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Recognition and Initial Management of Chemical Casualties

Emphasis: Organophosphates

Health	Threats/Agents
Readiness	Control

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Objectives

- Describe types of chemical warfare agents
- Recognize signs and symptoms of exposure
- Describe how to manage, decontaminate, and treat the victims of a chemical agent attack

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Overview

- Historical Perspective
- Nerve agents
- Vesicants
- Cyanide
- Pulmonary Intoxicants

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World War 1 First large-scale use

- Chlorine, 168 Tons
- Ypres, Belgium; April 1915
- 20,000 Casualties
- 5,000 Deaths
- 5 mile Front

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Protective Equipment

- Masks
- Skin protective equipment
- Very effective at limiting casualties
- Allowed soldiers to operate in chemically toxic environment



Figure 1-3. Contamination avoidance and fluid protective suit.





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Chemical Casualties in World War I

	Non-fatal	Deaths
British Empire	180,597	8,109
France	182,000	8,000
Russia	419,340	56,000
Italy	55,373	4,627
United States	71,345	1,462
Austria-Hungary	97,000	3,000
Germany	191,000	9,000

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- ### A Few Recent Experiences
- Battlefield
 - Egypt v. Yemen
 - U.S. in Vietnam (tear gas in tunnels)
 - Iraq v. Iran
 - Iraq v. Kurds
 - Terrorism
 - Aum Shinrikyo (12 deaths)
 - Counter terrorism
 - Moscow theater hostage incident (117 hostage deaths)

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- ### The Domestic Threat
- 25 to 30 countries have active chemical programs
 - Cheap, easy to use and transport
 - Government terrorist support
 - Dual use technology
 - Not if but when

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- ### Chemical Warfare Agents are
- Lethal
 - Available
 - Defeatable
 - Protection/Decontamination
 - Early Recognition
 - Medical Management

Current Domestic Preparedness

- Training lacking
 - Agent recognition
 - Patient management
 - Patient decontamination
 - Patient transport
- Supplies
 - Antidote
 - Protective gear
 - Decontamination

Chemical Agents

- Chemicals used in military operations to kill, injure, or incapacitate
- Incapacitating vs. Lethal (or Both)
- Local vs. Systemic Effects

Chemical Warfare Agents

- | | |
|------------------|-------------------------|
| • Nerve | Tabun, Sarin, Soman, VX |
| • Cyanide | H. Cyanide, Cyanogen |
| • Pulmonary | Phosgene, Chlorine |
| • Miscellaneous | Ammonia |
| • Vesicants | Mustard, Lewisite |
| • Riot Control | Mace, Pepper Spray |
| • Incapacitating | BZ, fentanyl |

Vesicants

- Blistering
- Military vesicants
 - Sulfur mustard
 - Lewisite
- Other vesicants
 - Poison ivy
 - Industrial chemicals
 - cancer chemotherapeutics
 - Sunlight



Mustard

- Vapor and liquid threat
- Latent period between exposure and effects
- Topical eye, lung, and skin damage
- Systemically toxic; similar to radiation
- Treatment is symptomatic



Skin decontamination

- Part of supportive treatment
- Physical removal by whatever means
 - Remove clothing
 - flush skin
- Must be done within minutes to prevent damage
- Decon later will not prevent illness, but will prevent cross-contamination

Management Skin

- Soothing cream/lotion
- Frequent irrigation
- Topical antibiotics
- Systemic analgesics
- Do not over hydrate; not a thermal burn

Management Eyes

- Topical mydriatics
- Topical antibiotics
- Vaseline on lid edges
- Topical steroids

Management Airways

- Steam, cough suppressant for mild
- Oxygen
- Assisted ventilation
- Early intubation
- Bronchodilators (steroids)
- Antibiotics AFTER organism identified

Lewisite

- Causes severe irritation to eyes, skin and airways immediately on exposure
- Vesicant effects similar to sulfur mustard
 - Produces skin blisters, eye and lung injury
- No bone marrow effects



