CHAPTER 12

BITES (SNAKE, INSECT, AND ANIMAL)

12-1. SNAKE BITES.

a. Classification of poisonous snakes.

(1) Crotalidae (viperine). Frequently called pit vipers (rattlesnake, moccasins, copperhead, bushmaster, fer-de-lance, habu, Russel's viper, etc.).

(2) Elapidae. This family is composed of coral snakes, kraits, cobras, mambas, asps, and others.

(3) Hydrophidae (sea snakes). All are extremely poisonous and many have more toxic venom than cobras.

(4) Colubridae. This family is represented by the backfanged boomslang.

b. Classification of snake venom. Snake venom is broken into two categories: hemotoxic and neurotoxic. Unfortunately snakes are not just hemotoxic or neurotoxic. They are primarily one or the other, but contain elements of both.

(1) Hemotoxic. Members of the Crotalidae family are primarily hemotoxic with the following substances in the venom:

(a) Thrombase. Action mainly at the site of the bite, causing local thrombosis, gangrene, and intravascular clotting.

(b) Hemorrhagin. This is the predominant substance in the venom, causing lysis of the capillary cells with resultant leakage into the tissue. This starts locally and then becomes generalized. Convulsions due to small hemorrhages in the brain sometimes occur.

(c) Anticoagulin. Causes a breaking down of proteins in the fibrin network of the clot.

(2) Neurotoxic. Members of the Elapidae, Colubridae, and Hydrophidae families are primarily neurotoxic with the following substances in the venom:

(a) Neurotoxin. Has paralytic effect on the respiratory center and the 9th, 10th, 11th, and 12th pairs of cranial nerves.

(b) Hemolysin. Found in some varieties; causes lysis of blood cells.

(c) Cardiotoxin. Causes toxic cardiac arrest.

c. Diagnosis of snakebite.

(1) Crotalidae. Symptoms are very marked and onset is rapid.

(a) Tissue swelling at site of bite, gradually spreading to surrounding area. Swelling begins within 3 minutes and may continue for an

hour with enough severity to burst the skin.

(b) Excruciating pain at site of bite.

(c) Often presence of fang marks.

(d) Bleeding from major organs that may show up as blood in the urine.

(e) Destruction of blood cells and other tissue cells.

(f) Severe headache and thirst.

(g) A marked fall of B.P. with a corresponding rise in pulse.

(h) Bleeding into surrounding tissue.

NOTE: Death may occur within 24-48 hours if bite is serious and untreated. Even with proper treatment, there is grave danger of loss of a portion of the extremities.

(2) Elapidae and Colubridae. Symptoms not as marked and onset is usually slower than Crotalidae.

(a) Impairment of circulation with irregular heartbeat, drop in B.P., weakness, and exhaustion terminating in shock.

(b) Severe headache, dizziness, blurred vision, hearing difficulty, confusion, and unconsciousness.

(c) Muscular incoordination and muscular twitching.

(d) Respiratory difficulty leading to respiratory paralysis.

(e) Irregularities of skin sensations such as tingling, parasthesia, excessive perspiration, and numbness of the lips and the soles of the feet.

(f) Chills and often rapid onset of a fever.

(g) Nausea, vomiting, and diarrhea.

(3) Hydrophidae. Neurotoxic, bite is usually painless, does not swell, and often there is no clue that treatment should be started. Poisoning should be suspected in those who have been in coastal waters frequented by sea snakes within 1-2 hours before complaining of:

(a) Muscular aches, pains, and stiffness of movement.

(b) Pain on passive movement of arm, thigh, neck, or trunk muscles.

(c) Urine becomes reddish brown within 3 hours.

(d) There is a consistent appearance of neurotoxic symptoms as outlined in Elapidae diagnosis.

NOTE: Without treatment death usually occurs within 12-24 hours.

d. Treatment of snakebite.

(1) General treatment for all snakebites.

(a) Kill the snake if possible, but do not spend more than a few minutes and avoid overexertion in the attempt. Try not to crush the head as this is the primary source of exact species identification.

(b) Have patient lie down. Immediately immobilize injured part. Keep patient warm and quiet.

(c) Tetanus booster and antibiotics are indicated.

(d) Symptomatic treatment as necessary.

(2) Treatment for Crotalidae (viperine).

(a) General treatment for all snakebites (see para d (1) above).

(b) If bitten on a large area of the body (i.e., thigh, calf, forearm, etc.), make an incision 1/8 to 1/4 inch deep along or in the direction of muscle (not across the tissue) through the puncture sites. (Do not make an X cut. Do not cut into joints, tendons, etc.) Then suction using a mechanical device; use mouth only as a last resort and then only if you have no cavities, cuts, or sores in the mouth.

NOTE: Incision and suction should not be used if antivenom can be given within 1 hour or if 1 hour or more has elapsed since the bite.

(c) Do not use a tourniquet, constricting bands, or cold packs.

(d) Do not allow the patient to eat any food or to drink alcohol.

(e) Have patient drink small amounts of water at frequent intervals.

(f) Initiate IV D5W, normal saline, or Ringer's to help prevent hemolytic shock.

(g) Administer specific antivenom, if available and species is known, or polyvalent antivenom as soon as possible.

1. Inject 0.1 cc. subcutaneously and observe patient for 15 minutes for symptoms of allergy such as itching, swelling, and redness at injection site.

2. If patient is not allergic, inject the antivenom in one dose IM at a site other than the bite area.

3. If patient is allergic to the antivenom, but there is no doubt that he has an effective bite by a very dangerous species and will surely die without the antivenom, inject divided doses of 1.0 cc. IM very slowly. Be prepared to treat anaphylactic reactions should they

occur.

(h) Use morphine or other suitable pain relievers as necessary.

(3) Treatment for Elapidae and Colubridae (boomslang).

(a) Apply a tourniquet around the affected limb, over a single bone (above the knee or elbow) proximal to the bite, tight enough to stop arterial flow. This tourniquet should be released for 30 seconds every 20 minutes to allow fresh blood into the affected area.

(b) Administer antivenom using the same rules and precautions as for viperine bites.

(c) General treatment for all snakebites (see para d (1) above).

(d) Do not use morphine or any drugs that cause respiratory depression.

(4) Treatment for Hydrophidae (sea snakes).

(a) Antivenom is the only treatment other than symptomatic care.

(b) Incision and suction are of no value.

12-2. INSECT AND SPIDER BITES.

a. Insect bites. Of all deaths per year due to bites, 40% are caused by insect bites compared to 33% for snake bites, 18% for spider bites, and 9% for animal bites.

(1) Bees, wasp, hornets, yellow jackets, and ants. Most of this group sting their victims and often leave the stingers and venom sac embedded in the skin. The stinger should be removed immediately to prevent more venom from entering the victim. Toxins from this group are similar to the venom of viperine snakes in having a hemolysin factor, but their primary effect seems to be the strong histamine they contain.

(a) Symptoms. Stinging, burning sensation with swelling. This swelling, when caused by stings around the head and neck, may be severe enough to impair the airway.

(b) Treatment.

