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Medical Manual of Defence against Chemical Agents

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Medical Manual of Defence Against Chemical Agents

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Note: These are not personal issues for retention by individual medical officers. They are to be held by units for use by medical officers while on unit strength.

Foreword

Chemical agents may be used singly, in combination or with smoke. They may precede, accompany or follow an attack with conventional weapons and may be fired from the ground, from the air or from ships.

There may be no warning of a chemical attack and the agent used may be one not previously suspected. Warsaw Pact countries now tend to include in their terminology of chemical agents certain chemically elaborated toxins that could be employed in field operations or covertly against important military and political objectives far behind the fighting front. The potential for effective dissemination by aerosol methods may give rise to clinical states differing from those produced by other means of contamination.

So that operational and medical commanders can give early and rapid warning of the need for protective and defensive measures, every medical officer must be on constant alert, not only to detect those syndromes described in this manual, but also for the appearance of unusual clinical manifestations among small or large bodies of troops in forward, rear or base areas. Such occurrences, however mild, demand immediate reporting so that military operations and casualty management continue effectively in a contaminated environment.

This manual should be read in conjunction with the *NATO Handbook on the Medical Aspects of NBC Defensive Operations.*

Preface to the fifth edition

Extensive revision of the manual has been made necessary by recent developments in the field of defence against chemical operations, including advances in therapeutics. The terms used are generally in agreement with the NATO Glossary and accordingly 'chemical warfare'has been replaced by 'chemical operations'. As in the previous edition, agents have been classified on the basis of their pharmacological actions, and a table has been included showing a comparison of the medical and equivalent service classifications.

Although the general format of the previous edition has been retained, there has been reorganization of the chapters. The section on Decontamination has been expanded, and because of the increased use of sensory irritant agents in riot control, a section on Riot Control Agents has been included. There is also a new chapter on Incapacitant Agents, which includes the more important group of psychotomimetic agents. Section III of the previous edition, Other Dangerous Gases, has been enlarged and replaced by Other Service Toxic Hazards. An addition to the section includes paragraphs on Fumes from Missile Propellants. Herbicides and Insecticides have been included because of their widespread use in agriculture and because of their potential use by an enemy.

Preface to the sixth edition

Further revision of the manual has been made to keep abreast of current developments in the field of defence in chemical operations and of advances in therapeutic techniques.

Although the general format of previous editions has been retained, there has been some reorganization of the chapters. The section on Recognition of the Chemical Casualty has been expanded to include some guidance on the medical aspects affecting casualty estimation, and a new chapter on Combined Injuries has been added.

The various formulae for chemical agents and drugs, and the diagrams showing methods of artificial respiration, have been left out of this edition to simplify presentation.

The Service code numbers or publishers of publications referred to in the text are given in the Bibliography.

Although it is not intended to encroach upon the matters discussed in the *Medical Manual of Defence Against Biological Agents*, in view of the ambiguous status of some toxins it is considered appropriate to include some explanation of this problem. This is given in a new Section 10.

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Section 1

General introduction

1 General description and history of chemical agents

Definition of chemical agents

0101 For the purposes of this manual, a chemical agent is defined as a chemical substance that is intended for use in military operations to kill, seriously injure or otherwise incapacitate man through patho-physiological effects.

0102 Excluded by this definition are riot control agents, herbicides, smoke and flame. However, the use of riot control agents, such as tear gas, has been extended in recent years to harass guerrillas, particularly to flush out or make untenable their hiding places; herbicides have been used to defoliate forest and bush areas in order to deny actual or potential sanctuary to an enemy; and smokes used in war or in peacetime field exercises may contain lung-damaging agents, as may smoke arising from burning materials, vehicles, tanks and ships. The medical services must consider these substances and the toxic effects of other poisonous agents which may be encountered under Service conditions. These aspects are covered in Sections 4 and 8.

History

0103 Burning sulphur is said to have been used by the Spartans at Thermopylae, but the first use of chemical operations in modern times occurred in 1915 during World War I. Chlorine gas was released from large cylinders in favourable wind conditions and the Allies, taken by surprise, suffered heavy casualties. The first respirator was improvized from a cotton pad soaked in sodium hypochlorite, glycerine and sodium carbonate, and successive improvements kept defence ahead of attack, at least as far as respiratory protection was concerned. However, the use of sulphur mustard gas against the Allies in 1917 was highly successful.

0104 Between World War I and World War II, mustard gas was used to considerable effect against Abyssinian tribesmen and troops.

0105 Chemical operations were not used in World War II, probably because of the high state of preparedness on both sides and because the Germans were not aware that the Allies had no nerve agent capability.

0106 As far as is known, chemical operations as defined have not been used to any great extent since World War II, although there have been reports of the use of chemical and biological agents in some theatres of hostile activity.

0107 Neither the advent of nuclear weapons nor the fact that chemical agents were not used in World War II precludes the possibility of their use in the future. It is known that the Warsaw Pact countries are well equipped and well trained in their use and it must be assumed that chemical weapons may be used in any future conflict.

Methods of disseminating chemical agents

0108 During World War I, chemical agents were used only in land weapons and the resulting casualties were due mainly to vapour and were confined largely to troops in the field. Between February 1915 and November 1918, 113 500 tons of chemical agents were used. These caused at least 1.3 million casualties of which at least 90 000 were fatal. In the last 18 months of the war, one in every six was a chemical casualty with mustard gas accounting for 70%, although the mortality from mustard gas attack was only 1.5%.

0109 In any future war, chemical agents could be disseminated by various methods; by shells or missiles fired from land or sea; by aeriel bombing or spraying; and by clandestine aerosol attack by saboteurs. Selected targets far removed from the fighting line, such as cities, factories, dockyards and air bases, are now within range of these methods of dissemination. It seems probable, therefore, that the nature and extent of casualty incidence will differ from that of World War I (see Section 2).

General factors influencing the choice and employment of chemical agents

0110 The effective use of any chemical agent is governed by its own physical and chemical properties and by meteorological conditions.

0111 For tactical purposes chemical agents may be divided into two main categories: *persistent* and *non-persistent.*

(a) Non-persistent agents are those which remain in effective concentrations for only a short time. They are released as airborne particles of a solid, droplets of a liquid, or as true gases. They are affected by prevailing weather conditions and are quickly dispersed, so that the locality in which they have been released soon ceases to be contaminated.

(b) Persistent agents are those which remain dangerous for some considerable time unless action is taken to destroy or neutralize them. They are usually liquid or solid at normal temperatures.

0112 The following meteorological factors are likely to influence the use of chemical agents:

(a) **Winds.** Strong winds rapidly disperse non-persistent agents in open country, although dangerous concentrations may take longer to clear from woods, dug-outs and built-up areas.

(b) **Temperature.** High temperatures increase the effectiveness of the less volatile persistent agents because they give off much higher vapour concentrations. Low temperatures may freeze persistent agents and will, in any case, increase their persistence. The danger of carrying such agents into a warm building on boots and equipment, and so giving off toxic vapour, should be borne in mind.

(c) **Rain.** Heavy rain reduces the effectiveness of chemical agents, but does not make them impossible to use.

(d) **Atmospheric stability.** When the air temperature is higher than that of the ground temperature (an inversion), agents in the vapour state will persist for longer periods than when the air temperature is lower than the ground temperature (a positive lapse rate).

0113 Downwind hazard distances are discussed in paragraph 0306.

Classification of chemical agents

0114 For medical purposes, chemical agents are usually classified according to pharmacological principles, but for general use throughout the Armed Services it is more appropriate to classify them according to their overall effects on combat efficiency. Medical officers must be familiar with both types of classification so that they can advise personnel of other arms. Table 1 — 1 shows the two methods of classification.

Table 1—1 Medical and Service classification of chemical agents

Medical classification	Service classification
Nerve agents (G and V)	Lethal agents (nerve)
Lung damaging agents (choking agents, phosgene (CG) and chlorine)	Lethal agents (choking)
Vesicant agents — blister agents (Sulphur mustard (HD), Lewisite (L), etc.)	Damaging agents (blister)
Psychotomimetic agents — incapacitants (LSD, BZ)	Incapacitating agents (mental)
Cyanogen agents (blood agents) (hydrogen cyanide (AC)) NB: the possible use of arsine (see paragraph 0135)	Lethal agents (blood)
Riot control agents (including vomiting agents) (DM)	Riot control agents — incapacitating agents (physical)

Brief outlines of these classes of agents are given in paragraphs 0115 — 0123, under two headings: those likely to be encountered in warfare and those likely to be encountered in warfare and/or in riot control. Full details are given in relevant chapters.

Agents likely to be encountered in warfare

Nerve agents

0115 Nerve agents (see Chapter 4) rapidly penetrate all mucous surfaces; the vapour is quickly absorbed by both upper and lower respiratory tract, and the cornea is permeable. They may be absorbed through the skin in either liquid or vapour form. They are almost odourless, effective in low concentrations and difficult to detect by the senses. Nerve agents in any form can act very rapidly and, when the dosage is heavy, convulsions may precede death. In percutaneous poisoning with liquid agents it may be some hours before toxic symptoms develop. With small doses of vapour the most prominent symptoms of exposure are constriction of the pupils, running of the nose and tightness of the chest. With large doses, preliminary symptoms are followed rapidly by iregular shallow breathing and slowing of the heart, possibly convulsions and death. If the dose is very large, death from absorption of vapour may occur in a matter of minutes. In percutaneous poisoning there are no early symptoms, but eventually a vague malaise develops. This is followed by nausea and vomiting, the same impaired breathing and possibly convulsions and death. The Service respirator gives protection to the eyes and respiratory tract, but special clothing is needed to protect the skin.

