- Past medical history: Previous cardiac DZ (AMI, ASHD, HBP); respiratory DZ (COPD); diabetes, anemia
- Medications/allergies

2. Physical examination

   a. ABC's

   b. Rales vs. rhonchi vs. wheezing

   c. Soot vs. burns

   d. Other injuries

   e. EKG's

F. Pre-Hospital Treatment

1. Entubation - Endotracheal vs. EOA

2. Humidified O_2 Via Mask - High flow

3. I.V.

   a. D_5W KVO

   b. L/R or N/S, 4mg/Kg/% BSA burned

4. Arrythmia therapy

5. Supportive care - Basic Life Skills!

6. Other medications

   a. Bronchodilators

   b. Diuretics

   c. Analgesics

G. Emergency Department Assessment

1. Physical examination

2. Laboratory examination

3. X-ray evaluation

4. Bronchoscopy

H. Emergency Department Therapy

1. IPPB vs. PEEP

2. Cardiac medications

3. Pulmonary medications

I. Post Inhalation Sequalae

II. Toxic Gases

A. Recognition and Response

1. Size up - consider the hazard

2. Evaluation

3. Personel safety

4. Consider the container

B. Points to Consider

1. Injury severity depends on

a. Solubility of gas in H_2O

b. Duration of exposure

c. Concentration of gas

d. Presence of underlying pulmonary disease

2. Types of gases and their effect

a. Hypoxia - Butane, CO_2, Methane, natural gas

b. Poisons - Carbon Disulfide, Carbon Monoxide, Methyl Bromide, Methyl Iodine, Hydrogen Cyanide

c. Pulmonary irritants - Chlorine, tear gas, Nitrous Fumes, Sulfur Dioxide

d. Systemic effect and pulmonary irritant - Acetylene, Hydrogen Sulfide, Nitrogen, Tetroxide, O zone

e. Ammonia

- Soluble, colorless gas, alkalis
- Extremely irritating to eyes and mucous membranes
- Used with refrigeration equipment
- Inhalation of concentrated ammonia produces asyhyxiation in minutes, lesser amounts produce pulmonary edema, chemical bronchitis

f. Methyls

- Used as refrigerants, and in organic synthesis and fumigants
- Methyl bromide used as herbicide - penetrates rubber gloves
- Methyl chloride used as bubbling fluid in ornaments
- Tasteless and odorless
- CNS depressants - similar to chloroform - can also cause liver, renal and myocardiac damage
- Symptoms include: fatigue, H/A, dizziness, ataxia, weakness
- Early deaths 2^{0} to pulmonary effects - similar to chlorine
- Late deaths 2^{0} to renal failure

g. Chlorine

- Nonflammable compressed gas
- DE agent for bleaching cloth and paper, H_2O purification or chemical by product - mixing household cleaners (chlorox) with acids (vinegar); mixing bleaches with household ammonia
- Heavier than air
- Rapid acting
- Becomes corrosive when mixed with H_2O
- Death 2^{0} to sloughing of bronchial epithelium and obstruction of the airway
- High concentration results in chemical burns of skin and mucous membrane

4. Pre-hospital assessment

a. History

b. Physical examination

- Lacrimation, conjunctivitis, burning in nose, throat (15 ppm)
- Coughing, choking, burning C/P, N/V, wheezing (30 ppm)
- Pneumonitis, pulmonary edema (40-60 ppm)

3. Major gases

a. Hydrogen Sulfide

- Characteristic odor of rotton eggs
- Found in sewers, product of putrefaction, shale oil industry, manufacture of fertilizers
- Highly toxic - death 2^0 to respiratory failure
- Effect - low concentration - eye irritation, cough, SOB. pulmonary edema
  - high concentration - initial CNS stimulation CNS depression, H/A, N/V, emotionally labile
- Causes cellular anoxia - effect similar to cyanide - treatment same as cyanide

b. Carbon disulfide

- Highly volitile liquid: Smells like chloroform, hazardous vapor
- Usually inhaled, can be inhaled or absorbed
- Used as solvent for oils, fats, rubber, wax; disinfectant, fumigant, and in manufacture of rayon and artificial silk
- Rapidly absorbed when inhaled
- Principle effect is CNS stimulation - H/A, N/V, fatigue, excitement, euphoria, hallucination
- Death 2^0 to respiratory failure

c. Anhydrous Ammonia (NH_3)

- Colorless, alkaline gas - nonflammable compressed gas
- Sharp pungent odor
- Two storage vessels - upon release from pressure vessel, liquid ammonia vaporizes and expands rapidly - absorbs heat with expansion
- Irritates skin and mucous membranes

d. Ammonium Nitrate

- Used in dynamite and blasting agents
- Colorless - white powder - decomposes into extremely toxic gas
- Fertilizer grade - decomposes into introus oxide and H_2O - this reaction liberate heat - increases decomposition → explosion
- TX's - tremor, slurred speach, atoxia, stupor, coma - clinical picture of liver failure

5. Pre-hospital treatment

   a. Decontamination - consider your safety

   b. Humidification of O_2

   c. I.V. therapy - D_5W KVO

   d. Bronchodilation

      - o Epinephrine (1:1000) - .3 ml SQ (adult); .01 ml/Kg SQ
      - o Aminophylline - 6 mg/Kg m 150cc D_5W over 20 mm
      - o Bronkosol - .5 ml in 2 ml saline

   e. Irrigate eyes with N.S.

   f. Irrigate skin burns with N.S.