Lewisite

- Skin irritation > mustard
- Tissue necrosis, pseudomembranes
- Increased capillary permeability
 - Volume depletion
 - Renal and hepatic injury

Lewisite Treatment

- Immediate decontamination
- British anti-Lewisite (BAL) for systemic effects

Cyanide

- Widely used throughout the US
 - Printing
 - Agriculture
 - Photography
 - Manufacturing of paper and plastics; many other uses
- Over 300,000 tons produced annually
- Combustion byproduct of burning synthetic materials (e.g. plastics)

Acute cyanide poisoning

- High concentration (inhalation)
 - 15 seconds - hyperpnea
 - 30 seconds - seizures
 - 3 to 5 minutes - breathing ceases
 - 6 to 10 minutes - asytle and death
- Normal or dilated pupils
- Initial absence of cyanosis

Cyanide treatment protocol

- Remove from area and remove clothing
- If conscious and breathing - oxygen, IV fluids and observe, no antidotes
- If unconscious - oxygen, bag mask ventilate
 - Amyl nitrite - give until IV started
 - Sodium Nitrite IV
 - Sodium Thiosulfate
 - Airway management, Bicarb, fluids

Pulmonary intoxicants

- Agents
 - Phosgene
 - Chlorine
- Cause severe life-threatening after inhalation
- Severe effects delayed
- Treatment is supportive

Phosgene

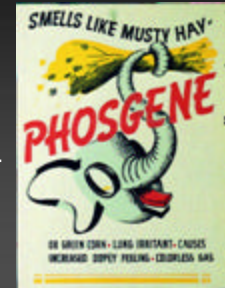
- Manufacturing of dyes, coal tar, pesticides and pharmaceuticals
- Used in WWI
- 1984 Bhopal tragedy
 - methylisocyanate released
 - 150,000 affected
 - 3,300 killed
 - Phosgene and isocyanate injuries



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Phosgene characteristics

- Colorless gas
- Freshly mown hay
- Four times heavier than air
- Gas above 47 degrees F



Decontamination: Phosgene

- vapor - fresh air
- liquid - copious water irrigation

Phosgene Hospital Treatment

- ABCs
- Chest X-ray, arterial blood gases
- Airway management (oxygen, intubation, PEEP)
- IV fluids—Treat hypotension/manage hydration
- No diuretics
- Bronchodilators
- NO EXERTION
- No long-term effects in uncomplicated cases

Chlorine

- Significant irritant to the eyes and respiratory tract
- Characteristic pungent odor
- Used in swimming pools and laboratories
- Industrial exposures may produce large numbers of casualties



Chlorine Characteristics

- Properties
 - Greenish-yellow gas
 - Less alkaline than ammonia
 - Chlorine + water = HCl + Free oxygen radicals
 - 30x more irritating to lungs than HCl
- Effects
 - Eye irritation, cough, SOB, wheezing
 - 12 to 24 hours, non-cardiogenic pulmonary edema; sudden death due to hypoxia

Chlorine Treatment

- Remove from source of exposure
- ABCs
- Flush skin and eyes with water
- Oxygen, cool mist, bronchodilators
- Airway management (intubation, PEEP)
- Hydration



Organophosphates

- Acetylcholinesterase inhibitors --used as pesticides.
- Acute effects-->SLUD (salivation, lacrimation, urination, defecation) due to *muscarinic activation*; *fasciculations also occur due to nicotinic activation*
- Intermediate (24-96 hrs post-exposure)--> *weakness due to nicotinic activation*

Organophosphates

- Chronic effects (2-4 wks after acute or chronic exposure.)
 - rapidly progressing *axonal neuropathy*; *predominantly motor*
 - CNS involved leading to *ataxia and spasticity*; this is attributed to *phosphorylation and inhibition of a neuropathy target esterase (NTE) and impairment of axonal transport*
 - *Chronically exposed persons should under AChE level testing*

