

CHAPTER 6

HAZARDOUS MATERIALS PROTOCOLS

INTRODUCTION

This chapter is exclusively for EMS responders with additional training. This material is NOT covered in the national DOT training program. Additionally, the risks involved (to the responder) in caring for these patients are far greater than the average patient.

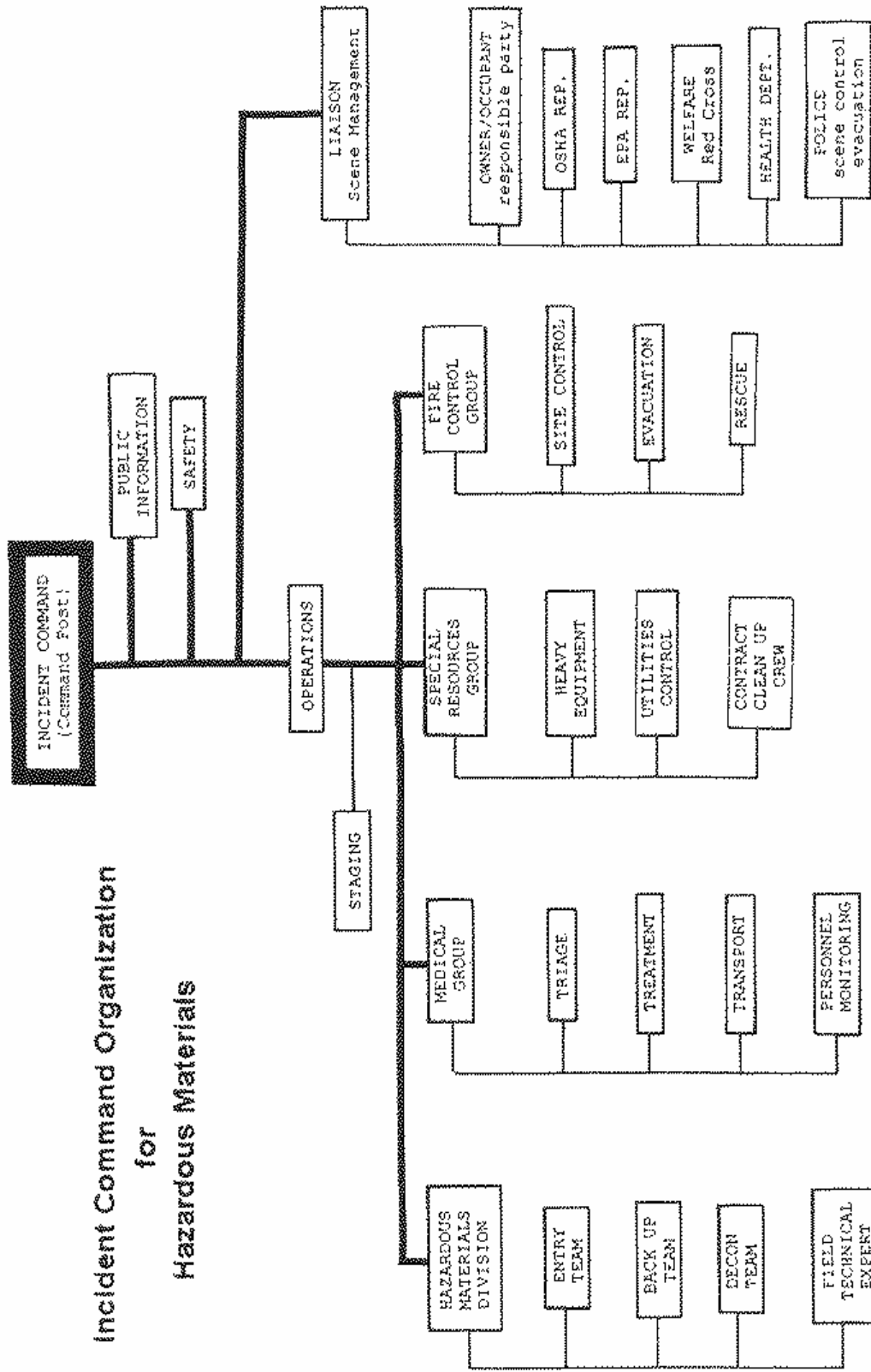
This chapter must ONLY be used by specially trained responders who work regularly with a Hazardous Materials Response Team. Those are the only personnel who will have adequate equipment, protective gear, and training to safely approach many of these patients. Safety of the rescuer is of primary importance. Unconscious or dead responders are no help to anyone!

It is critical for the average EMS responder to recognize a hazardous materials incident and notify the appropriate personnel. The following situations should raise suspicions of hazardous materials being involved:

- A. Train derailments.
- B. Vehicle related incidents involving Department of Transportation (DOT) placarded vehicles or labeled substances. Any incident involving a vehicle which is used for transporting goods that has a cargo suspected to be a hazardous material whether the vehicle is placarded or not.
- C. Vehicle related incidents involving unknown loads or unusual containers including liquid and gas transporters.
- D. Incidents involving unknown or suspicious substances or odors, especially if there is a spill or leak.
- E. Incidents involving storage areas which may contain hazardous materials.
- F. Scenes with multiple victims becoming ill for unknown reasons.
- G. Scenes involving explosions or explosive substances.
- H. Incidents involving aircraft -- "crop dusters" are particularly suspect.
- I. School laboratories often contain a number of dangerous chemicals.

