodium Nitrite (300 mg= 10 cc)

Sodium Thiosulfate (12.5 gm = 50 cc)

Cyanides (Chemical Asphyxiants)
Treatment

- **Cyanide Kit**

  - **Amy nitrite and sodium nitrite**
    - Produce methemoglobinemia: CN has more affinity to methemoglobin than cytochrome-oxidase
    - Main adverse effect: vasodilation & hypotension
  - **Sodium thiosulfate**
    - Convert cyanide to **thiocyanate:** less toxic and excreted in the urine

Cyanides (Chemical Asphyxiants)

Treatment

- **Hydroxocobalamin:**

  - Bind cyanide to produce **cyanocobalamin:** excreted in the urine and bile

Summary

- Preparation is the key element in such incidents
- Personal Protective Equipment are mandatory for your safety.. Know which one to choose
- Detection and identification of the agent optimize the medical care

Not all of us
can do great
things
but we can do
small things
with great love

MOTHER TERESA