1. Apply a paste of baking soda (sodium bicarbonate) or apply strong household ammonia to reduce discomfort. Infiltration of lidocaine into sting area often helps.

2. In severe cases give Benadryl 4 mg. per kg. IV stat. with 10 cc. of 10% calcium gluconate. Inject 2 to 4 cc. fairly fast until patient has a burning sensation in the tongue, palm, or soles of his feet. Then slow the injection of the remainder to avoid flushing.

3. If patient is allergic to the venom, treat the anaphylactic reaction.

(2) Centipedes, millipedes, and caterpillars.

(a) Centipedes are venomous with hollow fangs like snakes. If bitten, the patient will have immediate severe pain followed by redness and swelling. Sometimes necrosis with ulcer formation may occur.

(b) Millipedes secrete a toxin by glands in the body. When the fluid touches the skin, it produces burning and itching.

(c) Many caterpillars have hollow venom-containing hairs on their bodies. If these hairs contact the skin, they cause severe burning pain, redness, swelling, and necrosis of tissue. Scotch tape on the sting is effective in removing the broken off hairs from the skin.

(d) Treatment. Very similar to that of bee, wasp, and hornet stings. Antihistamines, ice, and pain medication are helpful. Treat anaphylactic reactions.

b. Spider and scorpion bites.

(1) Black widow spider. Only the female bites and has a neurotoxic venom. Identified by red hourglass on abdomen.

(a) Symptoms. Initial pain is not severe, but severe local pain rapidly develops. The pain gradually spreads over the entire body and settles in the abdomen and legs. Abdominal cramps and progressive abdominal rigidity may occur. Weakness, tremors, sweating, salivation, nausea, vomiting, and/or a rash may occur. Anaphylactic reactions can occur. Symptoms usually begin to regress after several hours and are usually gone in a few days.

(b) Treatment.

1. Calcium gluconate 10 cc. of 10% solution IM or injected slowly IV.

2. Robaxin 10 cc. given slowly IV over a 5-10 min period followed by 10 cc. in 250 cc. of D5W in IV drip over 4 hours.

3. Patients under 14 and over 50 should receive the specific antivenom if they are not allergic to horse serum.

4. Supportive care as necessary, tetanus booster, antibiotics, etc.

(2) Brown house spider (recluse). Identified by dark brown violin on the back of a small light brown spider.

(a) Symptoms. There is no pain or so little pain that most of the time the patient is not aware he is bitten. A few hours later a painful, red area with a mottled cyanotic center appears. A macular rash sometimes occurs. Necrosis does not occur in all bites, but usually after 2-3 days there is an area of discoloration that does not blanch with finger pressure. The area turns dark and mummified in a week or two. The margins separate and the eschar falls off leaving an open ulcer. Secondary infection and regional lymphadenopathy usually become evident at this stage. Many times the patient is unaware of any cause for the ulcer. The

persists for weeks or months. Physical exam reveals a hard indurated area of skin and superficial fascia with undermined edges.

In many cases there is a systemic reaction, in addition to the ulcer, that is serious and may lead to death. The systemic reactions occur chiefly in children and are marked by fever, chills, joint pain, splenomegaly, vomiting, and a generalized rash. These systemic reactions may occur at any time as long as the ulcer is present.

(b) Treatment. There is no antivenom for brown recluse bites. It is necessary to excise all the indurated skin and fascia before healing will start. If the ulcer is not excised, it may continue to grow until it is several inches in diameter.

Tetanus prophylaxis and antibiotics are necessary to control secondary infection. Cortisone will arrest the systemic reaction but will not affect the ulcer. Anaphylactic reactions may also occur and must be managed.

(3) Scorpions. All are poisonous to a greater or lesser degree. Fortunately none of the very poisonous varieties are found in the US, but deaths have been reported due to scorpion stings in the US.

(a) Symptoms. There are two different reactions depending on the species.

1. Severe local reaction only, with pain and swelling around area of sting. Possible prickly sensation around the mouth and a thick feeling tongue.

2. Severe systemic reaction with little or no visible local reaction. Local pain and hyperesthesia may be present. Systemic reaction includes respiratory difficulties, thick feeling tongue, tetanuslike body spasm, drooling, gastric distention, double vision, blindness, involuntary rapid movement of the eyeball, involuntary urination and defecation, hypertension, and heart failure. Death is rare, occurring mainly in children or adults with hypertension.

(b) Treatment.

1. DO NOT give morphine or morphine derivatives, including Demerol, because it has a synergistic effect with scorpion venom. Effective pain relief can be obtained by specific nerve blocks using lidocaine.

2. Ice packs or cold water helps slow spread of toxin and relieve pain.

3. Tetanus prophylaxis and antibiotics are indicated.

4. Specific antivenoms are available for the more toxic varieties.

5. Symptomatic care.

12-3. ANIMAL BITES.

a. Animal bites themselves are not usually serious. The main

problem is the diseases that can be transmitted by the bites. Number one among these is rabies.

b. Protective measures for bites.

(1) Capture and isolate animal for 8-10 days.

(a) An animal that is rabid should show unmistakable signs of rabies within 8 days.

(b) If the animal dies, cut off the head, freeze it, and ship it frozen to the nearest laboratory having facilities for rabies determination.

(2) Bites from animals that can't be captured and isolated should be considered as rabid, and patient should receive antirabies vaccine.

c. Treatment.

(1) All bites must be promptly and thoroughly cleaned with soap, Betadine or hexachlorophene and water. Then apply either 40-70% alcohol, tincture of iodine, or 1:10,000 benzalkonium chloride directly into the bite. This mechanical cleansing and disinfecting has been credited with blocking many cases of rabies as well as lessening the chances of other types of infection.

(2) Antitetanus prophylaxis is indicated.

(3) Avoid suturing or cauterizing the wound; use delayed secondary closure if at all possible.

(4) If suturing is absolutely necessary, infiltrate 50% of the first dose of rabies vaccine into wound area.

(5) Immediate judgment as to the advisability of administering antirabies serum is required. Take into account the circumstances of the bite and prevalance of rabies in the area.

(6) Symptomatic treatment as required.

CHAPTER 13

OVERDOSE AND POISONING

13-1. GENERAL PRINCIPLES.

a. ABCs in severe OD.

b. Gradation of coma.

Stage 0: Asleep but arousable.
Stage I: Comatose - withdraws from painful stimuli.
Stage II: Reflexes present - does not withdraw from painful stimuli.
Stage III: Reflexes absent - no respiratory or circulatory depression.
Stage IV: Reflexes absent - respiratory depression and/or circulatory collapse.

c. Removal of toxic drug.

(1) Emesis.

(a) More effective than lavage in awake patient.

(b) Ipecac: 15-30-45 cc. of syrup (not tincture) followed by 1 liter of warm water. Activity helps stimulate vomiting. Never leave ipecac in (cardio toxic).

(c) Apomorphine: 0.1 mg./kg. IV or SQ. Vigorous vomiting difficult to control. May act as respiratory depressant: counteracted by Narcan. (Rarely indicated.)

(2) Lavage.