   g. Pulmonary edema - as usual

   h. Supportive care

6. Pathophysiology of chlorine gas

7 Emergency Department assessment

   a. Physical examination

   b. Laboratory evaluation

   c. X-ray

8. Emergency Department therapy

   a. Minor injury

   b. Severe injury

9. Post injury sequalae

III. Organophosphates/Pesticides

A. Recognition and Response

1. Dangers 2 to chemicals themselves, smoke from burning, contamination from water runoff

2. Small amounts cause sickness

3. Easily absorbed, can be inhaled or ingested

4. Fastest way to enter systemic system - eyes!

5. Toxicity determined by

   a. Kind of pesticide

   b. Concentration

   c. Amount of exposure

   d. Type of exposure

6. Approach upwind

7. Avoid runoff

8. Use full turn out gear and SCBA

9. Maximum ventilation

B. Pre-Hospital Assessment

1. History

2. Physical examination - symptoms determined by type of chemical
   Common symptoms - N/V, tremor, H/A, dizzy, miosis, bradycardia
   - Salivation, lacrimation, urination, defecation (slud)

C. Pre-Hospital Treatment

1. Decontamination

   a. Wear gloves and gear

   b. Clean/dry - brush it off gently

   c. "Strip"

   d. Soap and water

   e. Bag it

2. Humidified O_2

3. Atropine

   a. 2-4 mgm IVP QI5 min. (adults); .05 mg/Kg (children) QI5 min.

   b. Continue until signs of atropinization (mydriasis, dry mouth, tachycardia)

   c. Correct the cyanosis first!

4. Valium 2.5-15 mg IVP - seizure therapy only

D. Pathophysiology

E. Emergency Department Evaluation

1. Physical examination

2. Laboratory

3. Other

F. Emergency Department Management

1. Continue atropine

2. Pralidoxine (Pam) - 1 gm (adults); 10-12 mg/Kg (children)

3. Supportive care

G. Post Incident Sequalae

IV. Tear Gas

A. Chloroacetophrone (CN) vs. Thochlorbenzalmalononitrine (CS)

B. Symptoms

1. Lacrimation, conjunctival burning

2. Chest tightness

3. Panic, anxious

4. Mucous membrane irritation

C. Pre-Hospital Assessment

1. Gas masks! Gloves!!

2. Get into fresh air

3. Physical examination as usual

D. Hospital Treatment

1. CN gas - eyes - irrigate with NS, or .4 gms sodium sulphite in 25cc of H_2O and 75cc glycerin
- skin - wash with LC amount of hot soapy H_2O; 4 grams sodium sulphite in 50cc of H_2O and 50cc of grain alcohol

2. CS gas - eyes - irrigate with NS, or 1% sodium bicarb solution
   - skin - bathe or shower 6 hours after exposure; use mild lanolin soap; use ethylenegycal or 5% solution sodium bisulfite; use steroid or antihistamine ointment if needed
   - observe for pulmonary edema

CASE PRESENTATIONS

I. At 2:30 a.m., you are called to respond to a reported apartment fire. Upon your arrival, you find a 4-story garden apartment; heavy black/grey and fire smoke is pouring from a ground level apartment. The smoke is raising to the upper floor levels where you find several people "trapped" in their balconies.

A. William Schmidt is a 37-year-old fireman who was part of the team which made the "knock down." He was wearing his SCOTT PAK initially but during overall had removed it to avoid increased fatigue. He is conscious but complains of nausea, dizziness, a severe bounding headache in both temples and blurred vision. His vital signs include BP 128/90-94-18; his s in is warm, dry, with some soot in his clothing.

B. Mollie Jones is a 24-year-old white female whose apartment was on fire. She had been found staggering in the hallway outside her apartment by fireman. She complains to you of SOB and a feeling of extreme tightness in her chest. Her face, head, neck, and night clothing are covered with soot and while you talk to her, her voice is raspy and she frequently has paroxysms of coughing which produces soot stained mucous. Her vital signs are BP 160/100-P-110 irreg.-resp. 40 and labored.

II. A. Jimmy Hall, a 23-year-old yard worker, is one of several employees working when a valve breaks in a bank of chlorine storage tanks, releasing 8,000 lbs of liquid chlorine. When you see him, he is complaining of severe burning of his eyes and nose, is tearing excessively, demonstrates a mild non-productive cough, and also states his throat is sore. His vital signs are BP 124/86-90-18; skin temperature and color are normal. Physical examination reveals no other apparent injuries; lungs are clear bilaterally.

B. Walter Hanger is a 48-year-old yard worker who was also working when the chlorine leakage occurred. He is in obvious respiratory distress and complaining of chest pain. His vital signs are B/P 140/98-102 irreg.-28; s in color is pale and slightly diaphoretic You also notice a frequent dry, hac ing cough. Physical examination reveals no apparent injuries, but auscultation of both lungs reveals scattered bilateral wheezing. His EKG shows sinus tachycardia with occasional multi focal PVC's.

III. Following the extinguishment of fire in the lawn and garden section of Hechinger's, personel begin an extensive overhaul. Ventilation with exhaust fans is continuing. Because of the high heat and humidity, several personel have removed their turnout gear and SCBA; several firemen, working in the area of origin, begin to feel nauseated, lightheaded and are staggering around. Your examination reveals that each one is tearing excessively and continuing to spit clear mucous.

They are all diaphoretic and in mild respiratory distress. Vital signs are stable. No apparent injury can be found. However, you do note that several of the individuals have light-colored dust mixed with the sweat on their forearms and around their eyes. Their pupils are miotic.

IV. A civil disturbance between a large crowd of hecklers and police breaks out. In order to quiet the riot, police are forced into using tear gas.

A. A 37-year-old male staggers toward you from a cloud of white malodorous and irritating smoke. He is rubbing his eyes, tearing profusely and has white powder all over his head, neck, and clothing. He is complaining of severe burning of his eyes and skin and subsequently begins to develop a choking sensation in his throat. He is panic stricken; his vital signs are unobtainable because of his agitation.

Mark Smith M.D.
Craig Deatley P.A.