Lung-damaging agents

0116 These are non-persistent gases that cause pulmonary oedema and therefore asphyxia (see Chapter 5). Although some members of the group are dangerous in concentrations that cannot be smelt, others of the group are obvious to the senses in concentrations that can be breathed without danger. Trained troops wearing respirators would be protected, but heavy casualties might occur if these agents were used against a population with no respirators or inadequate training in their use.

Vesicant agents

0117 These are compounds (see Chapter 6) that in both the liquid and the vapour state, may cause immediate or delayed irritation and burning in varying degrees to those parts of the body with which they come into contact. The areas most sensitive to this action are the conjunctiva and the cornea, followed by the respiratory tract. Moist skin is particularly affected whereas dry skin is more resistant. Sulphur mustard gas has a faint smell and does not cause immediate irritation of the eyes, lungs or skin, even

in dangerously high vapour concentrations. The tissue destruction produced is not usually visible for some hours. These insidious qualities make it a very dangerous agent. Death may occur from inflamation of the respiratory tract, from the aftermath of extensive burning or from cytotoxic effects when the agent has been absorbed. The Service respirator gives protection to the eyes and respiratory tract, but special clothing is required to protect the skin.

Psychotomimetic or mentally incapacitating agents.

0118 These agents (see Chapter 7) produce severe mental derangement; in minute doses they will merely give changes in mood, varying from an apparent drunken happiness to deepest despair; in larger doses they produce severe hallucinations, and the casualty no longer knows who he is or what he is doing. Their military effect, therefore, varies from the disturbance of morale to the complete breakdown of military discipline resulting from inability to appreciate and carry out orders. Onset may be delayed from one to several hours and the duration of effects also varies with the agent used from a few hours to several days. With the slower-acting compounds there may be a period of physical incapacitation before mental incapacitation becomes apparent. Normally, recovery occurs without treatment. Mental incapacitants are active in such small amounts that they could be used in a covert role to poison water or food and would not be detected; in the overt role they can be disseminated as smokes or dusts. Some are liquids and absorption through the skin could result from aerosol dispersal. The Service respirator gives protection to the eyes and respiratory tract, but special clothing is needed to protect the skin.

Immobilizing or physically incapacitating agents

0119 These compounds (see Chapter 7) have the effect of immobilizing and temporarily paralysing. Other compounds, e.g. BZ listed under Psychotomimetic agents, in Table 1 — 1 are also known to produce ataxia; effects may last from minutes to hours, but recovery is complete without treatment unless very large doses have been given.

Miscellaneous agents

0120 These include agents that cause little or no local injury, but produce effects after absorption into the body (see Chapter 8). The most important are hydrogen cyanide (prussic acid) and cyanogen chloride. At high concentrations death occurs rapidly through paralysis of cellular respiration, but cyanogen chloride at low

concentrations has both tear and choking effects. The Service respirator gives protection to the eyes and respiratory tract against both these agents.

Agents likely to be encountered in riot control and/or warfare (sensory irritants and vomiting agents)

0121 Many compounds in the forms of vapours, smokes or fine dusts, affect the eyes and upper respiratory tract and some of them, particularly at low concentrations, have a pronounced and immediate action, causing spasm of the eyelids and lachrymation, severe burning pain in nose and chest, violent coughing which may result in vomiting, and often profuse salivation (see Section 4). The skin may smart if it is moist. The concentrations required to produce these effects in the field do no permanent harm to the eyes or the respiratory tract. They are readily detected by their effects. Although exposed skin continues to feel sore, eye and chest symptoms pass off quickly when a respirator is put on, provided the respirator is cleared (i.e. by forcibly breathing out to the fullest extent immediately the respirator is in position and before inhaling). Hence these agents, when used against trained personnel, cause only transitory discomfort.

0122 Some of these agents may be used in a pressurized canister so that a jet of solution can be aimed at an assailant from distances of about 3.5 m.

0123 Vomiting agents are disseminated as smokes and cause pain in the upper respiratory tract, sneezing, eye symptoms, nausea and vomiting. These effects may last for several hours, but in field concentrations there is no residual damage. Complete protection is given by the Service respirator.

Service toxic hazards

0124 There are certain other substances, a few of which have at some time been considered for use in chemical operations, that are toxic and might cause casualties in a theatre of war. Such casualties will need to be differentiated from those caused by deliberate chemical operations. The more important substances or groups of substances are listed below:

(a) Carbon monoxide and carbon dioxide.

(b) Nitro-explosive fumes.

(c) Fumes from fuels and lubricants (petrol, diesel and jet fuels).

(d) Smokes and incendiary substances.

(e) Fumes from missile propellants, e.g., nitric acid and boranes.

(f) Fumes encountered in fire fighting, e.g., halogenated hydrocarbons and phosgene.

(g) Herbicides.

(h) Insecticides.

(i) Chloropicrin.

(j) Chlorine.

(k) Arsine.

(l) Hazards from refrigeration plants, e.g., ammonia and sulphur dioxide.

(m) Hydrogen sulphide.

Carbon monoxide and carbon dioxide

0125 Carbon monoxide occurs in coal gas and in exhaust fumes from internal combustion engines and jet engines. It is formed also by coke or charcoal braziers and may be encountered in bomb craters and in badly ventilated emplacements. Dangerous concentrations are not likely to occur in the open. It is invisible and has no smell. The gas produces its insidious effects through its interference with the respiratory functions of the blood. Exhaust fumes from engines may also contain acrolein and other products of combustion which can cause lachrymation but they are not dangerous in the concentrations normally produced by engines. Pockets of carbon dioxide from combustion may also occur, e.g., at the bottom of craters or in badly ventilated tunnels. This colourless odourless gas, being heavier than air displaces it. Service respirators give no protection against either carbon monoxide or carbon dioxide.

Nitro-explosive fumes

0126 These are given off by burning cordite or by nitro-explosives when detonation is incomplete. There is considerable danger from these fumes if high explosives of this type are burnt or detonated in enclosed areas with insufficient ventilation. This may occur in gun-pits, armoured vehicles, ships' magazines and turrets, and in mining and tunnelling operations. They act as powerful and very insidious lung irritants with delayed symptoms resembling those of

phosgene poisoning, and it is important to remember that carbon monoxide is often present at the same time. Service respirators generally give some degree of protection against nitrous fumes, but none against carbon monoxide. Fumes of unexploded cordite, mostly nitroglycerine, will cause headache and malaise.

Fumes from fuels and lubricants (petrol, diesel and jet fuels)

0127 In general these hydrocarbons have narcotic effects and in high concentrations they will produce unconsciousness and death. Petrol fumes are toxic to humans in concentrations over 1% except when exposure is short. Dangerous concentrations may be encountered in tanks, wagons or compartments which contain or have contained petrol. As the Service respirator gives only limited protection, personnel should be protected by airline respirators or oxygen apparatus. Kerosene (paraffin) and diesel fuels, being less volatile, are relatively less dangerous.

Smokes and incendiary substances

0128

(a) Screening smokes used in field exercises and in battle are generally non-toxic in the concentrations encountered. However, exposure near the source, or for long periods, or in a confined space or in conditions of low humidity can result in severe or even fatal pulmonary damage; particularly if the smoke contains zinc chloride. These smokes could also be used for the dissemination of chemical warfare agents.

(b) Smokes from burning vehicles, tanks, aircraft and ships may contain a variety of toxic agents which have severe lung-damaging or anoxic properties.

(c) The Service respirator gives complete protection from smokes in (a) above, but protection for a brief time only against heavy concentrations of smokes in (b) above as the filter rapidly becomes overwhelmed.

(d) Phosphorus, which is used as a smoke filling, is also a dangerous incendiary substance. The flying fragments which ignite in air cause burns which are multiple, deep and variable in size. The smoke is irritating. Thermite, magnesium and its alloys, napalm, and fuel oils are now familiar as incendiary substances.

See also paragraph 0510 — Treatment for lung-damaging agents.

Fumes from missile propellants

0129 Missile propellants vary from fuming nitric acid to hydrogen peroxide, boranes and hydrazines. They are unlikely to be encountered unexpectedly or over a large area. Their effects vary from respiratory distress (fuming nitric acid) to headache and convulsions (boranes). Hydrogen chloride occurs as an exhaust gas from certain missiles; it is not dangerous in the concentrations likely to be encountered. There is no vapour hazard from hydrogen peroxide or hydrogen chloride. The Service respirator gives limited protection against burning nitric acid, boranes and hydrazines. Appropriate protective clothing against liquid spills is essential.