(a) Use normal saline - 200-300 cc. per pass, or 10 cc./kg. per pass.

(b) Y tube set up with 28-32 Fr. Ewald Tube, larger for undissolved tabs.

(c) If no gag use endotrachial tube to protect airway.

(3) Contraindications to emesis and/or lavage.

(a) Caustics.

(b) Hydrocarbon ingestions that are not potentially lethal.

(4) Activated charcoal.

(a) Dose = 30-50 grams.

(b) Will deactivate ipecac. (Wait until patient stops vomiting.)

(c) Can be given prior to and after lavage.

(5) Purgatives.

(a) Castor oil or mineral oil: contraindicated in pesticide poisoning and hydrocarbon ingestion. Helpful in glutethimide.

(b) Mag citrate: 10 oz. Contraindicated in renal failure.

(c) Sodium sulfate: 250 mg./kg. diluted 1:2 or 1:4.

(6) Forced diuresis with alkalinization of urine.

(a) Especially useful in barbiturate and ASA OD.

(b) Protocol.

500 cc. D_5W x 2
500 cc. NS + 20 KCl at 200-500 cc./hr.
1 Amp $HaHCO_3$/2-3 liters.
Lasix 40 mg. IV q. 4-6 h.
Foley for urethral drainage.

(7) Forced diuresis with acidification of urine.

(a) Especially useful in amphetamine and PCP ODs.

(b) Protocol.

1. Ascorbic acid: 1-2 gm in 500 cc. NS IV q.i.d. and/or

2. Ammonium chloride: 2.75 mEq./kg./dose in 60 cc. saline P.O q.6h. until urine pH less than 5.

(8) Peritoneal and hemodialysis.

13-2. HYPNOTICS.

a. Barbiturates signs and symptoms.

(1) C.N.S. depression and/or agitation (coma is major toxicity).

(2) Temperature usually decreased.

(3) Pulse usually normal, may be increased.

(4) B.P. normal or decreased.

(5) Respirations normal or decreased (if increased, consider aspiration).

(6) "Barb burns" - skin necrosis at high dosage.

(7) Reflexes normal or decreased.

(8) Myocardial toxicity at high dosages.

(9) Ataxia, nystagmus, and vertigo in early OD.

b. Treatment for barbiturates.

(1) General OD protocol.

(2) Hemodialysis.

c. Nonbarbiturate hypnotics.

Glutethimide (Doriden)
Methyprylon (Noludar)
Quaalude (methaqualone)

(1) Signs and symptoms.

(a) Lethargy.

(b) Mydriasis.

(c) Decreased B.P.

(d) Flaccidity (except Quaalude).*
* Quaalude, Placidyl, or phencyclidine can cause hyperreflexia in the presence of coma.

(e) Respiratory depression.

(2) Treatment as for general OD: Glutethimide especially lethal because of enterohepatic circ and varying levels of coma. Use duodenal NG and cholestyramine.

13-3. C.N.S. STIMULANTS.

Amphetamines and cocaine.

a. Signs and symptoms.

(1) Agitation.

(2) Euphoria.

(3) Tachycardia.

(4) Hypertension.

(5) Hyperpyrexia.

(6) Cramps.

(7) Hallucinations (auditory and visual).

(8) Convulsions and coma.

(9) Perforated nasal septum suggests cocaine.

(10) Toxicity begins at 20-25 mg./kg. for amphetamines.

b. Treatment.

(1) Support and calming, avoid stimulation.

(2) Dialysis for amphetamines, but not for cocaine.

(3) Thorazine 25 mg. or valium 5 mg. as calmative.

(4) Acidify urine for amphetamines.

13-4. ANTICHOLINERGICS.

a. Examples.

(1) Atropine.

(2) Scopolamine.

(3) Belladonnna alkaloids.

(4) Tricyclic antidepressants.

(5) Phenothiazines.

(6) Antihistaminics.

(7) Antispasmodics (Pro-Banthine).

(8) Antiparkinsonian agent.

(9) Toxic plants.

(a) Jimsonweed.

(b) Morning glory seed.

(c) Deadly nightshade.

(d) Certain mushrooms.

(e) Potato leaves and sprouts.

b. Signs and symptoms.

(1) Tachycardia.

(2) Dry flushed skin.

(3) Mydriasis.

(4) Dry mouth.

(5) Nausea and vomiting.

(6) Urinary retention.

(7) Increased intraocular pressure.

(8) Confusion.

(9) Disorientation.

(10) Bizarre behavior.

(11) Paranoia.

(12) Hallucinations.

(13) Hyperpyrexia.

(14) Hypotension.

(15) Convulsions.

c. Treatment.

(1) Cooling.

(2) Support.

(3) Sedation.

(4) Physostigmine in severe OD.

13-5. MAJOR AND MINOR TRANQUILIZERS.

Major - Phenothiazines.
Minor - Librium, valium, Placidyl, meprobamate, etc...

a. Signs and symptoms.

(1) Hypotension.

(2) Lethargy.

(3) Respiratory depression.

(4) Coma.

(5) Seizures.

(6) Extrapyramidal ------ Phenothiazines.

(7) Loss of temperature control.

b. Treatment.

(1) Support and general OD measures.

(2) Gastric lavage or emesis.

(3) Control shock as necessary.

(4) Use barbiturates cautiously for control of convulsions.

13-6. USE OF PHYSOSTIGMINE.

Anticholinesterase - i.e., cholinergic drug counteracts

anticholinergic drugs.

a. Actions.

(1) Pupillary constriction.

(2) Contracts bronchioles, gut, and bladder.

(3) Stimulates salivation and sweating.

(4) Slows heart.

(5) Increases muscle contraction.

(6) C.N.S. stimulant.

b. Definite antidotes for:

(1) Atropine and belladonna alkaloids (jimsonweed).

(2) Tricyclic antidepressants.

c. Contraindications.

(1) Mechanical G.I. obstruction - absolute.

(2) Mechanical GU obstruction - absolute.

(3) Asthma - relative.

(4) COPD - relative.

(5) ASCVD - relative.

(6) Should be used to counteract life-threatening central and peripheral anticholinergic signs, or to reverse coma in the presence of those signs.

d. Central anticholinergic signs.

(1) Short-term memory loss.

(2) Disorientation.

(3) Hallucinations (visual and auditory).

(4) Anxiety and agitation.

(5) Psychosis.

(6) Coma.

(7) Twitchy and jerky movements.

(8) Pyramidal signs (Hyperreflexia, hypertonus, clonus).

e. Peripheral anticholinergic signs.

(1) Tachycardia.

(2) Mydriasis with decreased light reflex.

(3) Dry mucous membranes.

(4) Flushed dry skin.

(5) Decreased bowel sounds.

(6) Urinary retention.

(7) Hyperpyrexia.

f. Physostigmine dosage.

(1) 2 mg. in 10 cc. IV over 2 minutes repeated x 2 at 5-10 minutes.

(2) 2 mg. undiluted IM q. 20 minutes.

(3) 0.5 mg. in children.

g. Dangers of Physostigmine.