Toxic Inhalations

1. Smoke and gas inhalation wreaks havoc on the body in four ways:

   a. decreased inspired oxygen content in the inhaled gas leads to asphyxia.

   b. high temperature of the inspired gas leads to a thermal burn of the respiratory tree (mostly to the upper airway)

   c. presence of systemic cellular toxins leads to a chemical asphyxia.
   egs. carbon monoxide
   cyanide

   d. presence of pulmonary irritants leads to a chemical burn of the respiratory tree.
   egs. chlorine
   aldehydes and acids adherent to carbon soot
   hydrochloric acid from smoldering polyvinyl chloride

   Any particular toxic inhalation may show one, some or all of these processes. Chlorine gas fumes liberated during a storage tank spill have only a pulmonary irritant toxicity with no significant asphyxia, systemic toxicity or thermal burn.
   Smoke elaborated during an apartment house fire does most of its damage through carbon monoxide (a systemic cellular poison), hydrochloric acid (a pulmonary irritant elaborated from the heating of polyvinyl chloride), and organic acids and aldehydes adherent to carbon soot particles (another pulmonary irritant).

2. Asphyxia needs no further elaboration. Where there is no oxygen to breathe there is no life. Oxygen may be used up in combustion or displaced by another gas.
   The mechanism of death by asphyxia is probably through secondary circulatory arrest that speeds the brain death.

3. The respiratory tree has only a limited number of sites that can be damaged by thermal or chemical irritants and only a limited number of ways of manifesting damage.
   As a first approximation, the respiratory system can be divided into four parts: upper airway
   trachea and mainstem bronchi
   smaller bronchi and bronchioles
   alveoli

3. The upper airway responds with swelling that results in hoarseness, sensation of choking and constriction, stridor, and eventually obstruction.

   The trachea and major bronchi respond with swelling and outpouring of secretions that result in coughing and chest pain. Subglottinc problems, i.e. a severe tracheitis, can masquerade as upper airway obstruction.

   The smaller airways and bronchioles respond with swelling, spasm and outpouring of secretions and mucous plugging that result in wheezing.

   The alveoli respond by developing a leaky membrane with subsequent alveolar edema that results in tachypnea, dyspnea and cyanosis.

   In general, upper airway problems occur earlier in the course of a toxic gas exposure than does alveolar edema, which may be delayed from 8 to 48 hours post exposure.

4. Thermal damage in general is localized to the upper airway. Heat is dissipated quite rapidly as gas passes down through the nose,mouth, larynx, pharynx and trachea. Gas at 500° C. at the vocal cords becomes 50° C. at the tracheal bifurcation.

   Wet heat penetrates further down the respiratory tree than does dry heat; so always carefully evaluate a superheated steam exposure for laryngeal and tracheal damage.

Mark Smith M.D.
Craig DeAtley P.A.

Smoke Inhalation

1. Smoke is a suspension of fine carbon particles in gas. In most fires, the gas is primarily carbon dioxide and carbon monoxide, but may include pulmonary irritants like hydrogen chloride.
   Carbon particles of size .5 to 2 microns can make their may down to the alveoli; larger ones get trapped in the upper airway. Carbon particles per se are not lung toxins (if a patient who has been treated with a slurry of activated charcoal for an ingested poison accidentally aspirates the charcoal, no harm occurs). What is toxic are the organic acids (acetic acid) and aldehydes that are adherent to the carbon particles and for which the carbon soot serves as a vehicle. Wood and cotton smoke contain many times more toxic aldehydes than does kerosene smoke (and higher concentrations of carbon monoxide) and experimentally produces more pulmonary pathology.
   The products of combustion of plastics, in particular the HCl elaborated during the heating of polyvinylchloride, are the other major pulmonary irritant elaborated in smoke during a fire (see handout on chlorine gas)

2. The diagnosis of a significant smoke inhalation is made from:

   history- exposure to smoke in a closed space
   history of unconsciousness not attributable to other causes

   physical- facial burns
   oral mucosal burns
   singed nasal hairs
   hoarseness
   cough productive of carbonaceous sputum
   wheezes, rhonchi or rales
   respiratory distress

   lab- abnormal ABG (low pO_2, low pCO_2)
   elevated COHb (a carbonmonoxyhemoglobin level of over 15% correlates with a degree of smoke inhalation sufficient to cause pulmonary damage)
   chest xray (usually normal initially; abnormalities in the ER are probably from atelectasis rather than pulmonary edema which takes at least 4 to 8 hours to develop)

3. Everyone with a diagnosis of significant smoke inhalation should be admitted to the hospital for treatment and or observation. A reliable patient who has only minimal symptoms such as a mild cough, no physical exam evidence of smoke inhalation, and a normal laborat ry profile can be watched in the emergency department for several hours and allowed to go home with careful instructions.
The development of upper airway problems can be abrupt.
The development of pulmonary edema and bronchiolar inflammation be delayed 24-48 hours.
Better safe than sorry.

4. The initial treatment of any smoke inhalation is humidified oxygen, preferably 100% (on a rebreather mask) until a COHb level comes back from the laboratory.

   The treatment for upper airway obstruction is intubation.
   Make sure that impending upper airway obstruction is not missed.
   It is much more difficult to intubate a closed off larynx than one through which air is still being moved.
   It may be difficult to tell whether respiratory distress is caused by laryngeal swelling or by a severe tracheitis; in those cases laryngoscopy and or bronchoscopy with an endotracheal tube poised and threaded over the bronchoscope may help pinpoint the lesion.
   Other indications for intubation are:
   a. inability to handle the quantity of respiratory secretions.
   b. need for ventilator assistance.
   c. severe facial burns with progressive edema.

   The treatment of bronchospasm and wheezing is as for acute asthm
   a. sympathomimetics (epinephrine and micronebulized isoetharin or isoproterenol)
   b. aminophylline
   c. steroids (probably beneficial)

   Bronchoscopy may help unplug atelectatic segments that have collapsed because of mucous plugs.