Nerve Agents

- Tabun (GA), Sarin (GB), Soman (GD), VX
- Most toxic chemical agents
- Penetrate skin, eyes, lungs
- Loss of consciousness, seizures, apnea, death after severe exposure
- Diagnosis made clinically; confirmed in laboratory (cholinesterase)

MASSACRE BY POISON GAS



Many die, 1,200
collapse in Tokyo
Tube attack

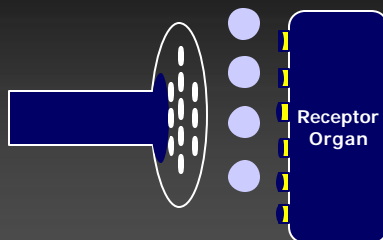
Nerve Agents

- Are organophosphates
- Are similar to insecticides
 - Malathion
 - Diazinon
 - Chlorpyrifos
- Insecticides easily obtained and commonly used in the community

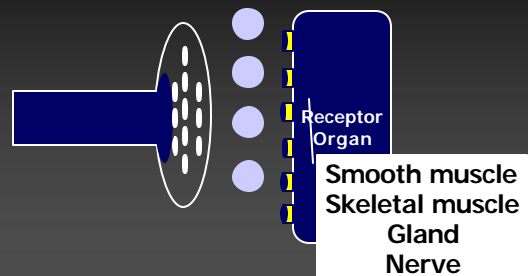
Nerve Agents

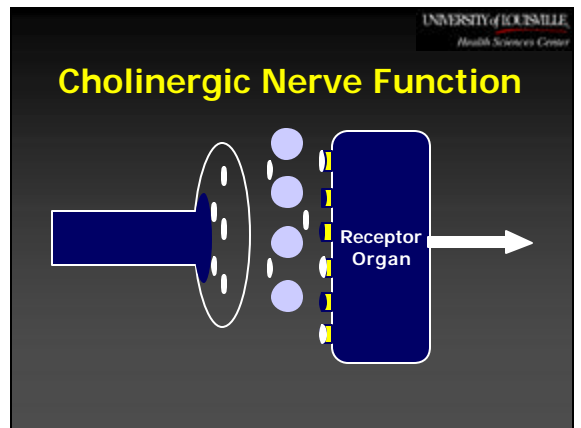
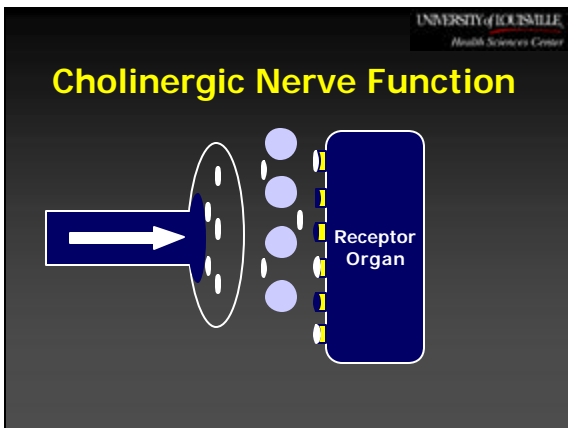
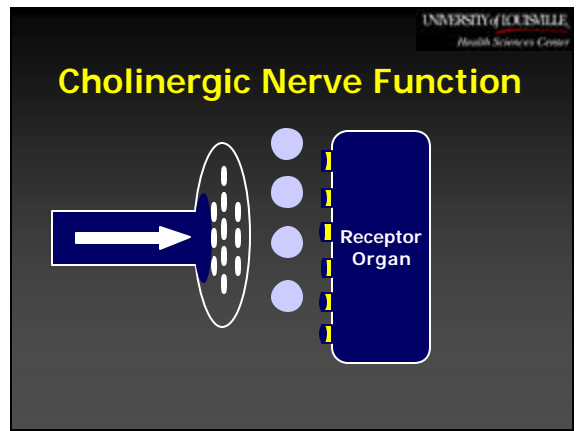
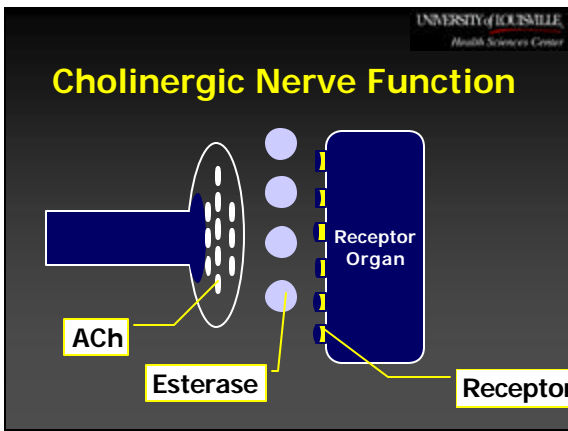
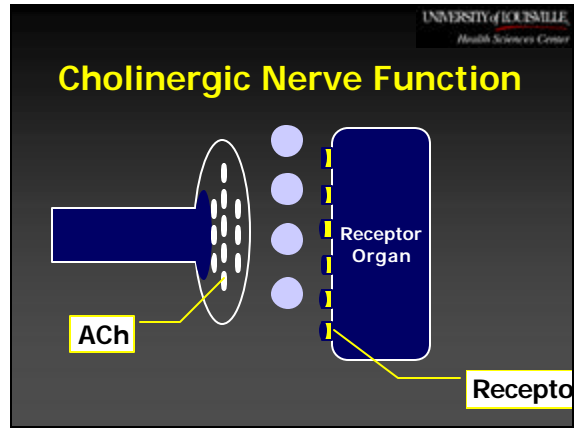
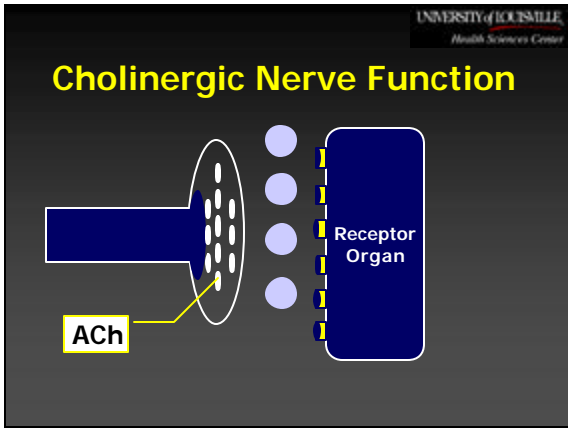
- Liquids at normal ambient temperatures
- G-agents volatile, non-persistent; vapor and liquid threat
- VX fairly non-volatile, persistent; liquid threat
- Vapor heavier than air

