

CHAPTER 10

NOXIOUS CHEMICALS

10-1. General

a. Toxic vapors from chemicals (other than those used in military operations) may be encountered, particularly in confined or poorly ventilated spaces. Most of these vapors are thermal decomposition products of chemical elements present in a wide variety of materials.

b. The most widely encountered noxious chemicals are CO, chlorine vapor, oxides of nitrogen, hydrogen sulfide, and ammonia.

c. The field protective mask and collective protectors are of no value against CO or ammonia, and are of only limited value against oxides of nitrogen and hydrogen sulfide. A special type of canister is required for protection against CO and hydrogen sulfide (TB MED 502). Protection against the above vapors may be obtained with a self-contained breathing apparatus.

10-2. Carbon Monoxide

a. *Physical Properties.* Pure CO is a colorless, tasteless, odorless gas. It is lighter than air into which it diffuses rapidly.

b. *Occurrence in Military Operations.* Carbon monoxide is formed by gun blasts, bursting shells, internal combustion engines, fires in confined spaces, and the incomplete combustion of fuels.

c. *Pathology.* Asphyxiation is caused by the inactivation of blood hemoglobin through a combination with CO. The resultant anoxia may produce nervous system changes. Postmortem examinations reveal little beyond the characteristic cherry red color of the blood and hemorrhages in the brain.

d. *Symptoms.* Carbon monoxide is very insidious in its action and poisoning may occur without appreciable initial signs. The symptoms progress from throbbing headaches, vertigo, yawning, and poor visual acuity, to the development of cherry red mucous membranes, weakness and coma, subnormal temperature, feeble pulse, and death.

e. *Diagnosis.* The diagnosis is made from the circumstances of exposure and the appearance of cherry red skin and mucous membranes color.

f. *Protection.* In general, exposure to CO should be avoided whenever possible. Adequate ventilation should be provided for all enclosed spaces where CO may be produced. The safety of air in the space for people to breathe may be tested by standard CO

indicator or detector devices. Individuals required to enter closed areas where high concentrations of CO are (known or suspected to be) present must be provided with respiratory protective devices. For the approved devices, refer to TB MED 502.

g. *Treatment.* Remove the victim to pure air. If respirations are weak or absent, begin assisted ventilation at once. Oxygen, if available, should be given by a face mask, preferably under pressure (up to 3 atmospheres). The patient should be kept warm and at rest (sedated, if necessary). After resuscitation, initial supportive measures (such as the need for parenteral fluids and pressor drugs) can best be decided by the medical officer. Ordinarily, methylene blue solution, morphine, and atropine should NOT be used (TB MED 269).

h. *Prognosis.* The longer the period of the coma, the less the chance for recovery. Most mildly exposed individuals recover with early treatment. Tachycardia and dyspnea may continue for months. There may be CNS disturbances ranging from simple neuritis to mental deterioration.

10-3. Oxides of Nitrogen

a. *Physical Properties.* The term "oxides of nitrogen" applies to a mixture consisting of nitric oxide, nitrogen dioxide, and nitrogen tetroxide. Nitric oxide is colorless. The other oxides are red-brown gases.

b. *Occurrence in Military Operations.*

(1) The danger of nitrous fume poisoning is great if high explosives (such as smokeless powder or cordite) are burned or detonated in poorly ventilation areas. This may occur in gun pits, armored vehicles, ship magazines, and turrets. This may also occur in mining and tunneling operations.

(2) In addition, nitrous fumes are emitted from fuming nitric acids (white and red) and are generated by the combustion of certain plastics.

c. *Pathology.* Inhalation of nitric oxide causes the formation of methemoglobin and does not appear to lead to any tissue lesions. Inhalation of nitrogen dioxide results in the formation of nitrite that leads to a fall in blood pressure and to the production of methemoglobin. Inhalation of high concentrations of nitrogen dioxide (above 0.5 mg per liter) causes rapid death without the formation of pulmonary edema. Somewhat lower concentrations result in death with

the production of yellow, frothy fluid in the nasal passages, mouth, and trachea and marked pulmonary edema. The findings in other tissues are negligible.

d. Symptoms. The symptoms following inhalation of nitrous fumes are due chiefly to nitrogen dioxide. The symptoms presented depend upon the concentration of the gas. Exposures to higher concentrations cause severe local irritation with choking and burning in the chest, violent coughing, yellow staining of the mucous membranes, expectoration of yellow-colored sputum, headache, and vomiting. Often, these early symptoms may be mild or entirely absent. After 2 to 24 hours, symptoms start with coughing, nausea, vomiting, frothy sputum, dyspnea, cyanosis, convulsions, and signs of lung edema. This train of symptoms may result in death. At exposures to very high concentrations for short periods of time, the onset of symptoms is very sudden and marked. Convulsions, unconsciousness, and respiratory arrest occur within a short time and death may follow rapidly.