The circumstances listed above could prove a deadly trap to the eager first responder or EMT. Restrain yourself and notify the proper authorities, before further investigation. The appropriate response to the hazardous materials incident goes against every instinct of the prehospital care provider. You can't run in to rescue someone if you might be killed in the process, but it is extremely difficult to stand back and alert authorities when your usual approach is to run in. Mentally prepare yourself ahead of time. The urge to run in is not worth your life!

INCIDENT COMMAND ORGANIZATION



**Incident Command Organization
for
Hazardous Materials**

APPROACH TO HAZARDOUS MATERIALS

GUIDELINES FOR HAZARDOUS MATERIALS RESPONSE

Approach to scene

- A. Prepare through familiarization with authorized Department of Transportation (DOT) placards, labels, 704 System, and observe site proximity for the presence of such labels.
- B. Obtain a copy of the DOT Emergency Response Guidebook and become proficient using it. Keep this book available in the vehicle at all times. Do not rely on your memory.
- C. Be suspicious of large trucks or tractor-trailers transporting goods even if placards are not visible. At buildings or locations with NFPA 704 placard, consider hazardous condition, prior to entering scene.
- D. If any serious consideration of hazardous materials contamination, contact dispatch to request Hazardous Materials Response Team (HMRT) and fire department immediately. Await arrival of responding units prior to any advancement into the scene.
- E. If dispatch information is received that a scene has hazardous materials involved or for any reason you suspect the presence of such materials **DO NOT ENTER THE SCENE!** The following guidelines should improve safety:
 - 1. Observe posted barriers. (**DO NOT CROSS BARRIER TAPE!**)
 - 2. Approach uphill and upwind from the incident and only when requested or assisted by the HMRT or Incident Commander.
 - 3. For leaks from drums, small containers or tanks -- maintain 600 to 800 foot distance.
 - 4. For large leaks or spills, maintain at least 1000 to 1500 feet distance.
 - 5. In the event of a fire involving hazardous materials, maintain 1/2 to 1 mile distance.
- F. An area should be established for staging ambulances as soon as possible. All crews and units shall stay in that area until advised by the HMRT or Medical Treatment Officer as designated by the Incident Command System (ICS).

Scene management

- A. Once an "Exclusion (Hot) Zone" is established there should be only one entrance and exit into that area which will be controlled by the HMRT exclusively.
- B. "Contamination Reduction (Warm) Zone" will be established for decontamination activities. Only personnel properly attired and trained for such activities will be admitted.
- C. A "Support (Cold) Zone" will contain other functions of the Incident Command System including the Staging and Treatment Areas.