Fumes encountered in fire fighting

0130 Carbon monoxide, nitrous fumes and oxygen deficiency, encountered when fighting fires in confined spaces, constitute hazards. Some chemical fire extinguishers contain carbon tetrachloride and others methyl bromide, both of which are toxic substances. When carbon tetrachloride comes into contact with molten magnesium it decomposes, producing phosgene, chlorine and hydrochloric acid. Chemical fire extinguishers containing carbon tetrachloride should not be used in any circumstances for controlling incendiary bombs, especially as the Service respirator will not protect against carbon monoxide or carbon dioxide. Methyl bromide in high concentrations has a profound effect on the central nervous system, producing unconsciousness, epileptoform seizures and paralysis. Owing to its rapid vaporization a toxic dose may be inhaled before the danger is appreciated; the resultant lung damage may be lethal. The liquid is also a vesicant. Other types of fire extinguisher may contain fluorinated hydrocarbons (refrigerants, freons); some of these decompose to phosgene under certain circumstances with a resulting toxicity hazard. Phosgene may also be produced from the combustion of certain plastics. The Service respirator protects against phosgene, carbon tetrachloride and methyl bromide, but some decomposition products of certain of these substances and of fluorinated hydrocarbons may not be excluded. The greatest hazard is carbon monoxide.

Herbicides

0131 Some herbicides have been employed as defoliants and anti-crop agents in war. Those used up to now are only slightly toxic to man.

Insecticides

0132 Organophosphorus and carbamate insecticides produce acute effects similar to those of nerve agents, but much larger doses are required.

Chloropicrin

0133 This oily liquid or pungent vapour was used in World War I as an irritant agent, and is now used in agriculture as a soil fumigant and disinfectant. The Service respirator gives complete protection to the lungs and eyes from vapour effects, but decontamination of the skin and clothing is required if liquid contamination occurs.

Chlorine

0134 This gas was used during World War I as a choking agent, and is now used extensively in industry and for the purification of water. It is a pale yellowish-green gas, having a strong smell resembling that of bleach. It is less deadly than phosgene, but causes more initial coughing and choking. The Service respirator gives complete protection.

Arsine (arseniuretted hydrogen)

0135 This colourless, sweetish-smelling gas has been considered for use in chemical operations and it may be encountered in certain industrial practices. Inhalation of a large quantity causes shivering, giddiness and vomiting. Its major clinical effects are anaemia, jaundice and haemoglobinuria due to haemolysis of red blood cells, and obliguria from acute tubular necrosis. Death is due to a combination of these pathological processes. The Service respirator gives complete protection.

Hazards from refrigeration plants (ammonia and sulphur dioxide)

0136 These refrigerant gases, available in cylinders, could be used as harassing agents as they cause lachrymation, blepharospasm and coughing. Ammonia has a characteristic smell; sulphur dioxide has an acrid sulphurous smell. The Service respirator will give some protection, especially against sulphur dioxide.

Hydrogen sulphide

0137 This gas, commonly encountered in sewers, may be, under certain circumstances, as dangerous as hydrogen cyanide (prussic acid), as it has a similar toxic action. Although the smell is distinctive, high concentrations quickly paralyse the sense of smell. The Service respirator gives complete protection.

Biological agents

0138 Biological agents may be micro-organisms including viruses, or toxins derived from living organisms or by chemical synthesis. It is important to note that some of these agents have very different effects when inhaled compared with those encountered in normal clinical practice (e.g., staphylococcal enterotoxin may cause respiratory effects when inhaled).

0139 Apart from the possibility of ingesting contaminated food and water, protection of the respiratory and gastro-intestinal tracts is given by the Service respirator. Immunization and the prophylactic use of antibiotics are other possible methods of protection but these methods depend on sure knowledge of the agents likely to be used. Toxins may appear to be a borderline case between biological and chemical agents, but are defined as biological by the Geneva Convention of 1972. Further discussion of biological agents is presented in Section 10, but for full consideration of this subject the *Medical Manual of Defence Against Biological Agents* should be consulted.

0140 Smokes, such as those used in screening, may be used in conjunction with, or to disguise, the use of chemical or biological agents. Mixture of these agents should be suspected if any unusual effects are seen as they may be disseminated during a conventional weapons attack.

Section 2

Recognition of the chemical casualty and expectation of casualty figures

2 Recognition of casualties

Introduction

0201 It is essential that medical officers are familiar with the signs and symptoms of chemical agent poisoning to avoid repetition of the experience of World War I in which '... medical officers frankly admitted that they were so handicapped by their lack of experience of cases of gas poisoning that they were often in doubt whether they were dealing with men suffering from gas poisoning or not'. (*Official History of the Medical Services — Diseases of War,* Vol 31 (1923) page 317, referring to the Battle of Loos (25—27 September 1915) when chlorine was being used).

0202 Some of the problems in the recognition and diagnosis of casualties suffering from the effects of chemical operations are discussed here and are summarized in Table 2 — 1. The medical officer must bear in mind that, with the nerve agents, incapacitating symptoms do not usually appear until the individual is severely poisoned, and in many cases the casualty will have already received first aid. Although choking agents are less likely to be employed, the possibility of their use should not be forgotten, and here the danger is that the quiescent period which follows the initial poisoning might be mistaken for recovery and men sent back to duty even after a lethal dose. Battle casualties whose behavioural changes are not compatible with the physical signs of disability must be examined carefully to exclude the possibility of a psychotomimetic agent having been used. When chemical agents have been used by the enemy, it is important that the fullest and earliest information be given to medical units to facilitate the diagnosis of individual cases and to permit adequate arrangements for the reception of casualties. Medical units should rely on information not only from detectors, but also from the casualties themselves. This applies particularly to agents for which at present there is no satisfactory detector, such as incapacitating agents.

Recognition of a casualty of chemical operations

0203 Any individual who suddenly becomes a casualty without being wounded or who is suffering a greater degree of incapacitation than is compatible with his wound should be considered a possible chemical casualty. It is unlikely that chemical agents would produce single casualties under field conditions and a chemical attack should be suspected with any sudden increase in numbers of unexplained casualties. If chemical operations are unlikely, and if only a few men are affected, a simple toxic hazard is more probable (see Section 8).

0204 Bearing in mind that the medical situation may be complicated by the psychological effects of the operational circumstances, the medical officer's questions should be along the following lines:

(a) To determine whether the casualty has been caused by a chemical agent.

(1) Was the casualty wearing full protective equipment at the time of the attack?

(2) Were there any aircraft or artillery bombardment in the area at the time of the attack?

(3) Was there any evidence of spray, liquid droplets or smoke?

(4) Was anybody else affected? How was he affected?

(5) Did the casualty notice any unusual smell? (This is not a very reliable sign under battle conditions, but it should be considered.)

(6) Did the available detection equipment respond positively? (See Table 2 — 2).

(b) To determine the identity of the agent.

(1) What subjective effects did the casualty notice and how soon?

(2) Was there any delay between exposure or contamination and the onset of effects, and if so, for how long?

(3) Did the effects persist after adjustment of the respirator?

(4) Has the casualty used any self-injection device? If so, did the symptoms improve or deteriorate?

(5) Is the casualty's behaviour normal?

(c) To assess the dose of agent received.

(1) Was the casualty exercising or at rest?

(2) Was the casualty in the open or under cover?

(3) For how long was the agent inhaled? How long was the interval between suspected contamination and decontamination?

Clinical appearances

0205 The principal effects of chemical agents and their differential diagnosis are summarized in Table 2 — 3. Further information on the recognition and handling of chemical casualties is given in the *NATO Handbook on the Concept of Medical Support in NBC Environments.*

Detector equipments

0206 Detector equipments and responses of various agents are given in Table 2 — 2.

Table 2—1 Summary of agents, properties, methods of recognition and first aid

Agent	Recognition*	Clinical effects	Self-aid	First-aid	Remarks
Nerve agents G agent—(non-persistent) V agent—(persistent)	Colourless gas and colourless to pale yellow liquid. In water use Water Testing Kit, Poisons	Tightness of chest; headache; rhinorrhea and salivation; miosis and dimming of vision if liquid or vapour come into direct contact with eyes; nausea and vomiting; sweating; convulsions; dyspnoea; respiratory failure	Combopen autoject. Repeat twice at 15-min interval if symptoms persist. Decontamination of skin exposed to liquid agent	Combopen autoject. Repeat twice at 15-min intervals if symptoms persist. Decontamination of casualty and resuscitator. Artificial resuscitation —NBC portable resuscitator	Speed is vital in treating casualties. Combopen injections must be given as soon as possible. Personnel at risk should already be taking Pyridostigmine 30 mg 8 hourly
Lung-damaging agents Phosgene (non-persistent)	Colourless gas which may form white cloud. Smell of new-mown hay. No device available for detection	Lachrymation; coughing choking; tightness in the chest with pain. Nausea and vomiting. Latent period 30 mins — 24 hrs followed by signs and symptoms of pulmonary oedema. Haemoconcentration, anoxia, circulatory collapse	Steroid inhaler	Steroid inhaler. Warmth, strict rest and oxygen if available. Coughing controlled by codeine	Initial symptoms not of reliable prognostic significance
Cyanide-type agents Hydrogen cyanide (non-persistent)	Colourless gas or volatile liquid. Smell of bitter almonds. Detected by Water Testing Kit, Poisons	Mild cases; headache, nausea and vertigo. Higher concentrations; in addition, convulsions and coma. High concentrations: increase in depth of respiration; violent convulsion and cessation of respiration within 1 min.	Steroid inhaler	Artificial respiration preferably by positive pressure and oxygen if available. Intravenous injection of 20 ml dicobalt edetate followed by 25—50 ml of 50% sodium thiosulphate	In severe cases death comes rapidly, therefore speed in treatment is most urgent. Canister life of respirator shorter than for other agents

*See also Table 2—2 for detection by NAIAD, CAM detector papers and RVD.

