

CHAPTER 5

LUNG-DAMAGING AGENTS (CHOKING AGENTS)

5-1. General

a. Chemical agents which attack lung tissue, primarily causing pulmonary edema, are classified as lung-damaging agents (choking agents). They include phosgene (CG), diphosgene (DP), chlorine, and chloropicrin (PS). Best known of these agents is CG. Agents in this class are called lung-damaging agents because irritation of the bronchi, trachea, larynx, pharynx, and nose may occur and, with pulmonary edema, contribute to the sensation of choking. Blister agents and certain systemic agents also may injure the respiratory tract. Since the action of CG is typical of the lung-damaging agents, it is used as the example in this chapter.

b. Persons exposed to CG need not be withdrawn during combat, unless signs of pulmonary distress appear. The physician at the supporting MTF should so advise the responsible commander.

5-2. Protection

The protective mask or a gas-particulate filter unit (collective protector) gives protection against lung-damaging agents.

5-3. Properties of CG

At ordinary temperatures and atmospheric pressure, CG is a colorless gas. The boiling point of CG is 47 °F (8.2 °C); it is extremely volatile making it a nonpersistent chemical agent. The vapor density of CG is 3.4 times that of air. Phosgene may remain for long periods of time in trenches and other low-lying areas. In low concentrations, CG has a smell resembling new mown hay. Phosgene is readily soluble in organic solvents and fatty oils. In water, CG is rapidly hydrolyzed with the formation of hydrochloric acid and carbon dioxide.

5-4. Pathology

Aside from mild conjunctival irritation, the direct effects of exposure to CG are confined to the lungs. Changes in other organs are secondary to the pulmonary alterations. The outstanding feature of CG poisoning is massive pulmonary edema. This is preceded by damage to the bronchiolar epitheliums, development of patchy areas of emphysema, partial atelectasis, and edema of the perivascular connective tissue. The trachea and large bronchi are usually normal in appearance. This contrasts with the findings

in chlorine and PS poisoning in which both structures may show serious damage to the epithelial lining with desquamation. The lungs are large, edematous, and darkly congested. Edema fluid (usually frothy) pours from the bronchi and may be seen escaping from the mouth and nostrils. With exposure to very high concentrations, death may occur within several hours. In most fatal cases, pulmonary edema reaches a maximum in 12 hours, followed by death in 24 to 48 hours. If the casualty survives, resolution commences within 48 hours, and in the absence of complicating infection, there may be little or no residual damage.

5-5. Symptoms

During and immediately after exposure, there is likely to be coughing, choking, a feeling of tightness in the chest, nausea, and occasionally vomiting, headache, and lacrimation. The presence or absence of these symptoms is of little value in immediate prognosis. Some patients with severe coughs fail to develop serious lung injury, while others with little sign of early respiratory tract irritation develop fatal pulmonary edema. There may be an initial slowing of the pulse, followed by an increase in rate. A period follows during which abnormal chest signs are absent and the patient may be symptom-free. This interval commonly lasts 2 to 24 hours but may be shorter. It is terminated by the signs and symptoms of pulmonary edema. These begin with cough (occasionally substernally painful), dyspnea, rapid shallow breathing, and cyanosis. Nausea and vomiting may appear. As the edema progresses, discomfort, apprehension, and dyspnea increase and frothy sputum develops. Rales and rhonchi are audible over the chest, and breath sounds are diminished. The patient may develop shock-like symptoms, with pale, clammy skin, low blood pressure, and a feeble, rapid heartbeat.

5-6. Diagnosis

Irritation of the nose and throat by CG may be mistaken for upper respiratory tract infection. Difficulty in breathing and complaint of tightness of the chest may suggest nerve agent poisoning or an acute asthmatic attack. Noncardiac pulmonary edema is like that produced by other agents and may be confused with the edema associated with heart failure. Diagnosis can only be established with certainty from a definite history of exposure to CG.

5-7. Prognosis

Prognosis during the acute phase should be guarded because of the insidious nature of the poisoning. Most deaths occur within the first 48 hours. The few which occur later are due largely to bronchopneumonia. Casualties from CG who survive more than 48 hours usually recover without sequelae. Exposure to CG rarely results in development of chronic bronchitis and bronchiectasis. Long-term pulmonary effects are generally the result of intercurrent infection or other exposures. The majority of U.S. Army deaths from CW agents used in World War I was caused by CG.

5-8. Self-Aid

a. The protective mask should be put on immediately when any of the conditions described in paragraph 1-7 a exist. Other indications of a CG attack are—

(1) Odor like newly mown hay. (Do not rely upon odor as an indication of a chemical attack.)

(2) Irritation of the eyes.

b. If some CG has been inhaled, normal combat duties should be continued unless there is difficulty in breathing, nausea and vomiting, or more than the usual shortness of breath during exertion.

c. If any of the above symptoms occur, there should be quiet rest until medical evacuation is accomplished.

