Chapter 4

LUNG-DAMAGING AGENTS AND TOXIC INDUSTRIAL CHEMICALS

Summary

NATO Codes: CG, Cl

Signs and Symptoms*: Central effects:* Eye and airway irritation, dyspnea. *Peripheral effects:* Chest tightness and delayed pulmonary edema.

Field Detection: Joint Chemical Agent Detector (JCAD). The M18A2 Chemical Agent Detector Kit and the M93 series Fox Reconnaissance System will detect small concentrations of CG; however, they will not detect Cl.

Decontamination: *Vapor:* fresh air; *liquid:* copious water irrigation.

Management: Termination of exposure, ABCs of resuscitation (airway, breathing, circulation), enforced rest and observation, oxygen with or without positive airway pressure for signs of respiratory distress, other supportive therapy as needed.

Overview

Over 1,800 toxic industrial chemicals (TICs) are used in industry, stored at industrial sites, and transported on the world's road and rail systems. Some of these chemicals were deployed as chemical warfare agents during the First World War, killing and injuring thousands, and can have the same deadly consequences today if released during an accident or through terrorist sabotage. Death

from exposure to TICs is more frequent when they are inhaled. Inhaling a TIC in the form of a gas, vapor (gas coming from a liquid), or aerosol (liquid or solid particles suspended in a gas) can cause a sudden closure of the larynx (laryngospasm), causing the victim to choke and collapse. TICs can also cause damage to the tissues of the upper airways, resulting in swelling, scarring, and airway narrowing, which can restrict breathing. TICs can damage lung tissues, allowing body plasma and other fluids to leak into the lung air sacs (alveoli), causing pulmonary edema and death from asphyxiation.

Exposure to TIC lung-damaging agents can occur on and off the battlefield. The care provider must know how to identify the signs and symptoms and provide appropriate lifesaving support to those exposed to these agents.

Understanding the Respiratory System

The respiratory system can be divided into two compartments. Understanding these compartments can greatly simplify the treatment problem-solving process (Figure 4-1).

The central airway compartment includes the nasopharynx (nose), oropharynx (mouth), larynx (vocal cords), and the trachea and bronchi (airway from the throat into the lungs). Tissues in this area are very moist and thin and can be damaged by TICs.

The peripheral lung compartment includes the lung sacs (alveoli) distributed throughout the lung tissue. During normal respiration, inhaled gases fill the alveoli and then move slowly through their walls. The gases then move through the thin walls of the blood vessels (capillaries) surrounding the alveoli and into the blood. TICs can damage the walls of alveoli and the capillaries surrounding them, allowing blood plasma and cells to leak into the air space of the alveoli.

Types of Lung-Damaging Agents

TICs are numerous. Those that pose a frequent threat to the soldier in the field are listed here. Though the list is not complete, casualties from other lung-damaging agents are managed the same way as in these examples. In low doses, highly reactive TICs

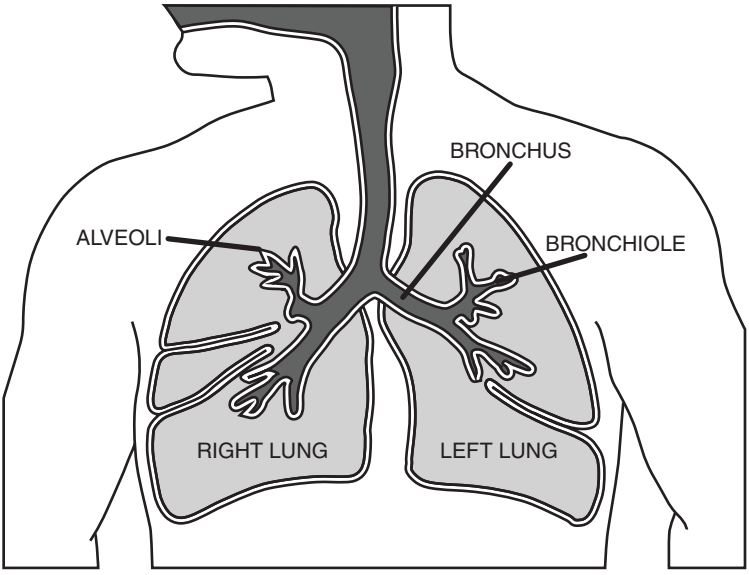

Figure 4-1. Central airway (dark gray) and peripheral airway (light gray).

have a greater effect on the central airway compartment; other TICs act on both airway compartments; and still others that are not as reactive in the central airway compartment travel deeper in the respiratory tract and destroy the tissues of the alveoli in the peripheral lung compartment. Any TIC inhaled in large doses will cause damage to both central airway and peripheral lung compartments.