(1) Precipitate cholinergic crisis and seizures.

(2) Central (medullary) and peripheral respiratory collapse.

(3) Excess salivary and tracheobronchial secretions.

(4) Bronchospasm and laryngospasm.

(5) Muscle twitching, fasciculations, and paralysis.

(6) Reversible - treat with atropine 1/2 physo dose IM.

h. Indications.

(1) Known OD on belladonna alkaloids or tricyclics who are in marked distress.

(2) Anticholinergic findings and hypertension, hallucinations, coma, convulsions, and dysrhythmias.

13-7. HYDROCARBONS.

a. 1 cc./kg. of hydrocarbon, or less if chlorinated or metal containing solvent. Leads to C.N.S. or respiratory depression.

b. If depression has occurred - intubate with cuffed tube gastric lavage and use general OD procedures.

c. Potentially lethal dose but no depression, use ipecac.

d. If less than 1/2 cc./kg., cathartic only - no emesis or lavage.

e. Activated charcoal is useless.

f. Don't use oil-based cathartics. May lead to lipoid pneumonias.

g. X ray initially and at 6-12 hours if evidence of aspiration (patient coughing).

h. Epinephrine contraindicated (dysrhythmias).

i. Steroids are questionably useful.

j. No antibiotics unless infection documented.

13-8. SALICYLATES: ASA AND METHYL SALICYLATE (OIL OF WINTERGREEN).

a. Symptoms at 100 mg./kg. (2 grains/lb toxic - 3-4 gr/lb fatal).

b. Is severe to fatal at 250-400 mg./kg.

c. Adult ingestion of 10 grams should be considered serious.

d. Symptoms.

(1) Early: Headache, dizziness, tinnitus, blurred vision, confusion, lethargy, diaphoresis, thirst, nausea and vomiting, diarrhea, abdominal pain, hyperpnea.

(2) Severe: Restlessness, incoherence, vertigo, tremor, diplopia, delirium, convulsions, coma, fever to 106°F. in children, respiratory alkalosis in adults and children, occasionally hypoglycemia.

e. Treatment.

(1) Basic principles, especially activated charcoal.

(2) Correct acid-base and electrolyte disturbances.

(3) Alkalinization of urine.

13-9. ACETAMINOPHEN OVERDOSAGE (PARACETAMOL).

a. In adult dosages over 5 grams, should be admitted for observations.

b. 10 grams ingestion is associated with severe hepatotoxicity and death.

c. Peak plasma levels usually occur in 40-120 minutes, may be prolonged in OD.

d. Metabolized in liver and conjugated with glucuronide, sulfate, cysteine, and mercapturic acid.

e. Effects are potentiated by drugs utilizing the same conjugating systems, i.e. ETOH and barbiturates.

f. Plasma levels 4 hours after ingestion.

(1) Greater than 300 mg./ml. leads to liver damage.

(2) Less than 120 mg./ml. does not lead to liver damage.

g. Manifestations.

(1) Pallor.

(2) Nausea, vomiting, and diarrhea.

(3) Hepatotoxicity.

(a) May not appear clinically for 2-6 days.

(b) Right upper quadrant pain.

(c) Increased liver enzymes.

(d) Jaundice.

(e) Encephalopathy.

h. Treatment.

(1) Emesis early.

(2) Charcoal early questionably useful.

(3) N-acetylcysteamine (Mucomist) - not yet approved.

(a) 140 mg./kg. P.O. loading dose.

(b) 70 mg./kg. P.O. q.4h. x 17 doses.

13-10. CAUSTICS.

a. Examples.

(1) Draino, Liquid Plummer, etc...

(2) Liquid and dry bleach.

(3) Acetest tablets.

(4) Lye.

(5) Any strong acid or base.

b. Treatment.

(1) No emesis.

(2) No lavage.

(3) Dilute with cool water.

(4) Do not neutralize with weak acid or base (generates heat and gas).

(5) All caustic ingestions should be scoped and followed by G.I.

or ENT.

c. N.B. - Absence of intra or perioral burns does not preclude ingestion.

13-11. ORGANOPHOSPHATES.

a. Examples. (insecticides replacing DDT).

(1) Organophosphate insecticides - chlorathion, Di-Captan, ethion, disyston, malathion, methyl parathion, parathion, phosphamidon, etc...

(2) Carbamate insecticides - cabofuran, dimeton, mexecarbate, etc...

b. Signs and symptoms consist of increased cholinergic stimulation.

(1) Bronchoconstriction with increased bronchial secretion and pulmonary edema.

(2) Nausea and vomiting.

(3) Abdominal cramps and diarrhea.

(4) Increased sweating.

(5) Increased salivation and lacrimation.

(6) Bradycardia and hypotension.

(7) Miosis (may be unilateral) and blurred vision.

(8) Urinary incontinence.

(9) Muscle cramps, weakness, fasciculations, and arreflexia.

(10) Headache.

(11) Restlessness.

(12) Convulsions.

(13) Coma.

c. Treatment.

(1) Mild intoxication requires removal from further exposure.

(2) Severe poisoning.

(a) Support (including removal of secretions).

(b) Decontamination.

(c) Administration of anticholinergic agent.

1. Atropine - 2-4 mg. IV slow q. 15 minutes.

2. PAM pralidoxime - only useful if administered within 24 hours (1 gram over several minutes, children 10-12 mg./kg.)

13-12. CYANIDE.

a. Sources of cyanide (amygdalin).

(1) Nitroprusside therapy.

(2) Laetrile.

(3) Various fruit pits such as peach, apricot, chokecherry, plum, lima beans, apple seeds, various grasses.

(4) Synthetic rubber.

(5) Some fumigant gasses.

(6) Photographic chemicals.

(7) Salts for electroplating, gold and silver extraction, metal cleaning, dehairing hides.

(8) Cyanamide (for fertilizing).

b. Signs and symptoms of cyanide poisoning.

(1) Smell of bitter almonds.

(2) Headache.

(3) Vertigo and faintness. C.N.S.

(4) Excitability.

(5) Opisthotonus and trismus.

(6) Convulsions and coma.

(7) Burning tongue.

(8) Salivation. Oral ingestion

(9) Nausea.

(10) Hypertension with bradycardia and blocks early.

(11) Hypotension, tachycardia, and cardiovascular collapse - late.

c. Treatment.

(1) 100% O_2 by bag and mask.

(2) Cyanide antidote kit:

(a) Amy nitrite by inhalation followed by,

(b) Sodium nitrite IV 10 ml. over 4 minutes (0.2 ml./kg. IV for peds), and

(c) 50 ml. of 25% sodium thiosulfate IV (1 ml./kg. IV for peds).

13-13. ARSENIC.

a. Sources.

(1) Herbicides.

(2) Insecticides.

(3) Rodenticides.

(4) Fungicides.

(5) Paints.

(6) Tanning agents.

(7) Some veterinary medicines.

b. Signs and symptoms (levels in urine, hair, and nails).

(1) Smell of garlic.

(2) Mees' lines in nails after 2-3 weeks.

(3) Hyperpyrexia.