5-9. Treatment

a. *Rest and Warmth.* A casualty exposed to a lung-damaging agent should be kept at rest until the danger of pulmonary edema is past, but the operational situation may prevent this. Tightness of the chest and coughing should be treated with immediate rest and comfortable warmth. The casualty should be evacuated in a semiseated position if dyspnea or orthopnea make a supine posture impractical. Mandatory evacuation by litter in cases of significant respiratory involvement has been advocated.

b. *Sedation.* Sedation should be used sparingly. Codeine in doses of 30 to 60 mg may be effective for cough. Restlessness may be a manifestation of hypoxia; therefore, only cautious use of sedatives is advised. Use of sedatives should be withheld until adequate oxygenation is assured and facilities for possible respiratory assistance are available. Barbiturates, atropine, analeptics, and antihistamines are all contraindicated.

c. *Oxygen.* Hypoxemia may be controlled by oxygen supplementation. Early administration of positive airway pressure (intermittent positive pressure breathing (IPPB), positive end-expiratory pressure (PEEP) mask ("PEEP mask"), or, if necessary, incubation with or without a ventilator) may delay and/or minimize the pulmonary edema and reduce the degree of hypoxemia.

d. *Antibiotics.* Antimicrobial therapy should be reserved for acquired bacterial bronchitis/pneumonitis. Prophylactic therapy is not indicated.

e. *Steroids.* After exposure to a sufficiently high dose of CG or similar agent, pulmonary edema will follow. Administration of corticosteroids has been recommended, but proof of their beneficial effects is lacking. It has been suggested that, when steroid treatment is initiated within a very short time of the exposure, this therapy may lessen the severity of the edema. Rest, warmth, sedation, and oxygen are also of great importance, as indicated above. Doses of steroids used are much greater than those used in asthma. Treatment for exposure to a lung-damaging agent or similar compound (except for zinc chloride smoke, for which an extended regimen is essential) should be judged on the basis of precautionary treatment for what seems a mild but possibly dangerous exposure, and definitive treatment for an exposure which is definitely expected to endanger life. Two regimes are in use: one using dexamethasone-sodium phosphate and the other using beclomethasone dipropionate or betamethasone valerate. In either case, treatment should be started as soon as possible, ideally within 15 minutes of exposure.

(1) Using dexamethasone-sodium phosphate, the procedure is as follows:

(a) Treatment should start at the earliest possible moment with the inhalation of the steroid from an inhaler. Four puffs (or strokes) should be inhaled at once. This should be followed by 1 puff every 3 minutes until any sense of irritation is overcome. After this, 5 puffs should be inhaled every 15 minutes until a total of 150 puffs have been inhaled (that is, the entire contents of one standard inhaler are finished). When this stage has been reached, 1 puff should be inhaled hourly during the day, and, as a preparation for sleep, 5 puffs should be inhaled every 15 minutes until a total of 30 puffs have been reached. This regimen should be repeated each day for at least 5 days or longer if there are any abnormalities, including indications of pulmonary edema or infiltrates on the chest x-ray, after which treatment may be withdrawn. If recovery is slow, the dosage may be reduced to 6 puffs a day and continued until recovery is complete.

(b) Definitive treatment for exposures which definitely endanger life should include the above regime of inhaled steroid treatment, supplemented by systemic steroids as follows:

Day 1	1,000 mg prednisolone, IV
Day 2 and 3	800 mg prednisolone, IV
Day 4 and 5	700 mg prednisolone, IV

Beginning with day 6, the dose of systemic steroids should be reduced as soon as possible, provided that the chest x-ray remains clear. If further early systematic treatment is necessary, adrenaline may be

given in the acute stage of bronchial spasm and oxygen may be necessary. Treatment of severe cases is very difficult because of tissue damage. Absolute rest and administration of oxygen are fundamental. Expectorants may also be used. Bronchopneumonia is treated by antibiotics.

(2) Using beclomethasone dipropionate or betamethasone dipropionate, the procedure is as follows: (The differences occur due to the various absorption characteristics of these steroids. Limited systemic therapy is necessary, even for precautionary treatment.)

(a) Treatment should commence as soon as possible with the inhalation of 10 puffs of the steroid from an inhaler. Five puffs should be given each hour for the next 10 hours. Then 1 puff should be given hourly for the next 24 hours for as long as inhalational therapy is considered necessary (at least 5 days). Systemic therapy is needed even for precautionary treatment during the first 24 hours and should commence as soon as possible with the IV injection of 20 mg of betamethasone or the equivalent dose of another systemic steroid. This dose should be repeated IV or IM every 6 hours for at least the first 24 hours. During the next 5 days, inhalation therapy should be continued but systemic therapy may be

reduced based on clinical response and chest x-ray improvement.

(b) Definitive treatment may call for longer periods of systemic therapy.

(c) Prednisolone, betamethasone, or methylprednisolone are preferred to other steroids for systemic use, as there is evidence that these steroids do not interfere with collagen metabolism. Antibiotic coverage should be considered with these high doses of steroids in patients predisposed to pulmonary infection complications. Side effects of high steroid dosages should be accepted provided they do not themselves endanger life. Any indication of pulmonary fibrosis will necessitate antifibrotic treatment.

5-10. Convalescent Care

Absolute rest must be continued until the acute symptoms have disappeared. Individuals must be closely monitored for signs of recovering from the acute effects of the CG poisoning. When the acute symptoms disappear, individuals should be—

a. Gotten out of bed.

b. Hospitalized as short a time as possible.

c. Encouraged and trained to resume physical exertion to minimize neurasthenic symptoms which may be a major disabling feature in these patients.