   The treatment for pulmonary edema secondary to smoke inhalation is as for any ARDS syndrome:
   a. restricted fluids
   b. positive pressure ventilation with PEEP or CPAP

5. Chest x rays are usually normal in the emergency department. They turn abnormal with the onset of pulmonary edema (8-48 hours post inhalation), atelectasis (from mucous plugging) or pneumonia (usually not until third post exposure day). Smoke inhalation patients are especially prone to staphylococcus and pseudomonas pneumonia; both are extremely necrotizing forms.

References

1. Schwartz et al: Principles and Practice of Emergency Medicine. W. B. Saunders, 1978, pp. 837-844.

2. Shibel E. and Moser, J.: Respiratory Emergencies. C. V. Mosby, St. Louis, 1977, ppl78-181.

3. Moylan, J. and Chan, C.: Annals of Sugery, vol. 188: 34-37, 1978.

4. Welch, G. et al: Surgery, Gynecolcgy and Obstetrics, 145: 539-544, 1977.

Mark Smith M.D.
Craig DeAtley P.A.

Irritant Gases

Chlorine

1. The most common toxic gases that act as pulmonary irritants are chlorine (Cl_2), phosgene ($COCl_2$), ammonia (NH_3), sulfur dioxide (SO_2) and nitrogen dioxide (NO_2). In addition to their intrinsic toxicity as gases, as they enter the tracheobronchial tree, they combine with water and get converted to caustics. Chlorine and phosgene become hydrochloric and hypochlorous acid, ammonia becomes ammonium hydroxide, sulfur dioxide becomes sulfuric acid and nitrogen dioxide becomes nitrous and nitric acid.

   Use of a water soaked towel over the eyes and mouth and nose by rescue personnel is logical in that it permits conversion of the gases to their caustic counterparts outside of the body's mucous membranes and endothelium.

2. The site of damage wreaked by these gases in the respiratory tree is a function of the solubility of the gas. The more soluble the gas (eg. ammonia),the more that the mucous membranes of the mouth, conjunctiva, nose and the upper airway are irritated. The less soluble the gas, the more that the lower airway and lungs are affected (eg. phosgene). Certain gases of intermediate solubility (eg. chlorine) affect all parts of the respiratory tree.

   The soluble gases that affect the upper airway and mucous membranes give warning of their presence by the intense eye, nose and throat burning they produce. The insoluble gases like phosgene cause significant pulmonary without warning symptoms of their presence.

3. The extent of respiratory tract damage is a function of the duration of exposure, the concentration of the gas, and the water content of the exposed tissue.

4. Chlorine is a yellow green gas that is denser than air. It is used in water purification, especially in swimming pools, as a reagent in chemical plants, and for industrial bleaching. The mixture of sodium hypochlorite (common household bleach) with an acid cleaner can elaborate chlorine gas fumes in the home.

5. Damage to the respiratory tree is directly caused by the chlorine gas and by the formation of hydrochloric and hypochlorous acid. The organ systems affected besides pulmonary are gastrointestinal, skin and eye.

   There is a fairly well worked out relationship between the concentration of the gas and the site and extent of the damage.

1 ppm -	gas is undetectable
4 ppm -	presence of gas is detected and may be tolerated for up to 1 hour with no ill effects
15 ppm-	eye, nose and throat irritation manifested by lacrimation, conjunctivitis and burning
30 ppm-	immediate coughing, choking, burning chest pain, nausea, vomiting, wheezing
40- 60 ppm-	lung injury (pulmonary edema)

   The minimum lethal concentration is approximately 400 ppm over 30 minutes. The pathologic changes in the lungs in severe exposure show exudative bronchitis with sloughing of bronchial epithelium and microatelectasis.

   The initiial responseto exposure is coughing and a choking sensation which may subside when the patient is removed from the scene. A quiescent period may ensue.
   The coughing and choking can then recur later as the inflammat progresses (6-24 hours) and be joined by stridor, wheezing, dyspnea, and possibly severe respiratory distress.
   Upper respiratory problems usually present before lower airway, bronchiolar and alveolar problems.
   The more severe the exposure, the more rapid the progression.

6. The emergency department assessment of chlorine gas exposure is essentially the assessment of the patient's pulmonary system.
   Symptoms to check for are cough, dyspnea, sore throat, chest pain.
   Signs to check are respiratory rate, use of intercostal muscles, stridor, hoarseness, wheezes, rales, rhonchi.
   Laboratory evaluation includes an arterial blood gas and a chest x ray. Arterial oxygenation should be normal unless a significant exposure has taken place. Any chest x ray abnormalities in the emergency department indicate very severe exposure (pulmonary edema or atelectasis secondary to mucous plugging).

7. The emergency department treatment for upper airway inflammation is inhalation of cold steam with 40% O_2 and early intubation if the airway is threatening to close.

   The treatment for bronchitis and bronchiolitis manifested by wheezing is cold steam, epinephrine subcutaneously, micronebulized isoetharine or isoproterenol, and aminophylline.

   The treatment for pulmonary edema are the usual modalities for ARDS: O_2, PEEP, fluid restriction.

   Bronchoscopy may be helpful in assessing the site of damage and in removing mucous plugs.

   The use of steroids in chlorine induced pulmonary edema or bronchospasm is not well studied. One report in the literature suggests fewer residual pulmonary problems with the use of steroids.

   Any patient presenting with upper airway symptoms or with more than minimal wheezing should be admitted to the hospital and observed for 24 to 48 hours to watch for upper airway obstruction or pulmonary edema.

