

CHAPTER 4

LUNG DAMAGING AGENTS (CHOKING AGENTS)

SECTION I - GENERAL

401. Introduction.*a. Definition.*

(1) Chemical agents which attack lung tissue, primarily causing pulmonary oedema, are classed as lung damaging agents. To this group belong phosgene (CG), diphosgene (DP), chlorine (Cl) and chloropicrin (PS). Certain other substances, while, not likely to be used as agents, are still likely to be met with on the battlefield (e.g., nitrous fumes and zinc chloride smoke in an undeliquesced state) and may have a similar action.

(2) Similar substances encountered in fires, e.g., perfluoroisobutylene (PFIB) and HCl may also induce lung damage.

b. Phosgene. The toxic action of phosgene is typical of a certain group of lung damaging agents. Phosgene is the most dangerous member of this group and the only one considered likely to be used in the future. Phosgene was used for the first time in 1915, and it accounted for 80% of all chemical fatalities during World War I.

SECTION II - PHOSGENE

402. Physical and Chemical Properties.

Phosgene is a colorless gas under ordinary conditions of temperature and pressure. Its boiling point is 8.2°C, making it an extremely volatile and non-persistent agent. Its vapour density is 3.4 times that of air. It may therefore remain for long periods of time in trenches and other low lying areas. In low concentrations it has a smell resembling new mown hay. Phosgene is readily soluble in organic solvents and fatty oils. In water, phosgene is rapidly hydrolyses with the formation of hydrochloric acid and carbon dioxide. Its physical properties are shown in Table 4-I.

403. Detection.

There are no automatic detectors available for use in the field.

404. Protection.

The respirator gives adequate protection against this agent.

Table 4-1. Physical Properties of Lung Damaging Agents

Property	Phosgene (CG)		Diphosgene (DP)	
Appearance	Colourless gas		Colourless gas	
Chemical formula	COCl ₂		ClCOOCCl ₃	
Molecular weight	98.2		197.9	
Density (g.cm ⁻³)	1.38	(20°C)	1.65	(20°C)
Melting point	-128.0°C		-57°C	
Boiling point	7.6°C		127°C	
Vapour density	3.4		6.8	
Vapour pressure (mmhg)	365	(-10°C)	1.0	(0°C)
	555	(0°C)	4.2	(20°C)
	1173	(20°C)		
Volatility (mg.m ⁻³)	528,000	(-40°C)	12,000	(0°C)
	2,200,000	(-10°C)	45,000	(20°C)
	4,300,000	(7.6°C)	270,000	(52°C)

405. Decontamination.

Because of its physical and chemical properties, the agent will not remain in its liquid form for long, and decontamination is not required except when it is used in very cold climates.

406. Mechanism of Action.

- a. The mode of action is still not fully understood. It has been suggested that phosgene may act by inhibiting enzymes, or by producing HCl in the alveoli. It has more recently been suggested that phosgene, which is itself a highly reactive molecule, may react directly and instantaneously at the alveolar and capillary wall, permitting plasma to flood the alveoli. Its effects usually reach a maximum 12-24 hours after exposure.
- b. Whatever the mechanism of action, phosgene increases the permeability of the alveolar capillaries with resultant pulmonary oedema. This interferes with pulmonary gaseous exchange, leading to hypoxia. The loss of fluid into the alveoli also results in haemoconcentration which, together with hypoxia, causes cardiac embarrassment which may proceed to cardiac failure.

407. Clinical-Pathological Effects.

The outstanding feature of phosgene poisoning is massive pulmonary oedema. This is preceded by damage to the bronchiolar epitheliums, development of patchy areas of emphysema, partial atelectasis, and oedema of the perivascular connective tissue. The trachea and bronchi are usually normal in appearance. This contrasts with the findings in chlorine and chloropicrin poisoning in which both structures may show serious damage to the epithelial lining with desquamation. The lungs are large, oedematous and darkly congested. Oedema 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 oedema reaches a maximum in 12 hours followed by death in 24-48 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.

408. Signs and 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 lachrymation. 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 oedema. There maybe 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 oedema. These begin with cough (occasionally substernally painful), dyspnoea, rapid shallow breathing and cyanosis. Nausea and vomiting may appear. As the oedema progresses, discomfort, apprehension and dyspnoea 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 feeble, rapid heartbeat.

409. Treatment.

- a. *Rest and Warmth.* It is desirable that a casualty exposed to a lung-damaging agent be kept at rest until the danger of pulmonary oedema 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 semi-seated position if dyspnoea or orthopnoea 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.* Hypoxaemia 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 minimise the pulmonary oedema and reduce the degree of hypoxaemia.
- 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 phosgene or similar agent, pulmonary oedema 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 oedema. Two inhalational regimes are in use: one using dexamethasone and the other using betamethasone or beclomethasone. In either case, treatment should be started as soon as possible, ideally within 15 minutes of exposure. Doses of steroids used are much greater than those prescribed in asthma and when steroids are used they should be given in high doses by inhalation and in severe cases by injection.
- f. *Other.* Rest, warmth, sedation and oxygen are of great importance, as indicated above. 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:
 - (1) Precautionary treatment for what seems a mild but possibly dangerous exposure; and
 - (2) Definitive treatment for an exposure which is definitely expected to endanger life.

410. Course and Prognosis.

During the acute phase, casualties may have minimal signs and symptoms and the prognosis should be guarded. Casualties may very rapidly develop severe pulmonary oedema. If casualties survive more than 48 hours they usually recover without sequelae.