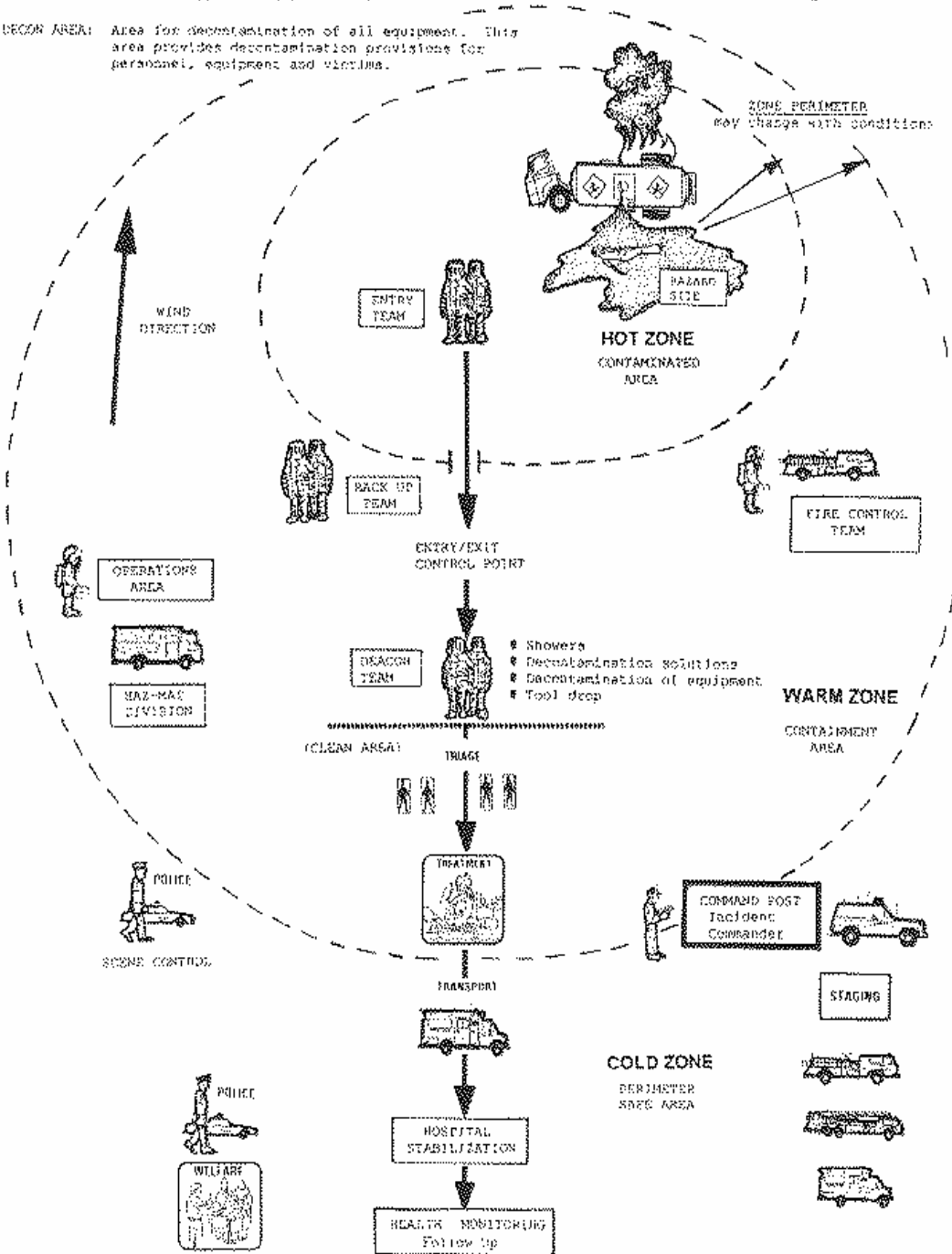
- D. A "Safety Perimeter" or "Crowd Control Line" will be established at the outermost safe limits for the incident area. Only people directly involved with the incident will be admitted.
- E. Keep non-contaminated people away from the incident scene or move them uphill, upwind, at a distance that is determined to be safe.
- F. Avoid gaseous clouds, concentrations of vapor, and smoke.
- G. Do not assume that if you can't see it or smell it -- it is not harmful.
- H. Keep contaminated victims away from noncontaminated people. A public address system may be necessary. Do not allow contaminated individuals, equipment, or materials to leave the "Hot Zone" until it is determined by the HMRT that it can be done safely.
- I. Do not enter an area without permission from the HMRT (or IC) and the proper protective gear.
- J. If you find yourself in a situation where you have been contaminated or you are within a "Hot Zone" on a hazmat scene, back out to a safe position, but DO NOT LEAVE THE SCENE. Isolate yourself from others and contact the HMRT for decontamination (decon) procedures.
- K. Any information obtained about the material should be passed on to the HMRT and/or Haz Mat Paramedic to be utilized in scene mitigation.
- L. All members of HMRT will be medically evaluated and rehabilitated prior to exiting the scene. This will be managed by Haz Mat EMS providers, Medical/Rehab Sector, and the Incident Commander.
- M. *Beware of changing conditions (weather, fire size, or intensity, etc.). Be ready to retreat rapidly by way of predetermined egresses.*

Decontamination

- A. When the HMRT dictates that the material involved requires proper decontamination, all victims must be decontaminated by going through the HMRT decon process prior to leaving the scene. *No patient will be transferred to the ambulance or emergency department until they have gone through this process.* Failure to complete this step could lead to numerous unneeded exposures and a compounding of an already serious problem.
- B. All personnel involved with the decon process should be in proper protective equipment. In general, decon personnel should wear the same or one level of protection lower than the HMRT entry team.
- C. Prior to transport the receiving hospital must be notified of the situation, material involved, and that the patient has been through the decontamination process.
- D. As soon as patient numbers, information on the material, and extent of exposure has been determined, Haz Mat EMS personnel or the Medical Treatment Officer will notify the receiving hospital(s).

Hazardous Materials Incident

- ♦ **HOT ZONE:** Proper level of protective clothing and assigned task.
- ♦ **WARM ZONE:** Operations area for assigned support functions.
- ♦ **COLD ZONE:** Safe perimeter area that is monitored by response team and supported by police department.
- ♦ **DECON AREA:** Area for decontamination of all equipment. This area provides decontamination provisions for personnel, equipment and victims.



MEDICAL MONITORING

The purpose of this section is to provide direction for medical personnel to perform medical monitoring of haz mat response personnel. Medical monitoring is the ongoing assessment of response personnel who are exposed to extreme environmental conditions and hazardous materials. The goal of monitoring is the early recognition and prevention of adverse effects related to a haz mat incident.

Objectives

- A. Monitor baseline vital signs and pertinent assessment findings.
- B. Identify and exclude from entry any personnel at high risk from the warm and hot zones.
- C. Recognize and treat personnel with adverse effects of on-scene activities.

Pre-Entry Monitoring

- A. Vital Signs – Blood pressure, pulse, respirations, temperature, pulse oximetry, EKG strip (if pulse abnormal or history of cardiac dysrhythmia).
- B. Skin Evaluation – Identify any rashes, open sores, wounds.
- C. Mental Status – Awake, alert, and oriented to time, place, person, and situation. Must have a steady gait.
- D. Medical History – Document history of any of the following:
 - 1. Medications, prescription or over-the-counter within the past 72 hours.
 - 2. Alcohol consumption within the past 24 hours.
 - 3. New medical treatment or diagnosis made within the past two weeks.
 - 4. Fever, nausea, vomiting diarrhea, or cough within the past 72 hours.
- E. Weight – Measure and record each person's weight.
- F. Hydration – Each person should consume 8-16 oz. Of water or diluted activity drink (1 part drink: 3 parts water).

Exclusion Guidelines

Any hot or warm zone personnel with the following conditions should be excluded from entry into respective areas.