Table 2—1 Summary of agents, properties, methods of recognition and first aid *(continued)*

Agent	Recognition*	Clinical effects	Self-aid	First-aid	Remarks
Cyanide-type agents *continued* Cyanogen chloride Cyanogen bromide (non persistent)	As above for hydrogen cyanide	Above Systemic effect modified by irritant properties involving eyes, nose and throat with tightness of chest and coughing	Steroid inhaler	As above for hydrogen cyanide and phosgene	
Vesicant agents Sulphur mustard (very persistent)	Colourless to dark-brown oily liquid or colourless gas. Smell of garlic. Detector paper changes colour in presence of liquid and vapour detected by Residual Vapour Detector. In water, detected by Water Testing Kit, Poisons	*Eyes.* Delayed effects after 1 hr or more: smarting lachrymation followed by severe conjunctivitis with pain, photophobia and blepharospasm. Oedema of eyelids and watery discharge. Temporary, rarely permanent, blindless	Mop up visible drops of agent. Decontamination procedures. If eyes involved, irrigate with water immediately. Use steroid inhaler even in absence of respiratory symptoms	Following irrigation of eyes use mydriatics, antibiotics and local anaesthetics. Treat skin blisters as for thermal burns. Respiratory symptoms treated symptomatically. Use steroid inhaler even in absence of respiratory symptoms	
Nitrogen mustards (persistent)	Colourless or pale-yellow oily liquids. Some compounds have slight fishy smell	*Skin.* Delayed effects: Erythema followed by vesication and blistering. Vapour particularly affects moist areas *Respiratory tract.* Symptoms after several hours with rhinorrhoea followed by hoarseness and cough. Bronchitis may be followed by bronchopneumonia *Haemopoietic system.* Depression following absorption from skin or respiratory tract			

*See also Table 2—2 for detection by NAIAD, CAM, detector papers and RVD.

Table 2—1 Summary of agents, properties, methods of recognition and first aid *(continued)*

Agent	Recognition*	Clinical effects	Self-aid	First-aid	Remarks
Vesicant agents *continued*					
Arsenical vesicants, e.g, lewisite and the Dicks (persistent)	Colourless to dark-brown oily liquids or colourless gas. Lewisite has geranium-like smell whereas the Dicks have a more fruity odour	Contact produces immediate pain in the eyes or skin. Erythema, vesication and eye injury develop rapidly. Effects of arsenical poisoning may be produced	Mop visible drops of agent. Decontamination procedures. If eyes involved, irrigate with water immediately. Use steriod inhaler even in absence of respiratory symptoms	In addition to above, dimercaprol should be used locally for the skin or eyes, and given by intramuscular injection in case of systemic absorption	Similar effects to those of sulphur mustard, but absorbed more quickly and acts more rapidly. Treatment must therefore be prompt
Phosgene oxime (persistent)		On contact with skin produces immediate pain. This is followed by skin necrosis and ulceration	The skin should be flushed with water to remove excess agent	Necrotic skin lesions treated as for thermal burns	
Psychotomimetic agents					
BZ (non-persistent)	Crystalline solid at normal temperature and disseminated as a smoke. No detection device available at present	Mydriasis, dry mouth, tachycardia followed by ataxia and drowsiness. Later confused mental state with delusions and hallucination	None	Firm restraint. Removal of dangerous objects. Symptomatic treatment	Physostigmine specific treatment for selected casualties. If hyperkinetic watch for heat illness
Riot control agents					
Sensory irritants CS CN CR	Crystalline solid at normal temperature. Disseminated as a smoke or mist or from explosive devices	Burning sensation in eyes with lachrymation. Sneezing, soreness of chest, coughing, difficulty in breathing. Higher concentrations produce nausea and vomiting. Irritation of skin and erythema. Conjunctivitis, tracheitis and bronchitis following exposure to high concentrations	Move to fresh air; face into wind with eyes open; breathe deeply	CS should be removed from skin with soap and water. Gross exposure of eyes treated by irrigation with water or normal saline	Normally treatment is not necessary

* See also Table 2—2 for dectection by NAIAD, CAM, detector papers and RVD.

Table 2—1 Summary of agents, properties, methods of detection and first aid *(continued)*

Agent	Recognition*	Clinical effects	Self-aid	First-aid	Remarks
Vomiting agents DM (non-persistent)	Crystalline yellow solid at normal temperature. Disseminated as a smoke which is canary yellow when concentrated	Acute pain in nose and sinuses. Burning sensation in throat with tightness and pain in chest. Irritation of eyes with pain and lachrymation. Nausea and vomiting. Mental depression in severe cases. Coughing and sneezing	Respirator must be worn in spite of coughing, sneezing, salivation and nausea.	Normally not necessary. Vigorous exercise will help to reduce intensity and duration of effects	

* See also Table 2—2 for detection by NAIAD, CAM, detector papers and RVD.

Table 2—2 Detector equipments

Nerve agent	NAIAD	CAM	Detector Paper No 1[1]	Detector Paper No 2	RVD
Liquid G		*	*	Yellow	
Liquid V		*	*	Green	
Vapour nerve agent	*	*			*
Mustard liquid			*	Red	
Mustard and vesicant vapour		*			*
Cyanide vapour[2]	*				

Note: Asterisk denotes positive response.

1. Detector Paper No 1 turns blue for all liquid agents.

2. Only in attack concentrations.

Table 2—3 Summary of clinical effects of chemical agents and their differential diagnosis

1. Skin		
(a) Colour	Grey or cyanosed	*Lung-damaging agent* — (late effects) respiration increased, cough, pain and dyspnoea, followed after latent period by expectoration and evidence of pulmonary oedema (transient cough — sensory irritants and vomiting agents)
		Cyanide-type agent — respiration slowed with increased depth, followed by cessation of respiration
		Nerve agent (late effects) — laboured respiration from broncho-constriction and fluid in airway
	Flesh pink	*Cyanide-type agent*
	Erythema	*Sensory irritant agent* — transient blotchy pattern, especially on moist areas on exposure to high concentrations (transient effect on eyes and respiratory tract)
		Vesicant agent — prolonged effect followed by vesication and/or blistering (may be associated with severe effects on the eyes and respiratory tract)
(b) Sweating	Excessive	*Nerve agent* — also salivation, rhinorrhoea and excessive bronchial secretions (transient salivation and rhinorrhoea from sensory irritants and vomiting agents)
	Diminished	*Psychotomimetic agent* (BZ)

Table 2—3 Summary of clinical effects of chemical agents and their differential diagnosis *(continued)*

2. Eyes		
(a) Red and watering		*Sensory irritant agents and vomiting agents* — transient effects only *Vesicant agents* — also oedema of eyelids, severe conjunctivitis and oedema of cornea. Temporary or permanent blindness
(b) Pupil	Miosis	*Nerve agent*
	Mydriasis	*Psychotomimetic agent* (BZ)
3. Central nervous system (CNS) effects		
(a) Mental state	Uninterested, aloof and unresponsive to commands	*Psychotomimetic agent* — e.g., LSD
	Confused with delusions and aimless behaviour	*Psychotomimetic agent* — e.g., BZ
		Cyanide-type agent — (delayed effect with residual brain damage)
	Depression	*Vomiting agent* — severe effect

Table 2—3 Summary of clinical effects of chemical agents and their differential diagnosis *(continued)*

(b) Neuromuscular	Unsteady gait and weakness	*Vomiting agent* — associated with sensory irritant effects
		Psychotomimetic agent — LSD or BZ, associated with mental symptoms
		Cyanide type agent — with residual brain damage
	Muscular weakness with fasciculation	*Nerve agent*
	Paralysis	*Nerve agent*
(c) Convulsions	Terminal	*Nerve agent*
		Cyanide-type agent — severe effects
	Delayed	*Cyanide-type agent* — mild effects
4. Abdominal effects		
(a) Vomiting	*Nerve agent* — profuse and uncontrollable, with other effects of systemic poisoning *Vomiting agent* — associated with excessive coughing *Lung-damaging agent* — associated with excessive coughing *Sensory irritant agent* — in high concentrations associated with excessive coughing *Cyanide-type agent* — occasionally seen in mild cases associated with nausea and vertigo	
(b) Incontinence of urine or faeces	*Nerve agent* — in near lethal exposure	

3 Expectation of casualty figures

Introduction

0301 This chapter should be read in conjunction with:

(a) *Manual of Nuclear Biological and Chemical Defence Training on Land*, Pamphlet No 2: *A Guide for NBC Advisers*, particularly Annex A to Chapter 10, and

(b) *Allied Tactical Publication* (ATP 45), Chapter 12.

0302 Casualty estimation is the responsibility of operational planners. So many variables are involved that it will be difficult to predict chemical casualty figures accurately. This chapter discusses these variables in broad outline so that medical officers are better able to advise commanders on the medical aspects affecting casualty estimation.