(4) Tremor and convulsions.

(5) Coma.

(6) Nausea, vomiting, and diarrhea.

(7) Liver and kidney damage.

(8) Polyneuropathy.

(9) G.I. complaints may preceed neuropathy, and by 2-3 weeks in acute cases.

c. Treatment.

(1) If acute, lavage or emesis.

(2) General support.

(3) Dimercaprol (BAL). (See chapter 14.)

(4) Follow urine arsenic levels.

13-14. METHEMOGLOBINEMIA.

a. Hemoglobin with Fe in the ferric state.

b. Incapable of reversible transport.

c. Causative agents (oxydizers).

(1) Inorganic agents:
Copper chlorates, chromates, nitrates, nitrites.

(2) Drugs:
Acetanilid, phenacetin, PAS, sulfonamide, lidocaine, benzocaine, nitroglycerin, chloroquine, menthol, primaquine, etc...

(3) Miscellaneous:
Alloxan, naphtalene, quinones anilines nitrosobenzene.

d. Symptoms.

(1) Cyanosis with level 727 (slate gray).

(2) Normal pO2.

(3) Dyspnea.

(4) Tachycardia.

(5) Stupor and coma.

(6) Nonspecific symptoms.

(7) Blood looks like Hershey's Chocolate.

e. Treatment.

(1) Support.

(2) O2.

(3) Time.

(4) Methylene blue - 1-2 mg./kg. IV slowly or 3-5 mg./kg. orally.

13-15. CARBON MONOXIDE.

a. Sources.

(1) Car exhausts.

(2) Poorly ventilated furnaces and fireplaces.

(3) Smoke inhalation.

b. Symptoms.

(1) Depends on level - become toxic over 20%, 10% if found in smoking.

(2) Cherry red lips and mucous membranes.

(3) Headache.

(4) Muscle weakness.

(5) Palpitations.

(6) Dizziness.

(7) Confusion.

(8) Coma and death.

c. Treatment.

100% O_2 under hyperbaric conditions for at least 1 hour; maintain body warmth and blood pressure. Give 50 ml. 50% glucose p.r.n. for brain edema.

13-16. LEAD POISONING (PLUMBISM).

a. Sources.

(1) Lead based paint.

(2) Cooking utensils - ceramic or earthenware with lead glaze.

(3) Plumbing.

(4) Stills.

(5) Industrial exposure:

(a) Smelters.

(b) Battery workers.

(c) Painters (auto).

(d) Demolition experts.

b. Signs and symptoms.

(1) Chronic.

(a) Vague aches and pains (may mimic other neuropathies).

(b) Wrist and ankle drop.

(c) Chronic nephritis.

(d) Anemia Hg 10 with basophilic stippling.

(e) Increased urinary aminolevulinic acid (ALA).

(f) Abdominal lead and leadlines on X ray.

(2) Acute.

(a) Metallic taste.

(b) Anorexia.

(c) Constipation and vomiting.

(d) Abdominal pain.

(e) Personality changes.

(f) Lethargy.

(g) Clumsiness.

(h) Ataxia.

(i) Convulsions.

(j) Coma.

(k) Anemia with basophilic stippling.

(l) Increased urinary ALA and coproporphyrins.

(m) Abdominal lead and lead lines on X ray.

(n) Blackstools (leadsulfide).

c. Treatment.

(1) Isolation from further contaminations.

(2) General OD principles.

(3) General support.

(4) Chelation.

(a) BAL 4 mg./kg. IM over 4 h.

(b) EDTA 50-75 mg./kg./day 1 m. 5-7 day courses.

(c) D-penicillamine 20-40 mg./kg./day as outpatient.

13-17. IRON INTOXICATION.

a. Source is usually prescription or over the counter Fe containing preparations.

b. Toxic to lethal dose of 150-300 mg./kg.

c. Signs and symptoms.

(1) Stage I.

(a) 30-120 minutes.

(b) Vomiting and diarrhea (may be bloody).

(2) Stage II.

(a) 6-12 hours.

(b) Latent or improvement.

(3) Stage III.

(a) 18-72 hours.

(b) Cardiovascular collapse.

(c) Coma.

(d) Convulsions.

(e) Coagulation defects.

(f) Hyperpyrexia.

(g) Metabolic acidosis.

(h) Liver failure.

(4) Stage IV.

(a) 4-6 weeks.

(b) Pyloric stenosis and G.I. scarring.

d. Treatment.

(1) Lavage with 5% sodium bicarbonate or emesis if pills still visible on abdominal X rays.

(2) Baseline blood work to include serum Fe.

(3) Chelation only in inpatients (Desferal).

(a) Used in all definitely lethal doses, 300 mg./kg.

(b) Serum Fe TIBC.

(c) Coma, convulsions, and shock.

(d) 40 mg./kg. Desferal 1., to be repeated at expanding time intervals. Do not exceed 1 gm IM initially followed by 500 mg. q.4h. x 2. Never exceed 6 gm in 27 hours.

(e) Urine will turn brown.

(f) Supportive treatment.

CHAPTER 14

NUCLEAR, BIOLOGICAL, CHEMICAL (NBC)

14-1. NUCLEAR. The major problems resulting from nuclear detonation are mass casualties and the destruction of medical care facilities.

a. Of the injured survivors, about one-third of the injuries will be caused by blast effects, one-third by thermal effects (burns), and one-third by both blast and thermal effects. Some in each of these groups will receive radiation from initial radiation and/or radioactive fallout.

(1) Initial treatment for these casualties will be first aid or self aid until they can get to or be brought to a functioning medical care facility.

(2) Once the casualties reach a treatment facility, they must be classified as to the type and urgency of treatment required so appropriate priorities can be established for treatment, evacuation, and hospitalization. This classification is known as triage. Triage is divided into four categories:

(a) Minimal (priority I). Requires only minor treatment, usually on an ambulatory or outpatient basis. This group includes small lacerations and contusions, closed fractures of small bones, second degree burns of less than 20% of the body that are not life threatening, and moderate psychological disorders.

(b) Immediate (priority II). Individuals with life-threatening conditions or moderate injuries that are treatable with a minimum expenditure of time, personnel, and supplies, and who have a good chance of recovery. Conditions include hemorrhage from an accessible site, rapidly correctable mechanical defects (sucking chest wound, respiratory obstruction or distress), severe crushing wounds and incomplete amputations, and open fractures of major bones.

(c) Delayed (priority III). After emergency care, these individuals may have definitive treatment delayed without significant jeopardy to recovery. These include moderate lacerations without bleeding, closed fractures of major bones, noncritical central nervous system (C.N.S.) injuries, and second degree burns between 20 and 40% of the body surface.

(d) Expectant (priority IV). Individuals requiring extensive therapy beyond our means and to the detriment of others. They receive emergency, comfort, and conservative care to the maximum extent possible. Included are critical respiratory and C.N.S. injuries, penetrating abdominal wounds, multiple severe injuries, and severe burns of over 40% of the body surface.

b. Burn and blast injuries are covered in chapter 10.

c. Radiation injuries (acute radiation syndrome) are directly related to the dose (amount) of radiation received. The dose is accumulative.