8. For mild chlorine gas exposure, the patient may be treated symptomatically.

For ocular irritation-	saline irrigation fluorescein evaluation for corneal abrasion if abrasion, patch with antibiotic ointment if no abrasion, ophthalmic wetting agent like Tear-a-Sol or Liquifilm Tears
For sore throat-	cold steamy showers lozenges
For cough-	antitussive

9. There is controversy in the literature whether there is residual pulmonary damage after chlorine gas exposure. There is good evidence that in severe exposure, the patient has an initial hypoxemia that then corrects to normal over several months only to be replaced by abnormally increased airway resistance (a function of the pulmonary scarring that has occured).

10. Hydrogen chloride is a gas that is elaborated as a result of the burning of plastics, especially polyvinyl chloride. These plastics are widely used and their fumes are present in many house fires.

    The effects of hydrogen chloride inhalation are similar to those of chlorine gas:

    constricting tightness to anterior chest
    dizziness
    burning sensation of the throat
    lacrimation
    conjunctivitis
    nausea

    The chest constriction and throat burning are especially prominent.
    The onset of symptoms may be delayed more than with chlorine gas exposure.
    There is a definite myocardial toxicity manifested by ST segment changes.

11. Phosgene is a colorless gas that smells like musty hay. It is used in aniline dye production and converted to HCl and HOCl.
    It is not soluble and thus not irritating to the eyes, mucous membranes, or upper airway. High concentrations can be inhaled before the patient is aware.
    Toxicity is to the lower half of the pulmonary tree.

    Sulfur dioxide is an irritating colorless gas with a pungent odor that is converted to sulfuric acid.
    It is a common atmospheric pollutant that is a byproduct of smelting and paper manufacturing. It causes so much upper respiratory irritation that toxic lung exposures are rare.

    Ammonia is a soluble colorless gas that is very irritating to the eyes and nose.
    It is more likely to give an upper airway inflammation with laryngospasm than chemical bronchitis or pulmonary edema

    Nitrogen dioxide is an insoluble reddish brown gas with a pungent odor.
    It gets converted to nitric and nitrous acid.
    There is more bronchial and alveolar disease than trachea and upper airway damage.

References

1. Shibel, E. and Moser, K,: Respiratory Emergencies, C. V. Mosby, 1977, pp 174-178.

2. Chester, E. et al, Pulmonary injury following exposure to chlorine gas. Chest 72:247-250, 1977.

3. Hedges, J. et al: Acute chlorine gas exposure. JACEP 8: 59-63, 1979.

4. Done, A. The toxic emergency: it's a gas. Emergency Medicine May 1976: 305-314.

5. Tintinalli, J.: A Study Guide in Emergency Medicine. ACEP, 1980, pp. 3-67 - 3-69.

6. Dyer, R. and Esch, V. : Polyvinyl Chloride toxicity in Fires. JAMA 235: 393-397, January, 1976

Mark Smith M.D.
Craig DeAtley P.A.
January 1981

Carbon Monoxide

1. Carbon monoxide is a colorless and odorless gas that is completely nonirritating to the respiratory tree. It exerts its toxic effects by a chemical asphyxia; it preferentially binds to the sites where oxygen ordinarily binds: to the hemoglobin or redblood cells, to the myoglobin in muscle cells, and to the cytochrome oxidase system in all mitochondria.
   The binding to hemoglobin is toxicologically the most important.
   Where there is carbon monoxide, oxygen is not.

2. The affinity of carbon monoxide for hemoglobin is approximately 250 times that of oxygen. Thus the inhalation of .01%(100 ppm) carbon monoxide from the atmosphere can lead to carbonmonoxyhemoglobin levels of 14%; inhalation of .1% carbon monoxide gives COHb levels of 60%. The normal amount of carbonmonoxy-hemoglobin in the bloodstream is less than 1% of the total hemoglobin.

3. Carbon monoxide exerts a second toxicity on hemoglobin: it shifts the oxyhemoglobin dissociation curve to the left. The oxygen that is carried by what free hemoglobin remains is bound with a greater than normal tenacity. Tissue oxygen delivery suffers. A 50% reduction in hematocrit from normal and a 50% COHb level provide the same amount of free hemoglobin for oxygen transport; the tissues see significantly less oxygen in the case of the carboxyhemo-globin because of the left shift to the dissociation curve.

4. The toxicity of carbon monoxide is manifested primarily by the organs most exquisitely sensitive to lack of oxygen: the central nervous system and the heart.
   A heart ravaged by atherosclerotic vascular disease can develop arrhythmias or acute ischemic events secondary to carbon monoxide poisoning.
   There is a well worked out relationship between the level of carboxyhemoglobin in the blood and the degree and type of clinical symptoms.
   A normal COHb level is less than 1%.
   With COHb levels of less than 10%, patients are generally asymptomatic.
   From 10-20% there may be reports of bandlike headaches and shortness of breath.

4. With 20-30% COHb there is throbbing of the temples, irritability, giddiness.
   With 30-40% COHb, there is headache, dizziness, visual symptoms, nausea, weakness, potential cardiac irritability, tachycardia.
   With 40-50% COHb, there is syncope, confusion.
   With 50-60% COHb, there is unconsciousness, seizures, myoclonic jerking.
   With 80% COHb, there is death.

   The relationship between clinical symptoms and COHb levels is only approximate.
   Underlying anemia, heart disease, or lung disease may exacerbate carbon monoxide toxicity.
   Decreased ambient oxygen and the presence of pulmonary irritants in the inhaled gas may complicate the carbon monoxide exposure.
   Significant time may elapse between the initial patient assessment and the measurement of carbon monoxide levels. A patient unconscious on arrival at the emergency room with a COHb level of 25% might have been treated with 100% oxygen en route to the hospital by rescue personnel.

5. Emergency Department Assessment
   a. History
      A history of exposure to smoke in an enclosed space should be considered to be a potential carbon monoxide intoxication. Carbon monoxide results from the incomplete combustion of fuels; the complete combustion yields carbon dioxide. Enclosed spaces have only a limited supply of oxygen for combustion.