0303 Whether a person becomes a casualty depends on the amount of chemical agent that penetrates the body, and it must be remembered that the type of chemical agent used is chosen mainly on its ability to incapacitate or kill when absorbed in only very small amounts. Although protective equipment is designed to prevent chemical agents from penetrating the body it is unlikely that full protection could be worn at all times. The chemical attack will therefore seek to surprise the target personnel when their nuclear, biological and chemical (NBC) protection is not complete.

Main factors affecting the numbers of casualties

0304 The numbers of casualties will depend on two main factors:

(a) The first is the speed and concentration at which the agent or agents can be delivered to the target. This in turn will depend on:

(1) The type of weapon system used for delivery;

(2) The characteristics of the agent;

(3) The weather and terrain in the target area;

(4) Whether the target population is concentrated in a well defined area (e.g., airfields or landing sites, workshops, supply dumps and artillery gun lines) or dispersed in tactically sited defences in forward areas or around installations in rear areas or home bases;

(5) Whether the target population is in the open or under cover (e.g., inside vehicles, aircraft, field defences, shelters or buildings).

(b) The second main factor is the speed at which the target population can assume full protection. This in turn will depend on:

(1) The effectiveness of NBC equipment;

(2) The current NBC alert state and the NBC dress state;

(3) The alertness of personnel and their state of NBC training.

0305 An additional factor is that casualties from persistent agents will be spread over a long period and will be difficult to predict, as will the effects of dual agents and any possible new agents.

Downwind hazard prediction

0306 For the purpose of downwind hazard area prediction, two categories of chemical agent and two types of chemical attack are recognized (see Table 3 — 1):

(a) **Categories of chemical agent:**

(1) *Casualty-producing agents.* These are agents that are normally expected to be dispersed as an aerosol or vapour cloud with little or no contamination of the ground, e.g., non-persistent nerve agent.

(2) *Ground-contaminating agents.* These are agents which are normally expected to be dispersed in liquid form to contaminate surfaces, e.g., sulphur mustard and persistent nerve agent.

(b) **Types of chemical attack**:

(1) Type A attack — casualty-producing agent;

(2) Type B attack — disseminating ground-contaminating agent.

NB: All attacks will be assumed to be Type A unless there is unmistakable indication of ground contamination.

Table 3—1 Downwind distance hazard areas

Means of delivery	Distance from centre of attack area along downwind axis when weather condition is: Unstable	Neutral	Stable
Type A attack			
Artillery, bomblets and mortars	10 km	30 km	50 km
Multiple rocket launchers, missiles and bombs	15 km	30 km	50 km

NB: When the type of munitions used in the attack is not known, the figures given for multiple rocket launchers, missiles and bombs will be used.

Type B attack

The maximum downwind distance of the hazard area in this type of attack is always 10 km

Section 3

Agents likely to be encountered in warfare

4 Nerve agents

Introduction

0401 The first nerve agent, Tabun (GA), was discovered in 1936 during research into the organophosphorus insecticides. It was noted to be active in very low dosage, with effects similar to those caused by anticholinesterase drugs. Since then, many related compounds have been found, some of which are even more potent. Those which are of military importance are now included in the generic term 'nerve agents'. Some have been given names but they are more usually known by code letters, e.g., GA, GB, GD and VX.

Physical and chemical properties

0402 Most nerve agents are liquid organophosphorus esters whose volatility varies over a range similar to that between petrol and heavy lubricating oil. They have low freezing-points, none freezing until $-40°C$.

(a) **Appearance**. Liquid nerve agents are pale yellow to colourless, but may have a slightly darker colour due to impurities. They are essentially odourless.

(b) **Stability**. Nerve agents are fairly soluble in water, being very slowly broken down by hydrolysis, yielding less toxic products. They are rapidly destroyed by strong alkalis and bleaching powder.

(c) **Powers of penetration**. Normal clothing is penetrated by these agents in both the liquid and vapour states. Liquid agents usually penetrate by diffusion of vapour through the fabric. Leather is penetrated in the same manner as skin, but rubber and synthetic materials such as butyl rubber and polythene are more resistant. Penetration of the Suit Protective NBC will not occur within 6 hours.

These agents can penetrate materials which are normally non-absorbent such as webbing, leather and wood and can continue to present a hazard by desorption of the vapour.

(d) **Persistence**. A wide variation in volatility between different members of the group leads to a wide range of persistencies. Additives may be used to alter the persistency of any one agent. The G agents are much less persistent than the V agents.

Detection

0403 Nerve agents can be detected by chemical reagents that induce a colour change and by methods that detect the inhibition *in vitro* of the enzyme cholinesterase or the presence of ionized agents. In liquid form the agents react with Detector Paper No 1 Mk II and Detector Paper No 2 Mk I. The vapour may be detected by the Nerve Agent Inhibited Enzyme and Alarm Detector (NAIAD) system, by the Chemical Agent Monitor (CAM) and by the Residual Vapour Detector (RVD). Water contamination above 0.5 ppm may be detected with the Water Testing Kit, Poisons. Contamination by some other agents may also be detected by these methods (see Table 2—2).

Protection

0404 Ordinary clothing gives very little protection against nerve agents and special protective garments are required. The Suit Protective NBC, Gloves Protective NBC and the Respirator NBC, give complete protection against these agents, in both the liquid and vapour state, for at least 6 hours. The suit and gloves should be changed within 6 hours. Boots which have leather uppers are slowly penetrated by the agent; additional protection can be gained by using overboots and by the liberal use of fuller's earth inside the boots.

Decontamination

0405 Liquid agent on the skin must be removed as soon as possible. It must also be removed from personal and unit equipment to prevent a continuing hazard.

(a) Decontamination of the skin is best carried out by means of the Decontamination Kit Personal No 1. This contains pads that release fuller's earth when dabbed and rubbed on the skin. The powder soaks up the liquid and retains it

by absorption. For large areas of contamination and for items of personal equipment, such as webbing and small arms, the Decontamination Kit Personal No 2, which is a puffer bottle containing fuller's earth is recommended.

(b) Expendable materials should be burned or buried with a quantity of bleach slurry to ensure destruction of the agent. Burning will cause toxic vapours and protection of individuals in the vicinity and downwind must be borne in mind.

(c) In attempting to decontaminate large items of equipment, such as vehicles, aircraft or weapons, it must be remembered that nerve agents are absorbed by such materials as paint, rubber and plastic and get into crevices such as screw threads. If time allows, decontamination can be attempted using the Decontamination Apparatus Portable NBC or by scrubbing with chemical agent decontaminant. However, as these procedures take a long time the usual policy will be to rely on personal protection and to *fight dirty* with contaminated weapons and equipment if necessary.

Mechanism of action

0406 Nerve agents inhibit the enzyme acetylcholinesterase. This enzyme hydrolyses acetylcholine which is liberated when nerve impulses reach cholinergic nerve endings. Thus, the effect of a nerve agent is poisoning by excessive accumulation of acetylcholine.

0407 The parasympathetic nerve endings most obviously affected are those supplying the iris and ciliary body; the lachrymal and salivary glands; and the glands and muscles of the bronchial tree and gastro-intestinal tract. Acetylcholine is also released in cardiac muscle from vagus stimulation and at the sympathetic nerve endings of the sweat glands. Symptoms due to an accumulation of acetylcholine at these sites are referred to as muscarinic symptoms and those due to acetylcholine accumulated at the neuromuscular junctions and the pre-ganglionic sympathetic synapses are referred to as nicotinic symptoms. In addition there are less well-defined central effects (see Table 4 — 1).

Table 4—1 Pharmacology of nerve agents

Type of action	Site of action	Response
Muscarinic	Glands Sweat Salivary Nasal Bronchial Gastro-intestinal	Increased secretion
	Smooth muscle Bronchial	Constriction
	Cardiovascular	Bradycardia
	Iris	Miosis
	Gastro-intestinal	Increased mobility Colicky pain Diarrhoea
	Bladder	Involuntary micturition
Nicotinic	Pre-ganglionic synapses	Hypertension Pallor
	Neuromuscular junction	Weakness Muscular twitching Fasciculation Paralysis
Central	Central nervous system	Apprehension Hyperexcitability Weakness Incoordination Convulsions Respiratory failure

0408 The nerve agents are cumulative poisons and repeated exposures to low concentrations, if not too widely separated, will eventually give rise to symptoms due to a gradual inhibition of acetylcholinesterase activity in the blood and tissues. Restoration of the cholinesterase activity to normal levels takes several weeks, but clinical recovery from acute effects usually takes place within a few days, due in part to a process of adaptation to lower levels of the enzyme.

Pathology

0409 The damaging effects of nerve agents are on function and not on structure. Post mortem examination reveals signs consistent with death from asphyxia and there is usually evidence of blocking of the air passages with fluid secretions, if the case has not been treated with atropine, and of oedema of the lungs. These, together with a decrease of acetylcholinesterase activity, which can be assayed in autopsy material, are the only objective signs.

Signs and symptoms

0410 These vary with the route and severity of poisoning. Some agents are normally vapours, e.g., GA and GB; others are normally liquids, e.g., VX. The former attack principally by the respiratory route, the latter mainly through the skin.