Centrally Acting TICs

Ammonia is a highly caustic and reactive gas used for industrial refrigeration, cleaning, processing of some illicit drugs, and numerous legitimate industrial processes. It is a good example of a TIC that, in low doses, is primarily centrally acting. It rapidly forms a strong base (alkali) when it contacts the moist tissues of the central airway compartment. The alkali burns and destroys the tissues it contacts. The victim may suddenly go into

laryngospasm and collapse. The tissues of the compartment will also become swollen. Scar tissue may form along the airway. Frequently, damaged tissue in the airway will die and slough off, obstructing the airway.

Sulfur mustard (HD) is an example of a chemical agent produced solely for warfare that acts on the central airway compartment if inhaled. HD will cause tissue to slough off in large sheets, known as pseudomembranes, which block the airway. The various degrees of airway obstruction may cause sneezing and coughing, hoarseness when talking, wheezing noises when breathing, or inability to breathe.

Peripherally Acting TICs

Phosgene is a major industrial chemical used in many manufacturing processes. More importantly, it is released from heating or burning many common chemicals or solvents. Carbon tetrachloride, perchloroethylene (a degreasing compound), methylene chloride (used in paint removal), and many other compounds break down to phosgene with flame or heat. Also, common substances such as foam plastics release phosgene when they burn. A soldier presenting with shortness of breath in the absence of a chemical attack or other obvious cause should be carefully questioned about whether he or she has been near any burning substances or chemical vapors near flame or other hot materials (eg, a heater with open coils).

Perfluoroisobutylene (PFIB) is given off when Teflon (DuPont, Wilmington, DE) burns at high temperatures, such as in a vehicle fire. Teflon is used to line the interior of many military vehicles, particularly armored vehicles and aircraft. Closed-space fires in these vehicles release PFIB. Survivors of vehicle fires who are short of breath should be questioned about their exposure to the smoke.

Oxides of nitrogen, or NOx, are components of photochemical smog that can be produced by burning gunpowder or industrial waste. These substances can build up to high concentrations where artillery is fired and there is inadequate ventilation. Soldiers who become short of breath after heavy firing should be suspected of exposure to this lung-damaging agent.

HC smoke is a mixture of equal amounts of hexachloroethane, zinc oxide, and approximately 7% grained aluminum or aluminum powder used in the military for obscuration. Zinc oxide can cause lung damage if inhaled in toxic amounts. Appropriate precautions, such as wearing protective masks, must be taken when HC smoke is used.

TICs That Act Both Centrally and Peripherally

Chlorine is a good example of a combination agent, one that acts on both airway compartments in low doses. It is widely used in industry for manufacturing plastics and lubricants and purifying water. It was the first chemical agent used effectively on World War I battlefields against unprotected military troops. Its effectiveness as a weapon was greatly reduced once protective masks were widely available for wear on the battlefield. Chlorine turns to hydrochloric acid when it contacts the moisture of the airway; it then causes chemical burns to the tissue. It produces signs and symptoms seen with exposure to both central and peripherally acting agents. Its action is a reminder that even though central compartment damage may seem like the primary concern in some patients (when they are coughing and wheezing), the medic must always treat casualties as if they could develop peripheral compartment symptoms, and take seriously any patient complaints about chest tightness or breathing difficulty.

Detection

There are no specific field detection devices for chlorine and ammonia. However, each has a distinctive odor: chlorine has an acrid or unpleasantly pungent odor, and ammonia has a pungent odor. Phosgene smells like newly cut grass, newly mown hay, or green corn. Unfortunately, odor is not a reliable detection method (if you can smell these gases, you are being exposed to their toxic inhalation effects).

Protection

Both the military protective mask fitted with a C2A1 filter canister, and the Joint Service General Purpose Mask with M61 filters, will protect against chlorine, phosgene, PFIB, NOx, and HC smoke in the open battlefield. Specific filters, or the use of a self-contained breathing apparatus, are mandated for other TICs, such as ammonia. Masks do not protect against carbon dioxide. Masks will not be effective in environments where the TIC displaces oxygen, creating a low oxygen environment (at or below 19.5% fraction of expired oxygen [FiO_2]). Masks should be used in these environments for escape purpose only. Using a self-contained breathing apparatus is recommended.

Physical Properties

Lung-damaging TICs are typically heavier than air and hang close to the ground when released. They tend to evaporate and disperse very quickly depending on temperature and wind conditions. If the TIC is in liquid form at room temperature, it will tend to give off a vapor. Vapors can become trapped in clothing fibers and "off-gas" to affect nearby individuals who have no respiratory protection. Although skin decontamination after vapor exposure is not a high priority, clothing should be removed and the underlying skin decontaminated with soap and water.

Mechanism of Action

Central Airway Compartment

Centrally acting TICs, such as ammonia and HD, will form strong acids or bases (alkali) with the water in the tissues of the central airway and then destroy these tissues. Damaged tissues will swell and can slough into the airway, obstructing breathing.

Peripheral Airway Compartment

Phosgene is the most studied peripherally acting agent. It causes pulmonary edema, which is life threatening. Less is known about the other compounds; however, they are believed to be very

similar. Phosgene causes effects in the lung by inhalation only. It does not cause lung effects when absorbed through the skin, injected, or orally ingested.