(1) 50-200 rad. Approximately 6 hours after exposure the individual may have no symptoms to transient mild headaches. There may be

a slight decrease in the ability to conduct normal duties. Less than 5% of individuals in the upper part of the exposure range will require hospitalization. Average hospital stay will be 45-60 days with no deaths.

(2) 200-500 rad. Approximately 4-6 hours after exposure, individuals will experience headaches, malaise, nausea, and vomiting. Symptoms are not relieved by antiemetics in the upper exposure range. Individual can perform routine tasks but any activity requiring moderate to heavy exertion will be hampered for 6-20 hours. After this period, individuals will appear to recover and enter a latent period of 17-21 days. If individual has received 300 rads or more, large quantities of hair will be lost between 12-18 days after exposure. Following the latent stage, symptoms will return, requiring 90% of the personnel to be hospitalized for 60-90 days. Probably less than 5% of those at the lower dose range will die, the percentage increasing toward the upper end of the dose range.

(3) 500-1000 rad. Approximately 1-4 hours after exposure, severe and prolonged nausea and vomiting develop that are difficult to control. Diarrhea and fever develop early in individuals in the upper part of the exposure range. Simple routine tasks can be performed by individuals in the lower dose range. Significant incapacitation is seen in the upper ranges. Initial symptoms last for more than 24 hours, then go into a latent period lasting 7-10 days. Following the latent stage the symptoms return requiring 100% of the individuals to be hospitalized. Of those in the lower range 50% will die, the percentage increasing toward the upper range. All deaths occur within 45 days. The survivors require 90-120 days hospitalization before recovery.

(4) 1000 rad or more. Less than 1 hour after exposure individuals develop severe vomiting, diarrhea, and prostration. There is no latent period. All require hospitalization and die within 30 days.

d. Treatment for radiation exposure includes washing individual thoroughly to remove any radioactive contamination, symptomatic treatment, and prevention of secondary infections.

14-2. BIOLOGICAL WARFARE (BW).

a. Biological agents are divided into two main classes:

(1) Living organisms such as bacteria, viruses, rickettsiae, and fungi.

(2) Poisonous products or toxins produced by living organisms.

b. The most practical method of initiating infection in BW is through the dispersal of agents as minute, airborne particles (aerosols) over a target where they may be inhaled. An aerosol may be effective for some time after delivery, as it will be deposited on clothing, equipment, and soil. When the clothing is used later, or dust is stirred up, personnel may be subject to a "secondary" aerosol.

c. Agents may be able to use portals of entry into the body other than the respiratory tract. Individuals may be infected by ingestion of contaminated food and water or even by direct contact with the skin or mucous membranes through abraided or broken skin.

d. Early warning, immediate detection, and rapid identification of

the agent used in a BW attack are of primary importance.

(1) Early warning can sometimes be supplied by intelligence sources, but early warning is not always available.

(2) Immediate detection can be by seeing a plane spraying or by bombs, shells, or mines producing dense clouds near your area. Immediate detection may not occur; for example, in the case of sabotage or an attack launched a considerable distance upwind from you, the first indication may be the appearance of casualties.

(3) Rapid identification of the biological agent. Due to the concentration and/or portal of entry (respiratory tract), there may be a more rapid onset and wide variances to normal symptoms of even common diseases. This can make diagnosis and treatment extremely difficult. Clinical samples should be collected from the first casualties and sent to the nearest laboratory, if possible.

e. Individual protection prior to and during a BW attack.

(1) Maintain body in the best possible physical condition.

(2) If a BW attack is detected,

(a) Use mask.

(b) Button clothing and tie clothing with string or extra shoelaces at the wrists and ankles. If special protective clothing is available, put it on.

(c) Put on gloves, if available.

(d) While in the contaminated area, practice the procedures outlined above.

(e) Upon leaving the area, decontaminate to the extent the situation permits. If bathing facilities and fresh clothing are available, carefully remove contaminated clothing and thoroughly wash the body and protective mask in soap and water prior to removing the mask. Then don fresh clothing. Give special attention to decontamination and treatment of skin lesions.

f. Group protection. The best protection is a pressurized shelter using filtered, forced air. A building or shelter without this feature provides only limited protection from aerosols. Eventually, microorganisms will penetrate through cracks and constitute a respiratory hazard unless the protective mask is worn. As in the case of individual protective measures, utilization of shelters depends upon early warning.

(1) Protection of food and water depends entirely on following good preventive medicine and veterinary procedures (see chapters 20 and 21). Some biological agents cannot be destroyed by normal water purification techniques. When biological agents are known to have been used, all drinking water must be boiled in addition to normal water treatment measures.

(2) Proper hygiene and sanitation procedures must be used (see chapter 20).

(3) Immunizations must be kept current.

g. Pending identification of the agent, measures should be taken to prevent epidemics as soon as possible after initial exposure. These measures include isolation, quarantine, and restriction of personnel movement. After identification of the agent and if it is not capable of producing an epidemic, these restrictive measures can be relaxed.

14-3. CHEMICAL WARFARE. This section deals mainly with the diagnosis and treatment of specific chemical agents.

a. General considerations.

(1) Chemical casualties who have not been decontaminated may endanger unprotected personnel. Handlers of these patients should wear protective masks, impermeable protective gloves, and chemical protective clothing. If conditions permit, an aid station should be established upwind from the contaminated area. The casualties should be undressed and washed thoroughly, downwind of the aid station, before being brought into the aid station.

(2) Most chemical agents can poison food and water. Suspect food and water must be examined by chemical test procedures, if available. If testing equipment is not available, avoid using the water or food, or get an animal to eat or drink a portion and watch it for at least an hour for adverse reactions. Canned foodstuff is completely protected, but the container might be contaminated and should be washed thoroughly with copious amounts of uncontaminated water. Avoid foodstuff that is not well sealed from vapor and liquid agents.

b. Nerve agents.

(1) Nerve agents are among the deadliest chemical agents. They include (GA) tabun, (GB) sarin, (GD) soman, and VX. They are colorless to light brown liquids, some of which are volatile. They are usually odorless, except for GA which has a faint, sweet fruity odor. Toxic liquids are tasteless. They range from nonpersistent to persistent. Nerve agents may be absorbed through the skin, respiratory tract, gastrointestinal tract, and the eyes. However, significant absorption through the skin takes a period of minutes and prompt decontamination is imperative.

(2) Effects of nerve agents.

Site of Action	Signs and Symptoms Following Local Exposure
Pupils	Constricted (miosis), marked, usually maximal (pinpoint), sometimes unequal.
Ciliary body	Frontal headache, eye pain on focusing, slight dimness of vision, occasional nausea and vomiting.
Conjunctivae	Hyperemia.
Nasal mucous membranes	Rhinorrhea, hyperemia.