Respiratory route

0411 Poisoning may be mild or severe:

(a) *Mild poisoning.* Signs and symptoms may become noticeable within a few minutes of inhalation of even low concentrations of nerve agent. These are tightness of the chest, rhinorrhoea and salivation, miosis with dimming of vision and difficulty in accommodation accompanied by frontal headache (see Plate 1). These clinical effects are largely due to the local absorption of nerve agent and may be expected to persist for only a few hours, although headache and visual difficulties may last up to 3 days.

(b) *Severe poisoning.* The signs and symptoms referred to above become more pronounced; salivation and rhinorrhoea become so profuse that watery secretions run out of the mouth and nose. Respiration becomes laboured because of obstruction from bronchoconstriction and excessive bronchial secretions and audible wheezing will occur. Systemic effects from general absorption of the agent will become apparent with severe sweating, profuse and uncontrollable vomiting, severe colicky abdominal pain and involuntary defecation and micturition. Progressive muscular weakness occurs with fasciculation, convulsions and paralysis. Advancing intoxication is accompanied by anxiety and depression of cerebral function; there may be

acute psychological disturbance. Death is due to asphyxia from combination of central and peripheral respiratory failure and the accumulation of fluid in the bronchial tree.

Cutaneous route

0412 Local effects as described in sub-paragraph 0411a do not occur. The syndrome consists of those systemic effects described in sub-paragraph 0411b preceded by general malaise. Miosis is not a significant feature of poisoning by this route. The progress of events is slower than in poisoning by the respiratory route.

Gastro-intestinal route

0413 The onset of signs and symptoms from the ingestion of food and water contaminated with nerve agent is likely to be more rapid that the onset following skin absorption. The syndrome, however, is similar. Vomiting may occur, but abdominal colic, a typical feature of nerve agent poisoning, is likely to be the presenting symptom when this route is involved. Miosis is not a significant feature of poisoning by this route.

Treatment

0414 Successful treatment of nerve agent poisoning depends upon the speed with which the asphyxiating effect of the accumulated acetylcholine is countered. This effect is due to three factors: paralysis of the respiratory muscles, especially the diaphragm; failure of the respiratory centre in the brain; and obstruction to breathing by bronchoconstriction and excessive bronchial secretions. Positive pressure resucitation may be necessary; three drugs are available for treatment, namely, atropine, oxime and diazepam, with pyridostigmine for pre-treatment. Atropine blocks the action of accumulated acetylcholine at muscarinic sites; oxime (pralidoxime mesylate, P2S) reactivates the inhibited cholinesterase, at least until the phenomenon of ageing due to dealkylation occurs. Diazepam acts as an anti-convulsant, but it also appears to have a specific action of its own, possibly at nicotinic receptor sites in the central nervous system. Pre-treatment with pyridostigmine protects a portion of the peripheral cholinesterase from inhibition by nerve agent and this portion of cholinesterase is rapidly released. The combination of pre-treatment and efficient therapy has been estimated to protect against some 10—20 lethal doses of most nerve agents, but ageing of the complex formed by GD and cholinesterase

occurs almost instantaneously so that therapy for poisoning by this agent is somewhat less satisfactory than that for GB or VX.

0415 The regime for nerve agent poisoning includes pre-treatment, decontamination and self-aid, first aid, and medical treatment. These are discussed below.

Pre-treatment

0416 Efficient pre-treatment reserves a portion of cholinesterase in blood and peripheral tissues in an unstable equilibrium and an adequate level of reservation is achieved when the activity of red cell cholinesterase is reduced by between 20 and 40% of its original level. This level of reservation of the enzyme is obtained by a dose of 30 mg of pyridostigmine bromide by mouth every 8 hours. It is essential that the dosage time is strictly adhered to if the optimum level of protection is to be maintained as the percentage of reservation falls significantly after 8 hours. The standard issue pack Nerve Agent Pre-treatment Set (NAPS) contains 7 days' supply of pyridostigmine. Pre-treatment is an operational command decision, but, in order to protect against slow absorption through the skin, it should commence immediately a chemical threat is declared and be continued for 24 hours after all risk of such attack has ceased. Good protection is obtained within 2½ hours of the first dose but it is not optimal until the third dose. No side-effects have been noted after extensive volunteer trials, but interaction with muscle-relaxant drugs used in anaesthetics may occur. See paragraph 1002.

Decontamination and self-aid

0417

(a) *Decontamination.* This should be undertaken at once by dabbing the Decontamination Kit, Personal on all areas of contaminated skin.

(b) *Triple therapy.* The Autoject Mk III L2A1 Combopen contains 2 mg atropine sulphate and 500 mg pralidoxime mesylate, and in the safety cap, a 5 mg tablet of diazepam. The treatment drill is swallowing the tablet and self-injection of the autoject needle through the clothing into the outer half of the thigh. The breath must be held when raising the respirator to swallow the tablet.

First aid

0418 First aid to a casualty who cannot help himself must include the decontamination of any areas of contaminated skin and the casualty's respirator, plus the use of his own autojects; the diazepam tablet must only be given if the casualty is conscious and able to swallow. Intermittent positive pressure ventilation should be given to casualties who are not breathing. Automatic or manual lung inflation equipment should be used if available. The casualty and the ventilation equipment should be decontaminated as soon as possible. If no such equipment is available, expired air ventilation should be used provided the atmosphere is non-toxic and the casualty's mouth has been decontaminated. Chest compression/arm lift methods are unlikely to be effective but may be tried if no other methods are available. In this event the respirator should be left on the casualty's face. For further details see Section 9.

Medical treatment

0419 Full medical treatment cannot be carried out in a contaminated environment, but where possible the following regime should be practised:

(a) Complete decontamination of the casualty. Subject to life-saving requirements, this will consist of removal and safe disposal of clothing and decontamination of the skin.

(b) A clear airway and adequate ventilation must be maintained. Excessive bronchial secretions will require postural drainage or aspiration. Aspiration should be by endotracheal intubation, if the casualty's condition permits.

(c) Atropine must be given until certain of its effects can be seen clinically. The required degree of atropinization is indicated by a dry mouth and a heart rate of 70—80 per minute. (Mydriasis and miosis are unreliable signs after nerve agent poisoning.) This state should be maintained for 24 hours at least, preferably until the heart rate without atropine is at least 70 per minute. The drug is conveniently administered in 2 mg doses intravenously. Repeated doses will be needed as indicated by the pulse rate, the interval between the doses ranging from a few minutes at first to several hours. Very large total doses, of the order of 200 mg may be required in cases of severe poisoning. Particularly when oximes have been given, the medical officer should be alert to the signs of atropine

poisoning, which are a combination of central and peripheral nervous effects. The central action may produce euphoria, hallucinations, anxiety, restlessness, excitement and delirium followed in severe cases by coma and depression of respiration. The more obvious peripheral effects are rapid pulse, dry mouth and throat, and dry hot skin. There may be hyperpyrexia. Urinary bladder dysfunction and retention of urine should be watched for and the patient catheterized if necessary. A special preparation of atropine containing 2 mg in 1 ml ampoules is available for treatment of nerve agent or organophosphorus pesticide poisoning and this should be given by ordinary syringe and needle. Atropine may also cause ventricular fibrillation in the hypoxic or hypothermic heart and therefore the intramuscular route is recommended except when intensive care facilities are available.

(d) Pralidoxime mesylate should be given concurrently in a dose of 1 g intravenously every hour up to four injections. If the casualty's condition allows, oral dosage should be maintained. Oxime in ampoules, each containing 1 g in 6 ml, is available for treating nerve agent or organophosphorus poisoning.

(e) Severely poisoned casualties showing fasciculation, twitching or convulsions, or those with marked anxiety or depression should be given intravenous or intramuscular diazepam.

(f) If circumstances permit, treatment of severe nerve agent poisoning by curarization and intubation, after admission to hospital, should be considered.

(g) Treatment of ocular symptoms. The instillation of 1% atropine eye drops or the application of 0.5 — 1% atropine ointment to the eyes is more effective than parenteral atropine in relieving headache and ciliary spasm. It should be noted that the use of mydriatics does not improve vision.

(h) Nerve agent poisoning may induce cardiac arrhythmias such as supraventricular techycardia and heart block. Myocardial failure may give rise to pulmonary oedema. These complications should be treated in accordance with established practice.

Special care in the tropics

0420 Since atropine inhibits sweating, extra care is required in tropical climates. Atropinization is still necessary in treating nerve agent casualties, but the possibility of heat effects must be borne in mind.

Course and prognosis

0421 The outlook depends upon the amount of agent absorbed and on the promptness and efficiency of remedial measures. Life can often be saved by treatment even though many times the lethal dose has been absorbed. The function of the respiratory centre and muscle power can return within 3—4 hours. However, reports of accidental severe organophosphate intoxication indicate that the danger of sudden reparalysis is present for up to 10 days. Such cases may require close medical supervision for an extended period before recovery is complete. Heightened susceptibility to further exposure will be present for some weeks; tolerance does not develop.