      A history of unconsciousness at the scene of the incident should be considered prima facia evidence for a carbon monoxide exposure.

      The presenting symptoms will vary with the degree of exposure. The relationships described above between COHb levels and patient complaints is a rough guide.

   b. Physical Examination

      Any patient who meets the criteria for a significant smoke inhalation (see Smoke Inhalation handout- oral and facial burns, cough productive of carbonaceous sputum, singed nasal hairs, etc.) should be considered to have suffered a carbon monoxide exposure until proven otherwise.

5. b. Tachycardia and tachypnea may or may not be present.
Much of the tachypnea seen with carbon monoxide inhalation may be a function of the exposure to the concomitant pulmonary irritant gases and smoke particles.
Since pO_2 may be normal in a carbon monoxide exposure the repiratory rate may not be increased
Tachycardia is more reliably present.

The classical cherry red glow to the patient's mucous membranes that is supposed to be seen with COHb levels of over 35% has not proved to be a useful clinical sign.

c. Laboratory Evaluation

Obtain an arterial blood gas. The pO_2 may be normal or decreased. The pCO_2 may be normal or may show a primary respiratory alkalosis.

It is important to realize that carbon monoxide should not significantly decrease the pO_2 of the blood. The pO_2 is a reflection of how much oxygen is dissolved in blood and not how much is carried by hemoglobin. Carbon monoxide simply lowers the number of available binding sites for oxygen.

Do not be fooled by a report of a normal arterial oxygen saturation on your blood gas result in the presence of a significant COHb level. The laboratory measure pO_2. This may be normal even with high COHb levels. The laboratory then calculates from a nomogram (and does not directly measure) the hemoglobin saturation. That nomogram assumes a zero level of carboxyhemoglobin. Were the laboratory to directly measure O_2 content and hence O_2 saturation, there would be no error.

There are two ways to measure carbonmonoxyhemoglobin; either via a blood sample or through an analysis of expired air. The expired air method uses a conversion table to translate CO concentration in the air to a COHb level in the blood. The blood method is more accurate; the expired air method faster.

6. Any patient with an initial COHb level of greater than 20% should probably be admitted for 24 hours of observation even if only minimally symptomatic when seen. That degree of carbonmonoxyhemoglobin is usually an index to a significant exposure to other smoke toxins.

7. The treatment of carbon monoxide poisoning is simple: give oxygen.

   There should be two goals in treating carbon monoxide poisoning: eliminate the carbon monoxide and correct the cerebral hypoxia. High concentration oxygen does both.

   The half life of carbon monoxide while breathing room air is 4-5 hours. (i.e. the half life is the time it takes for COHb to go from its current value to one half that value) While breathing 100% oxygen, the carbon monoxide exposed patient can reduce that half life to 80 minutes (some sources say 45 minutes).
   Breathing hyperbaric oxygen at 2 - 2.5 atmospheres reduces the half life still further to 25 minutes.

   High concentrations of oxygen help reverse the cerebral hypoxia. Up to one third of the normal O_2 demand can be satisfied by the oxygen that is dissolved in blood (as opposed to carried by hemoglobin) if the patient is breathing 100% O_2.

   Patients should be treated with 100% O_2 for four hours or until their COHb is less than 20% and then left on a 40% or 50% mask for the next 24 hours.

   Treatment with 100% O_2 should be initiated on the scene of a fire by EMT's and paramedics.

8. Carbon monoxide poisoning carries with is a significant neuropsychiatric morbidity. Complications have included basal ganglia and cerebellar problems (movement disorders), personality changes, and cognitive function abnormalities.

References

1. Myers, R. et al: Carbon monoxide poisoning: the injury and its treatment. JACEP 8: 479-482, 1979.

2. Stewart, R.: The effect of carbon monoxide on humans. Journal of Occupational Medicine 18:304-309, 1976.

3. Whorton, M.: Carbon monoxide intoxication: a review of 14 patients. JACEP 5: 505-509, 1976.

4. Winter, P. and Miller J.: Carbon monoxide poisoning. JAMA 236: 1502-1504, September 1976.

5. Stewart, R. et al: Rapid estimation of carboxyhemoglobin level in fire fighters. JAMA 235: 390-392, January 1976.

Mark Smith, M.D.
Craig DeAtley, P.A.

Organophosphate Poisoning

1. Before 1972 the most common pesticides in use were the chlorinated hydrocarbons, eg. DDT, dieldrin, BHC. These products remained intact in the enviornment, accumulated in animals, and exerted deleterious effects at many stages of the food chain.

2. Organophosphate insecticides replaced the chlorinated hydrocarbons as the pesticides of choice because within several days of application, the organophosphates decompose in the field to harmless compounds. However, long term toxicity problems were traded for acute poisonings. Acute organophosphate exposure has a significant morbidity and mortality. In 1972 there were almost 100 deaths from organophosphate poisoning.

   Parathion is one of the most popular industrial grade pesticides. It is highly toxic. Malathion, because of poor skin absorbtion and low oral toxicity, is the organophosphate most often used in the home.

3. Organophosphates are nerve poisons. They first achieved popularity not as insecticides but as nerve gases during world war II (tabun, soman, and sarin).

   They inhibit acetylcholinesterase and create a state of cholinergic overdrive in which there is too much acetyl choline at nerve endings.

   All of the toxic effects of organophosphates can be understood in terms of overstimulation and then subsequent inhibition at cholinergic synapses.

4. There are three divisions to the nervous system. Each has nerve synapses with acetylcholine as the neurotransmitter and so each is affected in organophosphate poisoning.

   a. central nervous system
   b. voluntary muscle
   c. autonomic nervous system

5. The autonomic nervous system is divided into two antagonistic subsystems: the sympathetic and parasympathetic. Their function is to control involuntary functions like peristalsis, heart rate, sweating, salivation, pupillary size, and pulmonary secretion.