Bronchial tree	Tightness in chest, sometimes with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, cough.
	Following Systemic Absorption
Bronchial tree	Tightness in chest, with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, dyspnea, slight pain in chest, increased bronchial secretion, cough, pulmonary edema, cyanosis.
Gastrointestinal	Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness (cardiospasm) with "heartburn" and eructation, diarrhea, tenesmus, involuntary defecation.
Sweat glands	Increased sweating.
Salivary glands	Increased salivation.
Lacrimal glands	Increased lacrimation.
Heart	Slight bradycardia.
Pupils	Slight miosis, occasionally unequal, later maximal miosis (pinpoint).
Ciliary body	Blurring of vision.
Bladder	Frequency, involuntary micturition.
Striated muscle	Easy fatigue, mild weakness, muscular twitching, fasciculations, cramps, generalized weakness, including muscles of respiration, with dyspnea and cyanosis.
Sympathetic ganglia	Pallor, occasional elevation of blood pressure.
Central Nervous System	Giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headaches, tremor, withdrawal and depression, drowsiness, difficulty concentrating, slowness on recall, confusion, slurred speech, ataxia, generalized weakness, coma, with absence of reflexes, Cheyne-Stokes respirations, convulsions, depression of respiratory and circulatory centers, with dyspnea cyanosis, and fall in blood pressure.

(a) Nerve agents are cumulative in their effect. Daily exposure to concentrations of a nerve agent insufficient to cause symptoms following a single exposure may result in symptoms following several days of exposure.

(b) Suspect nerve agent poisoning if any of the following occurs:

1. A feeling of tightness or constriction in the chest.

2. Unexplained runny nose.

3. Difficulty in breathing, either on inhaling or exhaling.

4. Small, pinpoint-size pupils seen in a mirror or in the eyes of individuals in the vicinity. (On exposure to vapor or aerosol, the pupils become pinpointed immediately. If the nerve agent is absorbed through the skin only or by ingestion of contaminated food or water, the pinpointing will be delayed or even absent.)

5. A drawing, slightly painful sensation in the eyes or unexplained dimness of vision occurring with pinpoint pupils.

(3) Treatment of nerve agent poisoning.

(a) Immediately don the protective mask and hood at the first indication of any chemical attack.

(b) Immediately remove any liquid contamination. (If a drop or a splash of liquid nerve agent gets in the eyes, immediately irrigate the eyes with copious amounts of water).

(c) Administer 2 mg. of atropine as soon as any local or systemic nerve agent symptoms are noted. (Do not give for preventive purposes before exposure to nerve agent.) If the patient has mild symptoms due to nerve agents, the IM injection of 2 mg. atropine should be repeated at 20-minute intervals, 10-minute intervals if moderate to severe symptoms are present, or until signs of atropinization (dry mouth, blurry near vision) are achieved. A mild degree of atropinization should be maintained for at least 24 hours by IM or oral administration of 1-2 mg. of atropine every 1/2 to 4 hours.

1. Atropine can be given IM, IV, or orally. Atropine given IM requires about 8 minutes before effects are noticed. Given IV, effects begin within 1 minute and reach maximum effect within 6 minutes. Atropine tablets require 20 minutes before effects are felt and 50 minutes before maximum effect takes place.

2. Atropine effects include dryness of the mouth and throat, with slight difficulty in swallowing. Patient may have a feeling of warmth, slight flushing, rapid pulse, some hesitancy of urination, and an occasional desire to belch. Pupils may be dilated slightly but react to light and near vision is blurred. Some individuals may experience mild drowsiness, slowness of memory, and the feeling his body movements are slow. Further doses of 2 mg. of atropine intensify the symptoms and prolong the effects. Effects of one to two 2 mg. injections last 3-5

hours, and the effects of four injections given at close intervals last 6-12 hours.

3. Patients with moderately severe nerve agent symptoms have increased tolerance for atropine, so fairly large doses may be administered before signs of atropinization appear.

(d) Severe nerve agent exposure may rapidly cause unconsciousness, muscular paralysis, and cessation of breathing. If this occurs, artificial respiration is required along with the atropine injections. If the patient is in severe respiratory distress or is convulsing, 4-6 mg. of atropine should be injected IV. If relief does not occur and bronchial secretions and salivation does not decrease, give 2 mg. of atropine q. 3-8 minutes until relief occurs and secretions diminish. In severe nerve agent poisoning the effect of each injection of atropine may be transient, lasting only 3-10 minutes. This requires the patient to be monitored closely and atropine repeated as needed. A mild atropinization should be maintained for at least 48 hours.

(e) Pralidoximine chloride (2-Pam Cl or Protopam Cl) can be used to increase the effectiveness of therapy in nerve agent poisoning. 2-Pam Cl reduces the time during which artificial respiration is required. Dosage: 2-Pam Cl, 1 gm in 100 ml. of sterile water, normal saline, or 5% dextrose and water; IV slowly over 15-30 minutes.

c. Blister agents (vesicants).

(1) Vesicants act on the eyes, lungs, and skin causing burns and blisters. They damage the respiratory tract when inhaled and cause vomiting and diarrhea when absorbed. Most vesicants are insidious in action causing little or no pain at the time of exposure. Lewisite and phosgene oxime cause immediate pain on contact. Vesicants poison food and water and make other supplies dangerous to handle. The severity of a chemical burn is directly related to the concentration of the agent and the duration of contact with the skin.

(2) Mustard (HD). An oily liquid ranging from colorless when pure to dark brown. Mustard is heavier than water, but small droplets float on water surfaces. It is only slightly soluble in water, but freely soluble in fats, oils, gasoline, kerosene, acetone, and alcohol. These solvents do not destroy mustard. Mustard is a persistent agent. It smells like garlic or horseradish. Even very small repeated exposures to mustard are cumulative in effect.

(a) Symptoms.

1. Eye effects. In a single exposure the eye is the most vulnerable. In mild exposure there is a latent period of 4-12 hours followed by tearing and a gritty feeling in the eyes. The conjunctiva and lids become red and edematous. Heavy exposure has a latent period of 1-3 hours followed by severe irritation and lesions. Ischemic necrosis of the conjunctivae, edema, photophobia, and blepharospasm may obstruct vision. Dense corneal opacification with deep ulceration and vascularization may occur.

2. Effects on the skin. Latent period depends on weather conditions. In hot, humid weather latency may be as short as 1 hour; in cool weather after mild vapor exposure, latency may be several

days. Normal latency is 6-12 hours. Initial symptom is erythema, resembling sunburn, followed by multiple pinpoint lesions that enlarge and form the typical blisters. The blisters are usually large, domed, thin walled, superficial, translucent, yellowish, and surrounded by erythema. The blister fluid is clear, thin, and straw colored at first; later it is yellowish and tending to coagulate. Liquid contaminaton of the skin usually results in a ring of vesicles around a gray-white area that does not blister.

3. Respiratory effects. Develop slowly taking several days to reach maximal severity. Symptoms begin with hoarseness (may progress to loss of voice). A cough, which is worse at night, appears early and later becomes productive. Fever, dyspnea, and moist rales may develop into bronchopneumonia.