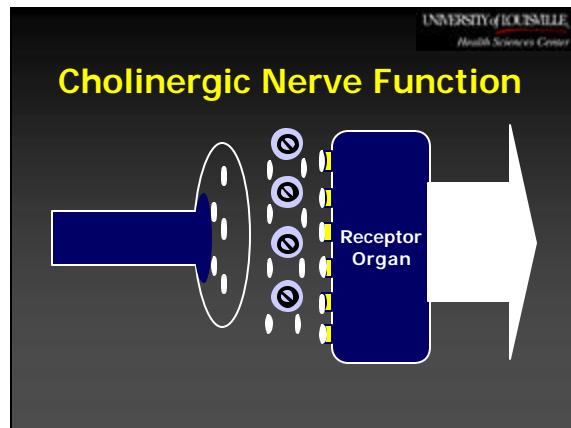
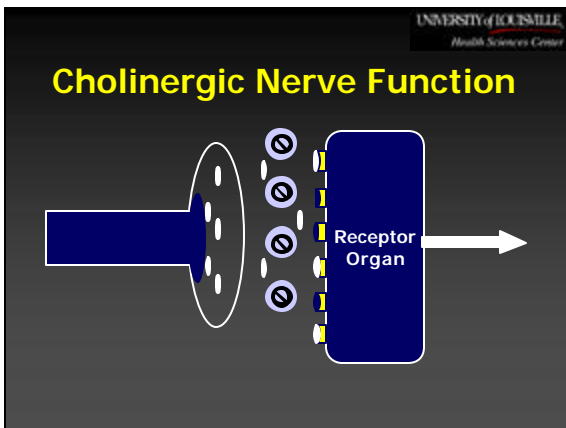
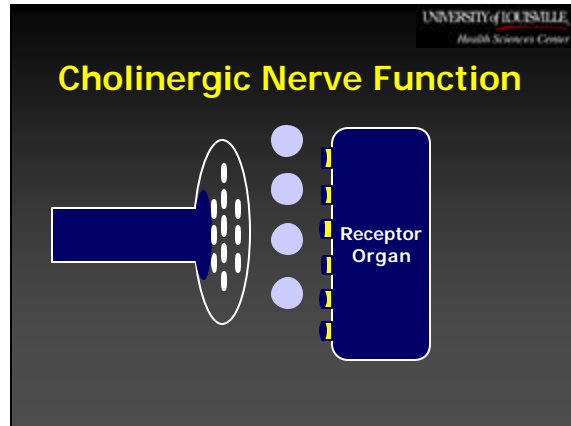
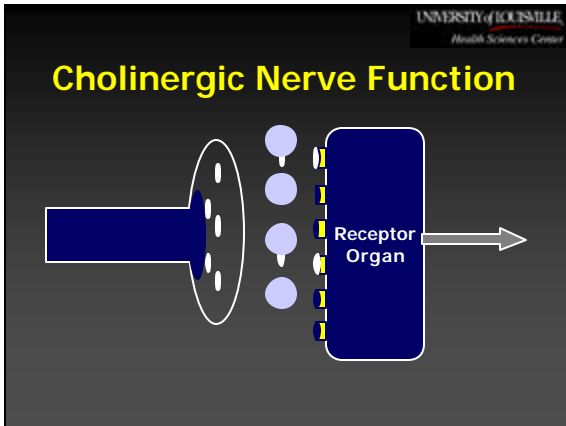
Cholinergic Nerve Function



Cholinergic Nerve Function







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- ### Organs with cholinergic receptors
- Muscarinic
 - smooth muscles
 - glands
 - Nicotinic
 - skeletal muscles
 - ganglia

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- ### Muscarinic Sites
- Miosis
 - Bronchoconstriction (shortness of breath)
 - Gastrointestinal hyperactivity (nausea, vomiting, and diarrhea)
-
- A close-up photograph of a human eye. The pupils are significantly constricted, which is a clinical sign of muscarinic receptor activation (miosis).

Nicotinic Sites

- Skeletal muscles
 - Fasciculations
 - Twitching
 - Weakness
 - flaccid paralysis
- Other (ganglionic)
 - Tachycardia
 - Hypertension

Other Nerve Agent Effects

- Cardiovascular
 - Tachycardia, bradycardia
 - Heart block, ventricular arrhythmias
- Central Nervous System
 - Acute
 - Loss of consciousness
 - seizures
 - Apnea
 - Prolonged (4 -6 weeks): Psychological effects

Nerve Agent Vapor Exposure

- Low Exposure
 - Miosis (dim vision, eye pain)
 - Rhinorhea
 - Shortness of breath
- High exposure
 - immediate loss of consciousness, seizures, apnea, and flaccid paralysis
- Vapor effects occur within seconds, peak within minutes; no late onset

Nerve Agent: skin exposure

- Small amounts (to 18 hours)
 - Localized sweating
 - Fasciculations
 - No miosis
- Moderate amount (<LD50) (to 18 hours)
 - Gastrointestinal effects
 - Miosis uncommon
- Large amount (LD50) (<30 minutes)
 - Sudden loss of consciousness
 - Seizures
 - Apnea
 - Flaccid paralysis