5. The parasympathetic system constricts bronchial smooth muscle and increases bronchial secretions, increases GI peristalsis, slows the heart, contracts the bladder, increases salivation, and constricts the pupil.

   The sympathetic system relaxes bronchial smooth muscle, decreases gut activity, speeds the heart, and dilates the pupil.

6. Both the sympathetic and parasympathetic systems have two neuron relays. The synapse between neuron 1 and neuron 2 is called the ganglionic synapse and the synapse between neuron 2 and the end organ is called the post ganglionic synapse

   Of the four nerve synapses in the autonomic nervous system (sympathetic ganglionic, sympathetic post-ganglionic, parasympathetic ganglionic, and parasympathetic post-ganglionic), acetyl choline serves as the neurotransmitter for three. The only exception is the sympathetic synapse between neuron 2 and the end organ: there norepinephrine is the neurotransmitter (except for sympathetics to sweat glands where acetylcholine remains the neurotransmitter).

7. Ordinarily, the smooth functioning of the nervous system requires the elaboration of acetylcholine at the nerve or neuromuscular synapse and the orderly breakdown and inactivation of acetylcholine by the enzyme cholinesterase.

   Organophosphates do their damage by binding to cholinesterase. Acetylcholine is then not inactivated and is left to stimulate nerve synapses.

   The binding of organophosphates to cholinesterase is tight and can become permanent. In the absence of quick therapeutic interventions to release that binding, the patient may have to be supported for the several weeks that it takes for new cholinesterase to be synthesized by the body.

8. Too much acetylcholine at the effector organ synapse of the parasympathetic system (postganglionic) results in cholinergic overdrive. This is the muscarinic receptor because muscarine specifically stimulates it.

   The clincial picture is best characterized by SLUD
   - Salivation
   - Lacrimation
   - Urination
   - Diarrhea

8. Some authors augment SLUD to SLUDGE with the addition of
   Gastrointestinal cramps
   Emesis

   The other important muscarinic effect is small pupils, although pupils are usually the last organ to change and ten per cent of organophosphate poisonings present with mid position or large pupils (perhaps a function of the ganglionic stimulation of the sympathetic nervous system).

   Bradycardia may or may not be present.

9. Too much acetylcholine at the receptor of the myoneural synapse in skeltal muscle (nicotinic receptor) results first in cholinergic overdrive with twitching and fasiculations and then in depolarizing block with muscular weakness.

   twitching
   fasiculations
   weakness

10. Too much acetylcholine at the ganglionic synapses of the sympathetic and parasympathetic systems gives more nonspecific results. The parasympathetic system is already being maximally stimulated at the muscarinic site of the end organ so additional parasympathetic input probably does not do much. However stimulation of the sympathetic nervous system may result in tachycardia, hypertension, and pallor.

11. Too much acetylcholine in the CNS results in restlessness, anxiety, headache, slurred sppech, ataxia, confusion and eventually convulsions and coma.

12. Organophosphates are well absorbed by inhalation, by skin absorbtion and by ingestion. There are no irritant warning signs of exposure; organophosphates pass silently through the skin leaving only localized sweating as a calling card. A child has died from licking the nozzle of a parathion sprayer.
    Patients must be completely decontaminated, first in the field and then again in the emergency department. Emergency department personnel should wear gowns and gloves (thick rubber recommended).

    Speed of onset of symptoms as well as the predominant symptom site depend on the route of exposure. Inhalation of organophosphate fumes gives the fastest onset of symptoms, usually respiratory. Skin exposure may show localized sweating and fasiculations.

15. Treatment

Excess pulmonary secretions and bronchspasm should be treated with frequent suctioning and with atropine. Bronchodilators like theophylline and sympathomimetics should be avoided.

16. Laboratory

Organophosphate poisoning is a diagnosis made on clinical grounds of history and physical examination. There are several nonspecific laboratory clues: leukocytosis and hyperglycemia, both probably secondary to sympathetic stimulation via the ganglionic (nicotinic) receptors.

The most specific diagnostic laboratory test is the RBC cholinesteras level. There are two cholinesterases in the blody: true cholinesterase found at nerve endings and in red blood cells, and pseudocholinesterase found in serum and in the liver. Depression of RBC cholinesterase is the most specific way of making a diagnosis of organophosphate poisoning. Decreases to levels of less than 25% of normal represent significant intoxications. The pseudocholinesterase may be nonspecifically decreased in other conditions and thus is not as good a test.

Atropine will not elevate a depressed cholinesterase level; pralidoxime will. Thus an RBC cholinesterase level may be sent after atropine is administered but must be drawn before pralidoxime is given.

8. Some authors augment SLUD to SLUDGE with the addition of
   Gastrointestinal cramps
   Emesis

   The other important muscarinic effect is small pupils, although pupils are usually the last organ to change and ten per cent of organophosphate poisonings present with mid position or large pupils (perhaps a function of the ganglionic stimulation of the sympathetic nervous system).

   Bradycardia may or may not be present.

9. Too much acetylcholine at the receptor of the myoneural synapse in skeltal muscle (nicotinic receptor) results first in cholinergic overdrive with twitching and fasiculations and then in depolarizing block with muscular weakness.

   twitching
   fasiculations
   weakness

10. Too much acetylcholine at the ganglionic synapses of the sympathetic and parasympathetic systems gives more nonspecific results. The parasympathetic system is already being maximally stimulated at the muscarinic site of the end organ so additional parasympathetic input probably does not do much. However stimulation of the sympathetic nervous system may result in tachycardia, hypertension, and pallor.

11. Too much acetylcholine in the CNS results in restlessness, anxiety, headache, slurred sppech, ataxia, confusion and eventually convulsions and coma.