When inhaled, phosgene travels to the very end of the smallest airways, the bronchioles, and causes damage to these airways. Additionally, it causes damage to the thin membrane that separates the smallest blood vessels (capillaries) and the air sacs (alveoli), the alveolar-capillary membrane. Phosgene reacts with proteins and enzymes in the alveolar-capillary membranes to cause damage to the membranes. These membranes usually function to separate the blood in the capillaries from the air in the alveoli, but when the membranes are damaged, they cannot perform this function. Blood, or at least the liquid part of the blood (plasma), can leak through the damaged membrane into the alveoli. When the plasma leaks into the alveoli, the air sacs become full of fluid, and air cannot enter them. Therefore, exchange of oxygen from the air into the blood is hindered, and the casualty suffers oxygen deprivation. The extent of oxygen deprivation depends on the extent of the phosgene exposure and the number of alveoli filled with plasma. This is similar to what happens in drowning, in that the alveoli fill up with fluid. However, in this instance, it is fluid from the blood, not from an external source. For this reason, phosgene poisoning is sometimes referred to as "dry land drowning."

Clinical Effects

Centrally Acting Agents

Immediately or shortly after exposure to these gases or vapors, the individual may develop laryngospasm (laryngospasm does not occur in all exposures). As the airway compartments are irritated and damaged, the individual will sneeze, will feel pain in the nose (nasopharynx inflammation), and may develop painful swallowing (oropharynx inflammation), hoarseness, a feeling of choking, noise with inhalation or exhalation (larynx inflammation), pain in the chest, coughing, and wheezing during breathing (trachea and bronchi inflammation). If the exposure has been enough to cause the TIC to reach the peripheral airway

compartment, effects in the peripheral compartment may follow. Scarring of the central airway compartment can create permanent airway narrowing, depending on the agent involved and the dose received.

Peripherally Acting Agents

Very shortly after exposure to phosgene or other agents affecting the peripheral airway, the casualty will typically have an asymptomatic period of 30 minutes to 72 hours, but most significant exposures involve a latent period of less than 24 hours. The duration and concentration of the exposure will determine the time to symptom onset. The casualty may notice irritation of the eyes, nose, and throat, but more commonly there will be no effects during or immediately after exposure. The major effects from phosgene exposure (and the other compounds), like the effects from mustard, do not occur until hours later.

The casualty will notice shortness of breath (dyspnea) between 2 and 24 hours after exposure. Initially, this may be mild, and the dyspnea's eventual severity will depend on the amount of exposure. As the damage progresses, dyspnea will become more severe, and soon a cough will develop. If the damage is severe, the casualty will start coughing up clear, foamy sputum (blood plasma that has leaked into the alveoli).

With a very mild exposure to phosgene (or another of these compounds), casualties will develop dyspnea 6 to 24 hours after exposure. They will notice it first after heavy exertion; however, later they will become short of breath after any activity. With proper care, these casualties will do well and recover completely.

A casualty with a severe exposure to phosgene (or another of these compounds) will notice shortness of breath within 4 to 6 hours after exposure. Increased difficulty breathing, even at rest, will occur, and despite intensive pulmonary care, the casualty may not survive.

The average casualty exposed to a lung-damaging agent will be in between these two extreme cases. When the onset of dyspnea is more than 6 hours after exposure, there may be

progression to dyspnea at rest. However, with good pulmonary care beginning early after the onset of effects, the casualty should recover completely.

Field Care

The care provider should be alert to the possibility that patients can be exposed to lung-damaging agents even when battlefield agents are not being used. Exposure to hazardous, lung-damaging TICs is a likely possibility from industrial sabotage, from exposure to the smokes from burning vehicles, and during common military operations.

A casualty who complains of shortness of breath should be questioned extensively about exposure to smoke from burning Teflon, gunpowder, or industrial chemicals. The most important things to do for such casualties are to ensure they are free from contamination (out of the smoke or wearing a mask) and are kept completely at rest (preferably placed on a litter so they do not walk). Even small exertions can greatly intensify the effects of these agents and speed the progress of pulmonary edema. A casualty who is short of breath may require oxygen, assistance with a continuous positive airway pressure device, or intubation and mechanical ventilation with oxygen.

Those suspected of exposure to a lung-damaging TIC should be observed for 24 to 36 hours, even if they do not have immediate difficulty breathing. Those with a complaint of chest tightness should immediately be made to rest. A dyspneic casualty must be evacuated as quickly as possible to a medical facility that can provide intensive pulmonary care, because the patient's condition can rapidly deteriorate once the lungs begin to fill with fluid. Survival is less likely for a casualty who becomes dyspneic within the first 4 hours after exposure, because pulmonary edema is already rapidly occurring. The casualty will certainly not survive without proper pulmonary care. A casualty who first experiences breathing difficulty more than 4 hours after exposure has a good chance of survival if appropriate medical care is provided.