- A. Diastolic blood pressure > 105 mm Hg.
- B. Pulse > 70% of the maximum heart rate (220-age).
- C. Respiration > 24/minute.
- D. Temperature > 37.5 C (99.5 F) oral or > 38 C (100.5 F) core.
- E. Dysrhythmia not previously known and cleared by medical direction.
- F. Any open sores, large area rashes or burns (> 10%, including sunburn), or significant wounds.

- G. Any altered mental status or unsteady gait.
- H. History of nausea, vomiting, diarrhea, fever, upper respiratory infection, heat illness, or heavy alcohol intake within past 72 hours.
- I. New or changed prescription medications within the past two weeks; over the counter cold, flu, or allergy medicines taken within the past 72 hours; or beta blockers taken within the past 72 hours without clearance from medical direction.
- J. Any alcohol within the past 6 hours.
- K. Pregnancy.
- L. Less than 6 hours sleep in the past 24 hours.

Ongoing monitoring While in Hot or Warm Zone

Any personnel noted experiencing any of the following findings should be immediately decontaminated, have their personal protective clothing removed, and be assessed.

- A. Unsteady gait, abnormal speech, abnormal behavior.
- B. Chest pain, dizziness, breathing difficulty, weakness, nausea, headache.
- C. Persistent heart rate greater than 80% of maximum calculated after resting for more than 1 minute.

Post-Entry Monitoring

- A. The same components of pre-entry monitoring should be assessed immediately and 10 minutes after decontamination and doffing of personal protection equipment. Further assessment should be completed at least every 10 minutes until heart rate is less than 75% of maximum pulse rate, and any signs of orthostasis or heat exposure have resolved.
- B. Medical direction should be contacted and further treatment and transport should be considered for:
 - 1. Body weight > 3% loss or positive orthostasis.
 - 2. Pulse rate > 85% of maximum pulse at 10 minutes.
 - 3. Temperature > 38 C (100.5 F) oral or 39 C (102 F) core.
 - 4. Nausea, vomiting, diarrhea, altered mental status, respiratory, cardiac, or dermatologic complaints.

GENERAL MEDICAL APPROACH

- A. Protect rescuers
- B. History
- C. Patient assessment
 - 1. NEED FOR DECONTAMINATION
 - 2. Airway, breathing and circulation
 - 3. Level of consciousness and gag reflex
 - 4. Secondary survey
- D. Generalized treatment
 - 1. DECONTAMINATION
 - 2. Assure airway, breathing and circulation
 - 3. Eye irrigation
 - 4. Supportive treatment -- treat signs and symptoms
 - 5. Prevention of absorption
 - a. DECONTAMINATION
 - b. Induce emesis, perform lavage
 - c. Charcoal
 - d. Cathartic
- E. Specific physiological antagonists
 - 1. Cyanide kit
 - 2. Atropine
 - 1. 2-PAM
 - 2. Methylene Blue
 - 3. Calcium gluconate
 - 6. Calcium Chloride
- F. Assess and treat for other injuries, illnesses

ACETYL CHOLINESTERASE INHIBITORSSource**A. INSECTICIDES****ORGANOPHOSPHATES**

Tetraethylpyrophosphate (TEPP)

Parathion

Phorate

Highly toxic

Disulfoton

Mevinphos

Diazinon

Coumaphos

Chlorpyrifos

Moderately toxic

Crufomate

Trichlorfon

Malathion

Low toxicity

CARBAMATES

Aldicarb

Carbofuran

High toxicity

Tirpate

Aminocarb

Befencarb

Moderately toxic

Methomyl

Carbaryl

Low toxicity

B. NON INSECTICIDE CARBAMATES

Physostigmine (Antilirium)

Neostigmine (Prostigmin)

Edrophonium (Tensilon)

C. NERVE AGENTS -- usually organophosphates

a. Tabun (GA)

b. Sarin (GB)

c. Soman (GD)

d. VX

Clinical presentation

- A. Early or mild exposure:
1. Fatigue, anorexia, nausea
 2. Vertigo, weakness
 3. Loss of concentration, blurred vision
- B. Moderate to severe exposure:
1. Muscarinic effects
 - D** --diarrhea
 - U** --Urination
 - M** --Miosis
 - B** --bradycardia, bronchorrhea, bronchospasm
 - E** --emesis
 - L** --lacrimation
 - S** --salivation, secretion, sweating
 2. Nicotinic effects -- mydriasis
 - M** -- mydriasis, muscle twitching and cramps
 - T** -- Tachycardia
 - W** -- Weakness
 - tH** -- Hypertension, Hyperglycemia
 - F** -- Fasciculations
 3. CNS effects --
 - C** --Confusion
 - C** --Convulsions
 - C** --Coma

Patient Treatment

- A. Assure safety of rescuers.
- B. Decontaminate.
- C. Airway, protect as needed.
- D. O₂, high flow (10-15 L/min). Titrate to pulse oximetry > 90%.
- E. Suction as necessary.
- F. IV -- volume expander (NS or RL), TKO or as directed.
- G. Administer atropine 0.5-2 mg IV, repeat every 5 minutes until bronchial secretions clear or signs of atropinization (hot, dry, flushed, or dilated pupils). Maximum dosage used – 20 mg.
Pediatric dose is 0.01-0.04 mg/kg, with a minimum dose of 0.1 mg IV, repeat if needed as above.
- H. In organophosphate poisoning administer pralidoxime – 1 Gm in 250 ml D5W or NS over 10-30 minutes, may need to repeat to effect.
Pediatric dose is 20-40 mg/kg up to a maximum of 1 gm administered over 10-30 minutes and repeated as necessary.
- I. Observe for seizures or pulmonary edema and treat as necessary.
Transport as soon as possible.