Organophosphate toxicity—treatment

- ABC's
- Withhold atropine until a cardiac monitor and a defibrillator are in place. Atropine can precipitate ventricular fibrillation.
- Continuous cardiac monitoring and an ECG are necessary. Electrical pacing is the treatment of choice for ventricular tachycardia associated with a prolonged QTc.
- cleanse patients with suspected OP exposure with soap and water. Consider clothing hazardous waste .
- Healthcare providers must avoid contaminating themselves while handling patients.
- Irrigate the eyes of patients with ocular exposures using isotonic sodium chloride solution or lactated Ringer's solution. Morgan lenses can be used for eye irrigation.
- Activated charcoal
- atropine or glycopyrrolate , 2-PAM, and diazepam



Nerve Agent Treatment

Self Protection
Decontamination
ABC
Antidotes

Nerve Agents Treatment

- Self-Protection
 - Patients may bring in liquid agent: contact hazard
 - Vapor hazard
- Decontamination
 - Remove clothing
 - Soap and water
 - Diluted bleach

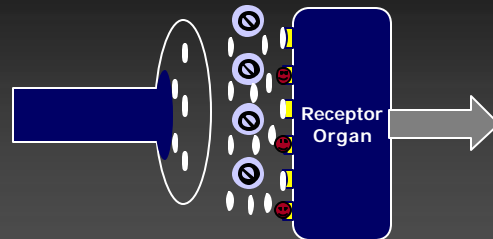
Nerve Agents Treatment

- Airway/ventilation
 - High resistance
- Antidotes
 - Atropine
 - 2-PAM Cl
 - Diazepam

Atropine

- Antagonizes muscarinic effects
- Dries secretions; relaxes smooth muscle
- Given IV, IM, ET
 - No effect on pupils
 - No effect on skeletal muscles
 - IV in hypoxic patient may lead to ventricular fibrillation

Atropine at Muscarinic Receptors



Atropine

- Starting dose- 2 mg
- Maximum cumulative dose - 20 mg
 - (Insecticide poisoning requires much more)
- Side effects in normal people
 - Tachycardia
 - Mydriasis
 - Decreased secretions and sweating

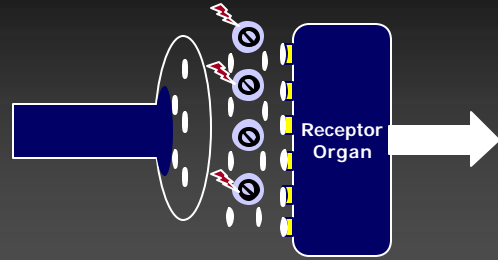
Atropine How Much?

- Until secretions are drying or dry
- Until ventilation is "easy"
 - If conscious or casualty is comfortable
- Do not rely on heart rate/pupil size

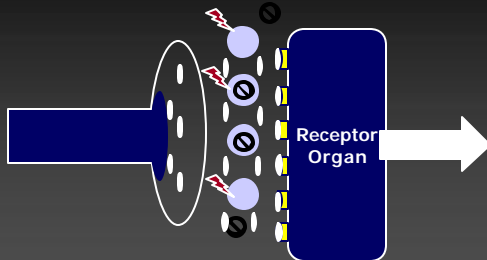
Pralidoxime Chloride (2 PAM Cl)

- Removing agent from AChE (if no aging)
- 1 gram slowly (20-30 minutes) in IV infusion
 - Hypertension with rapid infusion
- Little effect at muscarinic sites
- Helps at Nicotinic sites
- Aging

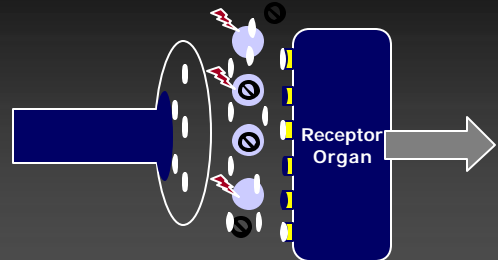
2 PAM Cl



2 PAM Cl



2 PAM Cl



Antidotal issues




Mark 1 Autoinjectors

- Atropine 2 mg
- 600 mg 2 PAM Cl
- Spring loading leads to injection under high pressure results in more rapid absorption
- FDA has approved the autoinjector for civilian use
 - (local protocols)