e. Diagnosis. The diagnosis is made from the history, symptoms described, and sometimes the pungent odor of the gas or the yellow discoloration of the exposed mucous membranes.

f. Treatment. Treatment of casualties with symptoms of pulmonary irritation is the same as for CG poisoning (chap 5).

g. Prognosis. The few cases with symptoms referable to the CNS either die quickly or, on removal to fresh air, recover spontaneously. Fatal cases usually die within 48 hours. Bronchopneumonia and varying degrees of pulmonary fibrosis and emphysema often follow recovery from the acute stage.

10-4. Hydrogen Sulfide

a. Physical Properties. This colorless gas in low concentrations has the odor of rotten eggs. In high concentrations it may dull the sense of smell and be difficult to recognize.

b. Occurrence in Military Operations. This gas is produced during the decomposition of sulfur-containing compounds in sewers, waste, coal bins or stacks, holds of ships, and waterfront excavations.

c. Pathology. In low concentrations (less than 0.15 mg per liter), hydrogen sulfide may produce inflammation of the eyes, nose, and throat if breathed for periods of 1/2 to 1 hour. Higher concentrations (0.75 mg per liter or greater) are rapidly fatal, presumably by combination of the hydrogen sulfide with the respiratory tissue pigments and the subsequent paralysis of the respiratory center.

d. Symptoms. The symptoms depend upon the concentration of the gas. At the lowest concentrations, the effects are chiefly on the eyes; that is, conjunctivitis, swollen eyelids, itchiness, smarting, pain, photophobia, and blurring of vision. At higher concentrations, respiratory tract symptoms are more

pronounced. Rhinitis, pharyngitis, laryngitis, and bronchitis may occur. Pulmonary edema may result. At very high concentrations, unconsciousness, convulsions, and cessation of respiration rapidly develop.

e. Treatment. Immediately remove the casualty from the contaminated atmosphere and administer assisted ventilation with oxygen, if possible. Treatment of pulmonary edema is the same as for that caused by CG (chap 5).

10-5. Ammonia

a. Physical Properties. Ammonia is a colorless gas, soluble in water, with a pungent odor. Liquid ammonia is a vesicant.

b. Occurrence in Military Operations. This gas has not been used in warfare but may be encountered in industrial accidents, bombings involving refrigeration plants, and holds of ships as a product of decomposing material.

c. Pathology. Exposure to high concentrations of ammonia produces prompt and violent irritation of the eyes and respiratory tract. There may be spasm and edema of the glottis or necrosis of the laryngeal mucous membranes. Pulmonary edema may develop and may be complicated by bronchopneumonia.

d. Symptoms. High concentrations produce violent, burning pain in the eyes and nose, lacrimation, sneezing, pain in the chest, cough, spasm of the glottis, and pulmonary edema. Often there is a temporary reflex cessation of respiration with spasm of the glottis. Edema of the glottis at a later period may seriously interfere with breathing. Concentrations of 0.1 percent are intolerable to humans.

e. Treatment. Treatment consists of prompt removal to pure air and administration of assisted ventilation. Later measures are directed toward the treatment of pulmonary edema, bronchitis, and pneumonia.

f. Prognosis. The mortality is high following severe exposure. With low concentrations, recovery is usually rapid although bronchitis may persist.

10-6. Hazards Caused by Fire

a. Fire produced by explosions, incendiaries, or other causes creates extreme problems. In fires, death may be caused not only by blast and direct flame but by anoxia, CO, heat, nitrous fumes, toxic fumes from burning plastics, and smoke. An oxygen rescue breathing apparatus must be used for protection in these circumstances.

b. The filter element of the field protective mask (which is designed to protect the wearer's respiratory system against the effects of chemical agents) provides only limited protection against smoke. Duration of the protection depends upon the type of smoke and its concentration. The filter element (or canister) does not generate oxygen but filters smoke and many agents

out of the air as they pass through it. Therefore, the field protective mask should not be used in air containing less than 16 percent oxygen or more than 3 percent CO, or in air having heavy concentrations of smoke from oil fires. In each case that smoke

penetrates the protective mask, a new filter element (or canister) should be provided before continuing further use of the mask. Some chemicals such as CO and ammonia are not filtered by the field protective mask.