Responder Treatment

- A. Antidotes for the treatment of responders are available in autoinjector form for IM administration.
Mark I Kit – Atropine 2 mg, 2 Pam CL 600 mg
CANA – Diazepam 10 mg
- B. Responders experiencing mild symptoms should self-administer 1 Mark I Kit IM into a lateral thigh (or buttocks) area.
- C. Wait 10-15 minutes after the administration of the first Mark I Kit. If you are able to walk, know who you are, and where you are, you WILL NOT need a second set of Mark I injections.
- D. If symptoms are not relieved after administer one Mark I Kit; seek someone else to check symptoms and administer a second Mark I Kit.
- E. If symptoms persist 10-15 minutes after the second Mark I Kit, a “buddy” should administer the 3rd Mark I Kit.
- F. If a provider experiences SEVERE symptoms from onset, another responder should administer 3 Mark I Kits in rapid succession.
- G. Seizures should be managed by the administration of 1 CANA IM into a lateral thigh (or buttocks) area.

Special notes

- A. Organophosphates, carbamates, and **nerve gas** are absorbed rapidly through every route -- oral, conjunctival, skin, or respiratory tract. Some act directly and very rapidly, others are toxic only after being metabolized and therefore the effects may be delayed.
- B. Organophosphates and carbamates act as acetylcholinesterase inhibitors. Acetylcholinesterase is the enzyme that digests or incapacitates acetylcholine. Acetylcholine is the primary neurotransmitter for skeletal muscle, the parasympathetic nervous system, the preganglionic sympathetic nerve endings, and much of the central nervous system (CNS). With no enzyme to digest acetylcholine the nerve endings continually fire. The effects are described as "muscarinic" (parasympathetic nerve ending stimulation), "nicotinic" (striated muscle and sympathetic ganglia stimulation) and CNS stimulation.
- C. When organophosphates and carbamates bind with acetylcholinesterase, it is initially reversible. The carbamates will spontaneously hydrolyze from the cholinesterase within 48 hours. Organophosphates will not spontaneously release, and in fact the binding is only reversible for 24 - 48 hours. After that time, if no antidote (pralidoxime) has been administered, the cholinesterase will be irreversibly destroyed.

CYANIDE**Source**

- A. Pest control
 - 1. Vermicidal fumigant
 - 2. Insecticide
 - 3. Rodenticide
 - 4. Soil sterilization
 - 5. Coyote "gitter" traps

- B. Industrial uses
 - 1. Metal polish
 - 2. Electroplating
 - 3. Extracting silver and gold from ore
 - 4. Photography
 - 5. Chemical synthesis
 - 6. Removing hair from hides

- C. Fires
 - 1. Wool
 - 2. Silk
 - 3. Polyurethanes
 - 4. Polyacrylonitriles
 - 5. Horsehair

- D. Plants and fruit
 - 1. Amygdalin (Laetrile)
 - 2. Peach, cherry and apricot pits
 - 3. Apple and pear seeds

- E. Sodium nitroprusside

- F. Cigarette smoke

- G. Artificial nail removers (acetonitrile)

Clinical presentation

- A. Early or mild exposure -- odor of bitter almonds
 - 1. Respiratory -- tachypnea, hyperpnea
 - 2. CNS -- anxiety, confusion, vertigo, headache
 - 3. Cardiac -- tachy or irregular pulse
 - 4. GI -- nausea, vomiting
 - 5. Skin -- flushed, hot and dry

- B. Late or severe exposure
 - 1. Respiratory -- gasping efforts then apnea
 - 2. CNS -- seizures and coma
 - 3. Cardiac -- bradycardia and cardiovascular collapse

Treatment

- A. Assure safety of rescuers.
- B. Decontaminate.
- C. Airway, protect as needed.
- D. O₂, high flow (10-15 L/min). Pulse oximetry will be inaccurate.
- E. Utilize Cyanide Antidote Kit only with a clear indication and patient with *significant* symptoms (unconscious, confused, combative). In patient with significant symptoms:
 - 1. Administer amyl nitrite by inhalation. Crush ampule in handkerchief and hold in front of patient's mouth for 30 seconds, alternate with high flow oxygen every 30 seconds until IV established. Use fresh ampule every 3-4 minutes. Discontinue as soon as IV access established.
 - 2. IV -- volume expander (NS or RL), TKO or as directed.
 - 3. Administer sodium nitrite 300 mg (10 ml of 3% solution) IV over no less than 5 minutes. Rate should not exceed 2.0 ml/min. Administration by drip will assure the slower rate. If drip is preferred, mix sodium nitrite 300 mg in 50-100 ml NS or D5W. Begin administration at a slow rate and monitor blood pressure. Rate can be increased if blood pressure is adequate. (Target rate is 60 ml over 5-15 minutes.) Pediatric dose is 0.2 ml/kg over not less than 5 minutes, not to exceed 10 ml. Drip is preferred for the pediatric patient to avoid severe hypotension.
 - 4. Administer sodium thiosulfate 12.5 Gm (50 ml of 25% solution) IV over 10-20 minutes. Pediatric dose is 1.5 ml/kg, not to exceed 50 ml.
 - 5. Consider other antidotes as available
- F. Administer naloxone 2 mg IV.
- G. If cyanide ingested -- consider charcoal or gastric lavage.
- H. Transport as soon as possible -- may benefit from hyperbaric oxygen therapy.