12. Organophosphates are well absorbed by inhalation, by skin absorbtion and by ingestion. There are no irritant warning signs of exposure; organophosphates pass silently through the skin leaving only localized sweating as a calling card. A child has died from licking the nozzle of a parathion sprayer.
    Patients must be completely decontaminated, first in the field and then again in the emergency department. Emergency department personnel should wear gowns and gloves (thick rubber recommended).

    Speed of onset of symptoms as well as the predominant symptom site depend on the route of exposure. Inhalation of organophosphate fumes gives the fastest onset of symptoms, usually respiratory. Skin exposure may show localized sweating and fasiculations.

13. Certain organophosphates require conversion in the liver before they exert their toxic effects. This may account for some delay in appearance of symptoms. Parathion, one of the most popular insecticides, must be converted to paraoxon in the liver before it becomes toxic.

    Regardless of the the route of exposure or the kind of organophosphate, symptoms in order to be considered to be secondary to organophosphate poisoning must occur within 24 (and probably 12) hours of exposure.

14. The diagnosis of organophosphate poisoning is made by approriat history, pathognomonic physical examination, response to atropine, and RBC cholinesterase levels.

    a. History- exposure to pesticides
       farm work
       green house

    b. Physical- the classic patient with organophosphate poisoning is comatose with pinpoint pupils, smelling of hydrocarbons and garlic, diaphoretic, having muscle twitches and fasiculations, and having excessive oral and bronchial secretions

    With a typical picture for severe organophosphate poisoning, begin treating at once.

    The mild organophosphate poisoned patient will have headache, vomiting, diarrhea, dizziness, weakness, salivations and increased secretions, and visual blurring.

    The severely poisoned patient will have coma, convulsions, bradycardia, paralysis, twitching, cyanosis, dyspnea.

    The mildly poisoned patient may rapidly progress to the severely poisoned patient.

15. Emergency Department Management

    Assure adequate oxygenation- organophosphate poisoning kills via its toxicity on the pulmonary system. The combination of excessive respiratory tract secretio (bronchorrhea) and weakness of the respiratory muscles leads to a respiratory death unless intervention occurs

    Complete decontamination- remove all clothing
    wash the patient top to bottom with soap and water

15. Complete decontamination- Clean residual accumulation spots like underneath the nails
Shampoo the hair
Wear gloves (thick rubber)

If the organophosphate exposure is an ingestion, evacuate the stomach using either ipecac or lavage depending on the level of consciousness of the patient. Be aware that organophosphates come in a hydrocarbon vehicle, but that the risk of organophosphate poisoning is greater than the risk of hydrocarbon pneumonitis that may occur with evacuation of the stomach.
Treat with charcoal.
Take precautions against aspiration.

Treat with atropine.
Atropine should be administered early, even before the decontamination procedures are being carried out. Most authorities stress the need to preoxygenate to avoid an atropine induced ventricular fibrillation.

The amount of atropine used is "heroic". Start with 1-2 mg. in an adult and .1 mg/kg in a child, intravenously. (intramuscular route can be used in the field or in the ER if no line is available)
Atropine antagonizes only the muscarinic (parasympathetic end organ) effect of the organophosphate; it has no effect on muscle weakness or CNS symptoms. It does reverse the SLUD symptomatology.
The atropine dose is repeated every 5-15 minutes in amounts sufficient to keep the patient mildly atropinized. Dryness of the mucous membranes of the mouth, flushing of the skin, slight tachycardia, and mydriasis are used as therapeutic endpoints provided that prior to atropine the patient had excess salivation, moist pale skin, bradycardia or meiosis. If the organophosphate poisoned patient initially presented with tachycardia or normal pupi as they may, the usefulness of those signs in judging atropinization is lost.

Atropine is continued for 24 hours. Patients sometimes need as much as 120 milligrams of atropine in that period.

Atropine can be used to make the diagnosis of organophospha poisoning in uncertain cases. If 1 milligram of atropine does not produce mydriasis, tachycardia, dryness of mouth and flushing of skin the patient can be considered to have a cholinesterase inhibitor in his system.

15. Treatment

Treat with pralidoxime.

Pralidoxime (2-PAM) encourages the organophosphate to separate itself from the cholinesterase.
The dose is 1-2 grams in an adult given no faster that 500 mg/min (otherwise side effects of dizziness and diaphoresis) or 20-50 mg/kg in a child.

Pralidoxime reverses the muscle weakness (nicotinic) effects in minutes. Although is is not supposed to affect the CNS toxicity of organophosphates, there are reports in the literature of patients waking up from an unresponsive state within minutes of pralidoxime administration. Pralidoxime does not work at the muscarinic end organ synapses (where atropine works). Side effects of pralidoxime are minimal.

Pralidoxime should be used whenever there are muscular symptoms or when there is a severe exposure.
It should be repeated in 1-2 hours if muscle weakness or fasiculation symptoms recur; anyone who benefited from a first dose deserves a second one 12 hours later.

Early use of pralidoxime may prevent late neuromuscular paralysis syndromes seen with organophosphate poisoning.

Organophosphate compounds undergo a process of ageing on the cholinesterase molecule such that after 24-48 hours the bond becomes absolutely inseparable and the pralidoxime is completely ineffective. Use pralidoxime within 24 hours of exposure only.

There is another class of cholinesterase inhibitors that are used as pesticides: the carbamates. The most commonly used ia carbaryl (Sevin). They are less toxic because the carbamate cholinesterase bond more quickly reverses than the organophosphate-cholinesterase bond. In addition, absorbtion is less complete. The therapeutic drugs edrophonium (Tensilon) and physostigmine belong to the carbamate class.
Carbamate poisoning presents with the same clinical findings as the organophosphates. Cases of it should be treated vigorously with atropine but there is some question as to whether pralidoxime should be used. There are some reports that it interferes with the effectiveness of atropine therapy, especially in carbaryl poisoning. However, current thinking seems to be to go ahead and use the pralidoxime regardless of the type of anticholinesterase poison.