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Diazepam autoinjector



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Diazepam

- Decreases seizure activity
- Reduces seizure-induced brain injury
- Give to severely-intoxicated casualties whether having seizures or not

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Nerve Agent: Age related treatment Diazepam

- Infants > 1 m to 5 y 0.2 to 0.5 mg/kg IV (max 5 mg) q 2 to 5 min
- Children > 5 years 1 mg IV (max 10 mg) q 2 to 5 min

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Nerve Agent Treatment protocols No signs or symptoms

- Reassure
- Observe
 - Vapor : 1 hour
 - Liquid: Up to 18 hours

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Nerve Agent Treatment protocols Mild vapor exposure

- Miosis, rhinorhea - observation only
- Increasing shortness of breath
 - 1 mark 1 Kit
 - or
 - 2 mg Atropine IV or IM and
 - 1 gm 2 PAM Cl IV
- Parenteral atropine will not reverse miosis - need homatropine or atropine ophthalmic ointment

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Nerve Agent Treatment protocols Mild liquid exposure

- Localized fasciculations and sweating
- One Mark 1 kit
- or
- 1 gram 2 PAM Cl IV
- 2 mg atropine IM or IV

Nerve Agent Treatment protocols moderate vapor exposure

- Miosis, rhinorhea, SOB, wheezing, secretions, muscle weakness, GI symptoms
- One or two Mark 1 kits or
- Give IV or IM:
 - 2 to 4 mg atropine
 - 1 gm 2 PAM Cl infusion

Nerve Agent Treatment protocols moderate vapor exposure

- Atropine: repeat doses every 5 to 10 minutes
- 2 PAM Cl: repeat 1 gm IV infusion at hourly intervals (for a total of 3 doses)

Nerve Agent Treatment protocols moderate liquid exposure

- Vomiting, diarrhea
- 2 mg atropine
 - repeat atropine in 5 to 10 minutes if effects worsen
- 600 mg 2 PAM Cl IM or 1 gm IV

Nerve Agent Treatment protocols Severe vapor or liquid

- Unconscious, seizures, flaccid, apnea
- Three Mark 1 kits or
- 6 mg atropine and 1 gram 2 PAM Cl
- Airway
- Ventilation/oxygen
- Consider diazepam 10 mg IM (2 to 5 mg IV)
- Repeat atropine every 5-10 minutes as needed
- repeat 2 PAM Cl in one hour

Nerve Agent: Age related treatment Atropine

- | | |
|------------------------------|------------|
| • Infant (<2) | 0.5 mg IM |
| • IV for infants or children | 0.02 mg/kg |
| • Child (2 to 10) | 1.0 mg IM |
| • Adolescent (>10) | 2.0 mg |
| • Elderly | 1.0 mg IM |

Nerve Agent: Age related treatment 2 PAM Cl

- | | |
|----------------|----------------------|
| • < 20 kg | 15 mg/kg IV |
| • > 20 kg | 600 mg IM |
| • autoinjector | |
| • Elderly IV | 1/2 dose (7.5 mg/kg) |
- 2 PAM Cl induced hypertension
 - Phentolamine Adult 5 mg IV
 - Child 1 mg IV

Differential diagnosis

- *"At chemical emergencies, we should not think only of war gas symptoms, ...problems such as heat stroke, (infectious) fever, listlessness and fatigue caused by activity, the presence of acute stomach symptoms without external signs of injury, can appear, either alone or along with the picture of some slight poisoning from some war gas such as nerve gas, and mislead us."*

Chemical Agent Detection

- Recognition
 - Signs and symptoms
 - Detectors/monitors
- Purposes of detection equipment
 - Hazard assessment
 - Levels of protection
 - Need for decon
- No substitute for observation of decon by trained personnel