Special notes

- A. Cyanide is commonly formed in many varied situations. Cyanide is a common ingredient used for pest control. It is used in metallurgy for extraction of gold and silver metals from their ores. It is used in chemical synthesis and the manufacture of many plastics. It is also found in the pits of many fruits as amygdalin, which is converted to cyanide only after it is metabolized by digestion. Finally, it has been increasingly recognized that cyanide is a byproduct of many fires; and may be a cause of death in fire victims and fire fighters more often than previously recognized.
- B. Cyanide is absorbed rapidly through every route -- oral, conjunctival, skin, or respiratory tract.
- C. Cyanide binds to iron in the ferric state. Any enzymes which cycle between ferric and ferrous states, are susceptible to inactivation by cyanide. The cyanoferric complex is relatively stable and the enzyme remains trapped in this inactive form of the enzyme. Cyanide produces cellular hypoxia by inhibiting the reoxidation of cytochrome oxidase. This is a hemoprotein with iron in the ferric state. It is also the final step of oxidative phosphorylation which provides the primary source of energy to the cell. Blocking this step causes the cell to utilize anaerobic metabolism. This leads to an increase of lactic acid, decrease of ATP, and eventually to cellular, organ, and organism death.
- D. The cytochrome oxidase-cyanide complex is dissociable. If the cyanide can be removed from the cytochrome oxidase before cellular or organism death, recovery may be the rule. The initial approach of the cyanide antidote kit is to produce methemoglobin. Both amyl nitrite and sodium nitrite will produce methemoglobinemia. This serves to attract cyanide from the cytochrome oxidase-cyanide complex to form cyanomethemoglobin complex. The methemoglobin may bind with any cyanide in the plasma, but is most effective in serving as a competitive binding site for cyanide already bound to cytochrome oxidase. Cyanomethemoglobin has relatively low toxicity. The next step in the treatment is to administer sodium thiosulfate. Sodium thiosulfate acts as a sulfur donor and permits the cyanide released from methemoglobin to combine and produce thiocyanate. The thiocyanate is relatively nontoxic and is excreted by the kidneys.
- E. Many other antidotes are currently being investigated and may be available soon. Hydroxocobalamin binds cyanide without producing methemoglobin, and does not have the side effect of significant hypotension. It is currently available in some European countries, but not in the U.S.

METHEMOGLOBINEMIA

Source

- A. Nitrites and nitrates
 - 1. Sodium nitrites
 - 2. Bismuth subnitrate (Pepto-Bismol)
 - 3. Nitroglycerin
 - 4. Nitroprusside (Nipride)
 - 5. Nitrate-rich food or water
 - 6. Silver nitrate
 - 7. Volatile nitrites
 - a. Amyl nitrite
 - b. Butyl nitrite
 - c. Isobutyl nitrite ("Rush")

- B. Local anesthetics
 - 1. Benzocaine (Unguentine, Solarcaine)
 - 2. Lidocaine (Xylocaine)
 - 3. Procaine (Novocain)

- C. Aromatic amino and nitroso compounds
 - 1. Aniline dyes (inks and shoe polishes)
 - 2. Nitrobenzene
 - 3. Phenylhydroxylamine
 - 4. Phenazopyridine (Pyridium)

- D. Miscellaneous
 - 1. Sulfonamides (Dapsone)
 - 2. Chlorates
 - 3. Phenacetin
 - 4. Primaquine
 - 5. Methylene blue (large doses)

Clinical presentation

Methemoglobin level	Signs & Symptoms
< 10%	None
10 -- 15%	Cyanosis
20 -- 40%	"Chocolate cyanosis" Headache, fatigue Weakness, dizziness
40 -- 60%	Lethargy, dyspnea Bradycardia Respiratory depression Stupor
60 -- 80%	Seizures, coma Cardiopulmonary arrest

Treatment

- A. Decontamination:
 1. Clothing removed, copious washing if external or
 2. Gastric lavage or charcoal if ingested.
- B. Airway -- protect as needed.
- C. O₂, high flow (10-15 L/min). Pulse oximetry inaccurate.
- D. IV -- volume expander (NS or RL), TKO or as directed.
- E. If patient severely confused, combative, or comatose:
 1. Administer naloxone 2 mg IV.
 2. Administer methylene blue 1-2 mg/kg of 1% sterile solution (10 mg/ml) slowly IV over at least 5 minutes. This is equivalent to 0.1-0.2 ml/kg or total 5 to 20 ml over 10 minutes.
 3. Test blood for glucose level
 4. Administer dextrose 50%, 50 ml, IV if glucose level < 60 mg/dl.
- F. Transport as soon as possible.