5 Lung-damaging agents (choking agents)

Introduction

0501 The most important member of this group is phosgene. It was used with great effect in World War I when it accounted for some 85 % of the deaths attributable to chemical agents. Other members of the group also used in World War I are chlorine (see paragraph 1506) and chloropicrin (see paragraph 1505). Cyanogen chloride and bromide, which are classed as miscellaneous agents and discussed in Chapter 8, produce some effects similar to the lung-damaging agents. Oxides of nitrogen contained in smoke from burning buildings, tanks, aircraft and ships are potent lung-damaging agents, as is zinc chloride, a constituent of some screening smokes (see Chapter 14). Since the action of phosgene may be regarded as typical of that exerted by other members of this group it is the only agent discussed in this chapter.

Physical and chemical properties of phosgene

0502 Phosgene (carbonyl chloride) (CG) is a colourless gas readily condensed by pressure or low temperatures to a colourless liquid with a boiling-point of 8 °C. It has an odour resembling that of new-mown hay. Phosgene reacts rapidly with water to yield non-toxic hydrolysis products.

0503 Although phosgene is a non-persistent agent, the vapour is somewhat heavier than air. It may, therefore, remain in dangerous concentrations in trenches, bunkers, valleys and woods for some considerable time, depending on the atmospheric conditions.

Detection

0504 There is no device available for detecting this agent.

Protection

0505 The Service respirator gives protection against this agent.

Decontamination

0506 Because of its physical properties, the agent will not remain long in its liquid state and decontamination is not necessary.

Mechanism of action

0507 Phosgene increases the permeability of the alveolar capillaries with resultant pulmonary oedema. This interferes with pulmonary gaseous exchange, leading to anoxia. The loss of fluid into the alveoli also results in haemoconcentration which, together with anoxia, causes cardiac embarrassment which may proceded to cardiac failure.

Pathology

0508 The outstanding feature of phosgene poisoning is massive pulmonary oedema. This is preceded by damage to the bronchiolar epithelium, 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.

Signs and symptoms

0509 During the latent period which may last from 30 minutes to 48 hours or more, the casualty suffers little discomfort and has no chest signs. He may feel well enough to continue normal duties. Thereafter, in those developing severe pulmonary damage, progressive oedema develops rapidly with shallow rapid respiration, cyanosis and a painful paroxysmal cough producing increasing copious expectoration of frothy white or yellowish liquid. Examination of the chest reveals increasing diminution of breath sounds with rales and rhonchi throughout the lung fields. The distress, apprehension, dyspnoea and cyanosis increase; the intense

loss of fluid causes hypovolaemia which together with progressive pulmonary damage and worsening hypoxia leads to circulatory failure and death. When possible blood gases should be monitored.

Treatment

0510(a) *Supportive therapy.* This should include wherever possible warmth and rest; rest during the latent stage is very important since any activity between exposure and the onset of pulmonary symptoms and or signs will greatly increase the likelihood of death. Coughs should be suppressed using codeine phosphate 30—60 mg; oxygen, humidified if possible, should be given as required. Morphine may be useful in the presence of shock, but should be used sparingly to avoid respiratory depression. As there is the danger of increasing lung damage, artificial positive pressure ventilation should only be given if the casualty is unable to ventilate himself adequately; positive end expiratory pressure may help in dealing with established pulmonary oedema. Diuretic therapy may be of some assistance.

(b) *Specific.* Steroids can be life-saving in poisoning by lung-damaging agents, but they must be given promptly and expectantly. Once pulmonary oedema has set in, these drugs are far less effective. Irrespective of whether or not pulmonary signs or symptoms are present, treatment using a steroid inhaler should commence as soon as possible after exposure; ideally within 15 minutes. The initial dose is five times that conventionally used in asthma, followed by about half of this dose for 12 hours and then the standard asthma dosages during the subsequent 72 hours until the risk of pulmonary oedema has passed (a list of steroid inhalers currently available in the UK is given in Table 5—1). Systemic steroids should also be started as soon as possible after exposure commencing with 2 g intravenously or intramuscularly of methyl prednisolone or an equivalent, repeated after 6 and after 12 hours. Thereafter this dose should be given 12 hourly for 1—5 days depending on the same criteria as for steroid inhaler therapy. Any side-effects of this large dosage should be accepted provided it does not itself endanger life. Antibiotic cover to prevent secondary infection is advisable and fungal supra-infection will require appropriate therapy.

Table 5 — 1 Steroid inhalers currently available in the UK

Beclomethasone diprionate Becotide dose 50 μg/puff. Contents 200 doses
Bethamethasone valerate Bextasol dose 100 μg/puff. Contents 200 doses
Busesonide Pulmicort dose 200 μg/puff. Contents 100 doses
Beclomethasone diproprionate Becloforte dose 250 μg/puff, i.e., 5 × strength of Becotide (probably preparation of choice)

6 Vesicant agents

Introduction

0601 Blister or vesicant agents are likely to be used both to produce casualties and to force opposing troops to wear full protective equipment thus degrading fighting efficiency, rather than to kill, although exposure to such agents can be fatal. They can be thickened in order to contaminate terrain, ships, aircraft, vehicles or equipment with a persistent hazard. The vesicant agents include sulphur mustard (HD), nitrogen mustard (HN), the arsenical vesicants such as lewisite (L) (this may well be used in a mixture with HD), and the halogenated oximes whose properties and effects are very different from those of the other vesicants.

Sulphur and nitrogen mustards

Nature and properties

0602 Sulphur mustard is 2,2′-di(chloroethyl)sulphide (HD). In its unmodified state it is a liquid boiling at 217 °C and freezing at 14.4 °C, but it may be thickened with additives; it sinks in water leaving a thin film on the surface. Of the nitrogen mustards only HN3, which is 2,2′,2″-tri(chloroethyl)amine, is likely to be used in war; its vesicant properties are almost equal to those of HD. Both HD and HN3 readily penetrate the body as well as many materials including wood, leather, rubber and paint. These agents are very persistent in cold and temperate climates, and when thickened with rubber or methacrylate their persistence is greatly increased. In hotter climates persistence is decreased, but higher concentrations of vapour occur. In water these agents are eventually hydrolysed by degree to harmless products, but in strong solutions interactions of products occur; when in dilute solution, 60 % of the original concentration is hydrolysed in 24 hours, but in static water the low solubility leads to layer formation and contamination may persist

for months; alkalinity and oxidants increase the rate of hydrolysis. Nitrogen mustards are less readily hydrolysed than sulphur mustard. It is possible that mustards may be used in a mixture with lewisite. Hydrolysis of this mixture may leave toxic arsenical compounds present in water. Sulphur mustard has a smell resembling garlic and nitrogen mustard has a fishy smell; but as the sense of smell diminishes this cannot be relied upon for identification. Both agents have an oily appearance and vary from brown to colourless according to the degree of purity.

Detection

0603 These agents can be detected with Detector Paper No 2 Mk I, which turns blue on contact with vesicants (and with nerve agents) or Detector Paper No 1 Mk II, which turns red with vesicants (yellow with G-type nerve agents and green with V-type nerve agents). Vapour can be detected by CAM or RVD. In water, mustards can be detected in concentrations above 2 ppm by using the Water Testing Kit, Poisons.

Protection

0604 Ordinary clothing gives very little or no protection against mustards. When worn correctly, the Suit Protective NBC with Gloves NBC, Overboots NBC and Respirator NBC give protection against these agents in both the liquid and vapour states for at least 6 hours. The suit and gloves should be changed before 6 hours of exposure has occurred. It is recommended that the boots be lined with fuller's earth.

Decontamination

0605 Liquid agent must be removed from the skin as soon as possible. It must also be removed from personal and unit equipment to prevent a continuing hazard.

(a) Personal decontamination is best carried out with the Decontamination Kit Personal No 1. The pads when dabbed or rubbed on the skin release fuller's earth which soaks up the liquid agent and retains it by absorption. For larger areas and for personal equipment, the Decontamination Kit Personal No 2, a puffer bottle containing fuller's earth, is recommended. Waste pads and fuller's earth which have been used for this purpose must be assumed to be a contamination hazard. Operational decontamination, where possible, should be carried out

using the Decontamination Apparatus NBC Portable or by scrubbing with chemical agent decontaminant.

(b) Expendable items should be buried or burned. The danger to the immediate vicinity and downwind from toxic fumes given off during burning must be remembered. A quantity of chemical agent decontaminant should be included in burying to ensure destruction of the agent.

(c) Large items of equipment such as vehicles, aircraft or weapons are difficult to decontaminate because the agents are absorbed by paint and rubber and penetrate crevices such as screw threads. If time allows, attempts at decontamination may be made with the chemical agent decontaminant or the Decontamination Apparatus NBC Portable, but the usual policy will be to use such contaminated equipment relying on personal protection.

Mechanism of action

0606 Mustards are powerful alkylating agents and react with amino, thiol, carboxyl, hydroxyl and primary phosphate groups; DNA is the most sensitive target in cells, so that mustards mimic the effects of ionizing radiation. Cytostatic, mutagenic and cytotoxic effects occur and actively proliferating cells in the haemopoietic and gastro-intestinal systems are especially vulnerable. Following exposure, irreversible binding takes place within 3—5 minutes.

Toxicity

0607 Mustards are insidious in action and cumulative in effect: they attack the skin, eyes and respiratory tract, or if swallowed, the gastro-intestinal tract. Repeated exposures may be expected to produce more severe effects on a cumulative basis; mustards are also potent sensitizers.