About triage

- *Immediate:* Casualties need lifesaving measures performed without delay if they are to survive.
- *Delayed:* Casualties can wait for definitive treatment without causing additional harm.
- *Expectant:* Casualties will not survive or will require extensive resources and time if they are to be saved.
- *Minor:* Casualties are generally ambulatory and are only slightly injured



Nerve agent triage

- **Group A:** *"confusion or comatose, with severe muscarinic or nicotinic symptoms"*
 1. CPR
 2. IV atropine 4 mg
 3. IV (oxime therapy)
 4. More atropine q 5-10 minutes
 5. DO NOT use mydriatic eye drops
 6. IM diazepam 10 mg

Nerve agent triage

- **Group B:** *"fully conscious, can walk, severe muscarinic symptoms. Suffer from shortness of breath, cough, nausea, vomit, severe miosis and blurred vision"*
 1. IM atropine 4 mg AND IM oxime, ideally in same syringe; IV if few patients to see
 2. IM diazepam 10 mg
 3. Salbutamol spray x 4

Nerve agent triage

- **Group C:** *“relatively well, no systemic poisoning. Cholinesterase is normal. Not vomiting. Suffer from pupillary retraction, slightly blurred vision and shortness of breath. May be treated as outpatients.”*
 - 1. Atropine sulphate tablets 0.6 mg qid x 24 h
 - 2. Cycloplegic eye drops prn headache q 6 h x 1 -2 days

Nerve agent triage

- **Group D:** *“hypochondriacs who delay treatment for the other three groups by having to be patiently reassured regarding their good health”*
 - *“taken to a separate area where they are either given military leave or are transported to their operation zone ”*

Relative death rate

- *“The highest number of deaths may in fact result from [Group C] rather than from the patients who die due to chemical contamination.”*

Overwhelming the system

- In the Hosseiniyeh attack in 1987, the triage system completely broke down. Over 300 “severe” patients arrived in a 5-hour period with 1700 other patients. Total gridlock ensued.
- Lesson: bypass plan crucial

Cases

- Soldier admitted with bag valve mask for respiration, severe pulmonary secretions, miosis.
- Cholinesterase (Ellman method) went from 250 U/L to 1200 U/L in 15 minutes
- 2 hours post- treatment patient *“began recovering and introduced himself”*

Cases

- Soldier admitted in coma with miosis, respiration provided by bag valve mask
- Cholinesterase went from 300 to 1200 U/L
- A few hours later he was able to give his name and unit

Cases

- Soldier arrived *“not apparently severely poisoned”*, but developed *“aggressive psychosis”*
- Had to be restrained, received 50 mg chlorpromazine and biperiden
- Next day at recovery unit in Ahwaz *“behaved very courteously”*

Recovery

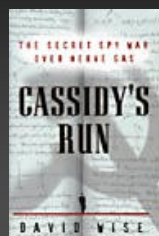
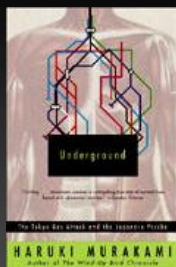
- *“The most pleasant moment of my work was the time when the wounded in critical condition were beginning to recover. One must remember that their recovery stage was exciting and a dangerous one for the casualties. They vomited and needed intensive care until full recovery.”*

Lessons: similar to our teachings

- Must separate out psychologic casualties
- Rapid treatment means survival
- A system of care, including triage and evacuation, is crucial
- Mild nerve agent casualties will recover quickly
- Worthwhile retaining cyanide instruction

Lessons differing from our teachings

- Use more atropine and faster than US advises
- Use less oxime, and later [possibly due to necessity]
- A few late cardiopulmonary arrests
- Cyanide can be a confounder
- Use pulse as guide for atropinization
- Nerve agents are not photogenic!
- Keep surviving troops out of combat zone for *“weeks”*



Center for Health Hazards Preparedness (CHHP)

If you have any questions about this program send an email to chhp@louisville.edu

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