Special notes

- A. Nitrates and nitrites have variable rates of effect depending on the route of administration. Inhalation of the volatile nitrates cause a fall in systolic blood pressure within 30 to 60 seconds with maximum effect in 1-3 minutes. The necessary metabolism of the nitrates to the methemoglobin producing nitrites would delay the onset of symptoms. Nitrates and nitrites both produce relaxation of smooth muscle in

blood vessels, GI tract, bronchi, and ureters. This dilatation has long been utilized to treat patients with coronary artery disease (initially with amyl nitrites, now with nitroglycerin). At the higher doses, and with prolonged administration, however, methemoglobinemia can be a problem even from therapeutic administration of these medications.

- B. Methemoglobin is an abnormal hemoglobin in which the usual reduced ferrous (Fe^{++}) state of the heme molecule is oxidized to the ferric (Fe^{+++}) form. Methemoglobin cannot reversibly bind or carry oxygen or carbon dioxide. The normal physiologic level of methemoglobin is less than 1%. Methemoglobinemia is defined as a methemoglobin level greater than 1%. Levels of 2-3% have been reported from use of amyl nitrites for 5 minutes. Intravenous nitroglycerin has been reported to produce levels over 12% on occasion. The administration of sodium nitrite 600 mg IV to treat cyanide poisoning, was reported to result in a methemoglobin level of 58% in one patient. Yet for all of the exposures, very few patients require treatment for methemoglobinemia, so many factors are involved in the metabolism and physiologic response.
- C. The initial presentation of methemoglobinemia is darkened blood and a "slate gray" or "chocolate brown" cyanosis. This may be apparent only around the lips and mucous membranes. This color is the result of the pigment from the abnormal hemoglobin *not* from hypoxic cyanosis. In most normal individuals the methemoglobin level must be above 10% before the color can be distinguished.
- D. Methylene blue acts as a cofactor in a reaction to accelerate the NADPH-dependent methemoglobin reductase system. This system requires the production of reduced NADPH by the pentose phosphate shunt, the reductase enzyme and cofactor such as methylene blue. The result is the reduced (functional) form of hemoglobin being produced from the methemoglobin (nonfunctional) form.

SULFIDES

- A. Hydrogen sulfide
- B. Carbon disulfide
- C. Mercaptans
- D. Sulfides found or used in
 - 1. Sulfur springs
 - 2. Volcanic gases
 - 3. Liquid manure
 - 4. Insecticides
 - 5. Soil fumigants
 - 6. Petroleum industry
 - 7. Farming
 - 8. Jet fuels
 - 9. Metal refining
- E. Sulfides used in the manufacturing of
 - 1. Rubber
 - 2. Synthetic fabrics
 - 3. Heavy water
 - 4. Leather
 - 5. Plastics
 - 6. Asphalt

Clinical presentation

- A. Low concentration
 - 1. Irritation
 - Eye -- "gas eye," keratoconjunctivitis
 - Respiratory tract (pharyngitis, bronchitis)
 - Gastrointestinal tract
 - 2. Headache
 - 3. Nausea and vomiting
 - 4. Weakness
- B. High concentration
 - 1. Neurologic -- Agitation, seizures, coma, respiratory paralysis.
 - 2. Cardiac -- Disorders of conduction, various dysrhythmias.
 - 3. Local -- Caustic burn.

Treatment

- A. Assure safety of rescuers.
- B. Decontaminate.
- C. Airway, protect as needed.
- D. O₂, high flow (10-15 L/min). Pulse oximetry will be inaccurate.
- E. Administer amyl nitrite by inhalation. Crush ampule in handkerchief and hold in front of patient's mouth for 30 seconds, alternate with high flow oxygen every 30 second until IV established.
- F. IV -- Volume expander (NS or RL), TKO or as directed.
- G. Administer sodium nitrite 300 mg (10 ml or 3% solution) IV over no less than 5 minutes. Rate should not exceed 2.0 ml/min. Pediatric dose is 0.2 ml/kg, not to exceed 10 ml. Administer *very slowly* or as drip.
- H. Observe for seizures and treat with diazepam 5-10 mg IV slowly until seizure stops or 10 mg has been given.
- I. Observe for signs of acute pulmonary edema and treat as necessary.
- J. Transport as soon as possible -- may benefit from hyperbaric oxygen therapy.

Special notes

- A. Hydrogen sulfide is absorbed primarily through inhalation. Percutaneous absorption is minimal, although toxicity has been reported following application of sulfur-containing dermatologic preparations. Hydrogen sulfide is a highly toxic, odorous ("rotten egg" smell), and irritating gas. It is the cause of a number of fatalities, many multiple, due to inadequately protected rescuers.
- B. Hydrogen sulfide, like cyanide, binds to cytochrome oxidase and prevents aerobic metabolism at the cellular level. The administration of sodium nitrite induces methemoglobinemia which acts as a competitor with cytochrome oxidase to draw the sulfide off the enzyme to form sulfmethemoglobin. This is a relatively benign compound that is auto degraded to nontoxic forms of sulfur, which are excreted by the kidneys.