4. Systemic and gastrointestinal effects. Ingestion of contaminated food or water produces nausea, vomiting, abdominal pain, diarrhea, and prostration. Skin exposure may cause malaise, vomiting, and fever appearing about the same time as the erythema. With severe exposure, symptoms may be so marked as to result in prostration. Severe systemic mustard poisoning may present C.N.S. symptoms such as cerebral depression, bradycardia, and cardiac irregularities.

(b) Treatment of mustard agent.

1. Immediately don protective mask and hood.

2. Immediately remove any liquid contamination. (Speed in decontamination of the eye is absolutely essential. Rinse the eye with copious amounts of water.)

3. After rinsing the eyes, apply a steroid antibiotic eye ointment. Patients with severe photophobia and blepharospasm should have 1 drop of 1% atropic sulfate instilled in the eye t.i.d. The eyes must not be bandaged or the lids allowed to stick together.

4. All blisters should be opened and the fluid drained with care, as the fluid itself may be irritating and cause secondary erythema and blisters. Area should be cleansed with tap water or saline and burn cream applied (10% Sulfamylon burn cream).

5. Respiratory tract injuries are treated symptomatically with steam inhalation.

6. The biggest part of the treatment is symptomatic and preventing or treating secondary infections.

(3) Nitrogen mustard (HN). Oily, colorless, pale yellow liquids; some have a faint fishy odor, while others are odorless.

(a) Effects of HN on the eye. Slight to moderate exposure produces symptoms within 20 minutes that wax and wane until they become persistent about 2 1/2 hours later and reach their maximum in 8-10 hours. Severe exposure causes immediate symptoms that progress for 24 hours. In general the symptoms are the same as mustard, but more severe and requiring intensive and early treatment.

(b) The most specific effect is in the blood and lymph

tissue. .Within 5-10 days after exposure, anemia may develop and W.B.C. can fall to less than 500.

(c) Treatment of HN is generally the same as for mustard, but frequent checks of the hematocrit and W.B.C. are necessary.

(4) Arsenical vesicants. Colorless to brown liquids, soluble in most organic solvents but poorly soluble in water. They are generally more volatile than mustard and have fruity to geraniumlike odors. Vapors are unlikely to cause significant injuries. Liquids will cause severe burns of the skin and eyes and can gradually penetrate rubber and most impermeable fabrics.

(a) Liquid agent symptoms:

1. Effects on the eye include immediate pain and blepharospasm on contact. Edema follows rapidly, causing the eye to close within an hour. Severe exposure can cause permanent injury or blindness.

2. Effects on the skin are more severe than those from liquid mustard. Stinging pain is usually felt 10 to 20 seconds after contact. The pain increases in severity with penetration and in a few minutes becomes a deep aching pain. About 5 minutes after contact a gray area of dead skin appears resembling that seen in corrosive burns. Erythema resembles that caused by mustard, but is accompanied by more pain. Itching and irritation persist for about 24 hours whether or not a blister develops. Blisters are often well developed in 12 hours and are painful at first (mustard blisters are relatively painless). The pain lessens in 48-72 hours.

3. Respiratory effects are similar to those produced by mustard agent. Systemic absorption of arsenicals causes a change in the capillary permeability. This can permit sufficient fluid loss from the blood stream to cause hemoconcentration, shock, and death. Acute systemic poisoning from large skin burns causes pulmonary edema, diarrhea, restlessness, weakness, subnormal temperature, and low blood pressure.

(b) Treatment. Mask and immediately decontaminate any liquid agent (flush contaminated eyes with copious amounts of water). Treatment for the eyes is mainly symptomatic; atropine sulfate opthalmic ointment or atropine drops should be used in conjunction with an opthalmic antibiotic ointment. BAL ointment should be applied to areas of skin contamination BEFORE any blistering appears and remain on the area for at least 5 minutes. (BAL ointment occasionally causes stinging, itching, or urticarial wheals. Frequent application on the same area of skin causes mild dermatitis.) Treatment of blisters is the same as for mustard agents.

(c) Indication for systemic treatment.

1. Cough with dyspnea and frothy sputum, which may be blood tinged, and other signs of pulmonary edema.

2. Skin contamination the size of the palm of the hand or larger in which there is gray or dead-white blanching of the skin or in which erythema develops over the area within 30 minutes.

(d) Two types of treatment may be used.

1. Local neutralization by liberal application of BAL ointment that must remain on the affected area. Remove any other protective ointment before applying BAL ointment.

2. IM injection of dimercaprol (BAL) 10% solution in oil. For mild to moderate poisoning give 2.5 mg./kg. (1.5 ml./60 kg.) q.4h. x 2 days, then one injection q.12h. the third day, fourth to the tenth day give one injection once or twice a day. For severe poisoning give 3 mg./kg. (1.8 ml./60 kg.) q.4h. x 2 days, third day give one injection q.6h., fourth through fourteenth day one injection twice a day. Up to 5 mg./kg. can be given in severe cases.

Symptoms caused by BAL include dryness of the mouth and throat, mild tearing, slight reddening of the eyes, feeling of constriction in the throat, burning sensation of the lips, generalized muscular aching, abdominal pain, mild restlessness and sweating of the hands, apprehension, mild nausea and vomiting on eating, and a transient rise in blood pressure. Symptoms start 15-30 minutes after injection and last about 30 minutes. Unless they are severe or prolonged, they are not a contraindication for continuing therapy.

(5) Phosgene oxime (CX). A powerful irritant that is especially effective as a liquid. It has a disagreeable penetrating odor and is readily soluble in water.

(a) Phosgene oxime is violently irritating to mucous membranes of the eyes and nose. Even low concentrations can cause tearing. Any exposure to liquid or vapor that produces pain will also produce skin necrosis at the site of contact. The area becomes blanched and is surrounded by an erythematous ring within 30 seconds. This is followed by a wheal within 30 minutes. Within 24 hours the original blanched area acquires a brown pigmentation. An eschar forms at about 1 week and sloughs at about 3 weeks. Itching may be present throughout the entire course of healing. Healing may take 2 months or more.

(b) Decontamination is not effective after pain starts, but the contaminated area should be flushed with copious amounts of water to remove any agent that has not yet reacted with the tissue. Treat as any other ulcerated necrotic skin lesion, plus supportive care, as needed.

(6) Mixtures of blister agents. Arsenical vesicants are often mixed with mustard to confuse and make diagnosis difficult. These mixtures do not produce more severe lesions than either agent alone.

d. Choking agents (lung irritants). Best known of these agents is phosgene, a colorless gas with an odor of new mown hay, grass, or green corn. Phosgene is a nonpersistent agent that is broken down rapidly by water (fog, rain, heavy vegetation).

(1) During and immediately after exposure there is likely to be coughing, choking, a feeling of tightness in the chest, nausea, occasionally vomiting, headache, and tearing. There may be an initial slowing of the pulse followed by an increase. These symptoms may not appear, but if they do, a latent period follows that commonly lasts 2-24 hours but may be shorter. Following the latent period, signs and symptoms of pulmonary edema develop. They start with rapid shallow breathing, painful cough, and cyanosis. Nausea and vomiting may appear. As edema progresses, discomfort, apprehension, and dyspnea increase and frothy