FLUORIDESource

- A. Hydrofluoric acid
 - 1. Glass etching
 - 2. Petroleum refining
 - 3. Dental work
 - 4. Rust removal
 - 5. Fertilizers
 - 6. Manufacturing
 - a. Fire extinguishers
 - b. Dyes
 - c. Tanning agents
 - d. Refrigerants
 - e. Plastics

- B. Other fluoride compounds
 - 1. Sodium fluoride
 - 2. Cryolite
 - 3. Toothpaste, mouthwashes
 - 4. Insecticides and rodenticides
 - 5. Dietary supplements

Clinical presentation

Skin -- Concentrated hydrofluoric acid causes lesions which are immediately, intensely painful. Dilute acid can delay treatment with prolonged absorption.

Lungs -- Concentrated vapors are intensely irritating to lungs and conjunctivae. May lead to respiratory tract damage and pulmonary edema.

GI -- Direct corrosive effect -- nausea, vomiting and abdominal pain.

Other:

- A. Fluoride ion chelates calcium -- lowers serum calcium may result in paresthesias, tetany, convulsions and cardiac dysrhythmias.
- B. Fluoride impairs the formation of collagen tissue and has direct action on muscle and nerve tissue. May result in a variety of musculoskeletal and neurologic complaints, including headache, paresthesias, visual disturbances, and mental deterioration.
- C. Fluoride interferes with many enzyme systems -- glycolytic enzymes, cholinesterases, and others.

Treatment

- A. Assure safety of rescuers.
- B. Decontaminate.
- C. Airway, protect as needed.
- D. O₂, high flow (10 - 15 L/min). Titrate to pulse oximetry > 90% if possible.
- E. IV -- volume expander (NS or RL), wide open to 20 ml/kg, unless contraindicated by pulmonary edema.
- F. Cardiac monitor.
- G. Apply calcium gluconate gel to any skin areas which are symptomatic.
- H. For patients with significant exposure and systemic signs of hypocalcemia -- administer calcium gluconate, 10% solution 10-30 ml slowly IV. Pediatric dose 0.2-0.3 ml/kg.
- I. Calcium Chloride may be used for patients with significant systemic signs of hypocalcemia -- administer 5-10 ml slowly IV. Pediatric Dose 0.1-0.2 ml slowly IV>
- J. Consider administration of magnesium sulfate 1-2 Gm IV.
- K. Transport as soon as possible.

Special notes

- A. Hydrofluoric acid is one of the strongest acids known. It is used extensively in chemical and industrial plants for a variety of applications. On direct contact hydrofluoric acid causes liquefaction necrosis by action of the hydrogen ion that is identical to other acid burns, disrupting the outer layer of skin and immediately proceeding to destroy the subcutaneous tissues. The fluoride ion penetrates into the subcutaneous tissues and complexes with calcium and magnesium to form insoluble fluoride salts. This process continues and can result in hypocalcemia or hypomagnesemia.
- B. The fluoride ion also acts as an enzyme inhibitor which inhibits cellular metabolism. Severe hydrofluoric acid burns can be associated with systemic fluoride toxicity.

HYDROCARBONS

Source

- A. Aliphatic Chemicals
 - 1. Methane
 - 2. Ethane
 - 3. Propane
 - 4. Butane
 - 5. Hexane
 - 6. Cyclohexane

- B. Aromatic hydrocarbons
 - 1. Benzene
 - 2. Toluene
 - 3. Xylene
 - 4. Aniline
 - 5. Phenol

- C. Halogenated Hydrocarbons
 - 1. Methyl chloride
 - 2. Methylene chloride
 - 3. Chloroform
 - 4. Carbon tetrachloride
 - 5. Ethyl chloride
 - 6. Trichloroethane
 - 7. Trichloroethylene
 - 8. Tetrachloroethylene

- D. Mixtures
 - 1. Gasoline
 - 2. Mineral spirits
 - 3. Kerosene
 - 4. Turpentine
 - 5. Pine oil
 - 6. Pine tar

Clinical Presentation

- A. Respiratory
 - 1. Tachypnea
 - 2. Cough with sputum production
 - 3. Crackles and wheezes
 - 4. Hypoxia

- B. Cardiac
 - 1. Tachycardia
 - 2. Ischemic changes
 - 3. Ventricular dysrhythmias

- C. Nervous
 - 1. Headache, dizziness, weakness, confusion
 - 2. Agitation, seizures
 - 3. General anesthesia and narcosis
 - 4. Coma

- D. Other
 - 1. Chemical burns & dermatitis
 - 2. Lacrimation, blurred vision, corneal and conjunctival irritation
 - 3. Nausea, vomiting, diarrhea
 - 4. Kidney failure

Treatment

- A. Assure safety of rescuers
- B. Decontaminate (copious water & mild soap for Skin)
- C. Airway, protect as needed
- D. O₂, high flow 10-15 L/M). Pulse oximetry.
- E. Labetalol 20 mg slow IV for significant, persistent, ventricular dysrhythmias and tachycardias.

Special Notes

- A. Exposure to hydrocarbon gases and vapors can cause simple asphyxia. If hypoxia is not corrected, the decrease in oxygen will initially cause CNS stimulation followed by CNS depression.
- B. Hydrocarbon vapors cause irritation and drying of mucous membranes in the respiratory tract. Prolonged exposure can lead to a chemical pneumonitis.
- C. Hydrocarbons are classified as volatile organic compounds which are quite flammable and frequently have carcinogenic effects. Decontamination before transport is essential. Responders should consider the use of air supplied breathing apparatus and chemical protection clothing (Level B) when caring for patients contaminated with hydrocarbons.