Pathology

0608

(a) **Skin.** The effects may be divided into four stages, namely, a latent period, erythema, vesiculation and finally necrosis. Blistering occurs in all but the most trivial exposures and in more severe cases the necrosis extends into dermis (see Plate 6). The damaged tissues are highly susceptible to secondary infection and fibrosis occurs in the healing process. Regeneration of these tissues is extremely

slow because of the radiomimetic effect of the agent on cell nuclei and at higher concentrations by the direct action of sulphur mustard on other cell constituents.

(b) **Respiratory system.** The mucous membrane of the whole respiratory tract may be involved with serious effects. Initially, there is inflammation followed by necrosis. Desquamation of the epithelium together with coagulated exudates results in the formation of a pseudo-membrane. Removal of this thick yellowish slough reveals an erythematous granulating surface (see Plate 3). Microscopy reveals pulmonary oedema and congestion of the bronchial and alveolar capillaries. There is necrosis of mucosal cells with desquamation into the lumen associated with infiltration by inflammatory cells. Obstruction of the smaller bronchi and bronchioli results in localized areas of atelectasis and compensatory emphysema which may be extensive and give rise to pneumothorax. Secondary infection causing bronchopneumonia is common.

(c) **Alimentary tract.** If liquid agent is swallowed there is congestion along the whole tract. The most severe damage occurs to the oesophageal and gastric mucosa causing necrosis which may lead to perforation.

(d) **Systemic effects.** These agents can cause pathological manifestations in distant organs, the most notable being the bone marrow and lymphoid tissues. Initially, there may be transient neutrophilia but subsequent depression of the bone marrow results in neutropenia which may be followed by thrombocytopenia and anaemia. In severe cases of hypoplasia of the bone marrow, the pale yellow areas in the long bones extend into areas normally occupied by active red marrow, e.g., the ribs, vertebrae and sternum. Hypoplasia of the lymphoid tissue is shown by the lymphopenia.

Signs and symptoms

0609

(a) The effects of vapour and liquid agent on the skin and eyes differ only in degree. Although all effects are delayed on onset, those from liquid appear sooner than those from vapour and are more severe. Vapour may be inhaled causing delayed pulmonary irritation and proceeding to effects comparable with lung-damaging agents; if the agent

is swallowed, delayed nausea, vomiting and purging occur.

(b) Mustards readily penetrate the skin and mucous membranes, causing delayed irritation and smarting followed by erythema and vesication; secondary infection and ulceration may ensue. There is evidence to suggest that mustards spread further and penetrate more deeply when the skin is dirty and greasy or hot and sweaty.

(c) Slow healing is a feature of mustard poisoning. Possible sequelae include permanent blindness of visual impairment, cutaneous scarring, chronic bronchitis, bronchial stenosis and sensitivity to further exposure to mustards. The mutagenic effects suggest that malignant sequelae may occur even after acute exposure, and mustard has been incriminated for malignancies occurring after prolonged industrial exposure to low concentrations.

The eyes

0610 There is no immediate irritation from either vapour or liquid agent in the eyes. The first symptoms, which develop after an hour or more, are a smarting and watering of the eyes with the development of a severe conjunctivitis. Severe burning pain with photophobia and blepharospasm may follow and oedema of the eyelids closes the eyes. There is a watery discharge from between the eyelids (see Plate 4); this may become profuse and purulent with the development of secondary infection and temporary adherence of the eyelids. The conjunctivae become hyperaemic and the corneae oedematous. Iritis and posterior synechiae may develop. Convalescence tends to be prolonged, being hindered by the persistence of photophobia, lachrymation and blepharospasm. In most cases recovery is complete. In severe cases, vascularization of the corneae and secondary oedema set in subsequent to the primary oedema or after it has subsided. Such changes, occurring where there has been prolonged exposure to vapour or direct contamination with liquid, are serious as they may recur for months or even years, accompanied by some impairment of vision which becomes worse with each attack (see Plate 7).

The skin

0611 The effects of sulphur mustard on the skin are greatly influenced by both the severity of exposure and the prevailing weather conditions. Hot humid weather markedly increases the rapidity and degree to which the agent attacks the warm moist

skin of the perineum, genitalia, axillae, antecubital fossae, and the face and neck. Again there is no immediate effect. After a delay of some hours, or possibly days, the first sign to develop is erythema at the site of contact. Vesication follows as a result of an inflammatory exudation of fluid which may continue for several days according to the depth of penetration, and which leads to the formation of tense serum-filled vesicles. In the case of liquid contamination, adjacent vesicles may coalesce to form large areas of blistering (see Plate 5). The blisters are not in themselves painful, except where they occur on the more sensitive areas of skin such as the penis and scrotum, or where they occur near joints and are involved in movement. In appearance they resemble thermal burns and like these become infected very easily. World War I experience indicates that healing of these skin lesions is particularly slow (see Plate 2). In some cases a transient recrudescence of erythema occurs some 10 days after healing. The scar following a sulphur mustard burn may be thin, soft and depigmented.

The respiratory tract

0612 The first symptoms may develop several hours after exposure when the nose begins to run and there may be sneezing as in an early head cold. Dryness and a burning sensation in the throat progress to a hoarse voice and a harsh dry cough. After severe exposure the cough and laryngitis worsen over the next 24-48 hours, gradually proceeding to bronchitis. There is an abundant expectoration of muco-pus in which may be seen sloughs of the tracheal lining. With necrosis and secondary infection throughout the respiratory tract, bronchopneumonia develops. In such cases death can follow in a few days, but may be delayed for 2 or 3 weeks. Where leucopenia develops, the outlook is particularly grave.

Treatment

0613

(a) **First aid.** First aid, or self-aid, should commence with immediate decontamination using fuller's earth on the skin and washing the eyes with plenty of water (2 % sodium bicarbonate is preferable if available). Affected areas of skin should be covered with dry dressings after the application of dimercaprol ointment (British Anti-Lewisite (BAL)) because of the likelihood of a mustard-arsenical mixture being used. It should be noted that ointment silver sulphadiazine (Flamazine) used for thermal

burns is pharmacologically incompatible with BAL and their combined use is therefore contra-indicated.

(b) **Medical.** This is basically symptomatic but there is evidence that the lesions may be reduced in severity if intravenous sodium thiosulphate is given within 30 minutes of exposure, the recommended dose being 35 g (500 mg/kg). There is no good evidence that steroids either locally or systemically benefit cutaneous or ocular damage.

(c) **Specific treatment:**

(1) *The eyes.* These are the most vulnerable to injury by mustards. The merest suspicion of ocular contamination calls for the regime described under first aid above. Any delay at all may result in severe damage which may be aggravated by further washing. Fuller's earth must NOT be used in the eyes.

(2) *The skin.* Infection is the most important complicating factor in the healing of mustard burns, and antibiotics should be given as early as possible. Healing in any case is slow and the prompt first aid measures described above are the mainstay of treatment. Later treatment is similar to that of thermal burns except that Flamazine should not be used.

(3) *The respiratory tract.* Because of the possible delay in clinical manifestations of pulmonary damage and the inability to asess the amount of vapour inhaled, the immediate use of inhaled steroids is recommended as described in sub-paragraph 0510b, although the efficacy of this treatment has not been confirmed. The severe effects of inhalation, pulmonary oedema, respiratory tract obstruction and infection will require appropriate treatment.

(4) *The haemopoietic system.* Marrow damage may result in aggranulocytosis or aplastic anaemia. Although these conditions may require blood transfusion, marrow stimulants or marrow transplant, and nursing in an infection-free environment, it will be appreciated that facilities for these regimes may not be available; even if they are available circumstances may prevent their use.

Arsenical vesicants

Introduction

0614 Arsenical vesicants include lewisite (L), which is 2-chlorovinyl-dichlorarsine, and methyl-, ethyl-, and phenyldichlorarsines, the last three being known as the Dicks. They are all oily liquids, more volatile than sulphur mustard, and varying in appearance from clear and colourless to dark brown. Some, but not all, samples of lewisite have a geranium-like smell, whereas the Dicks smell more fruity. All these agents are poorly soluble in water but dissolve in organic solvents. Hydrolysis in water leaves a vesicant oxide, but alkalis and oxidants break these agents down to non-vesicant compounds, although toxic amounts of arsenic may remain.

0615 The vesicant effects of arsenical agents are similar to those of the mustards, except that the onset of discomfort and pain is immediate. Systemic arsenical poisoning may follow injury by these agents.

Signs and symptoms

0616 Contact with the liquid agent, unlike contact with sulphur mustard, results in immediate pain in the eyes or skin; the vapour is strongly irritant to both the eyes and respiratory tract. Erythema, vesication and eye injury develop more rapidly than with sulphur mustard, the effects being severe within 4—8 hours. The pathology is similar to that of mustard poisoning, but arsenic can be detected in the lesions and tissues.

Treatment

0617 This should follow the principles outlined for the treatment of sulphur mustard casualties. In addition, specific treatment with dimercaprol (BAL) should be instituted. This preparation should be used locally as an ointment for the eyes and skin, but it should not be used in combination with silver sulphadiazine (Flamazine) (see paragraph 0613). (Because BAL ointment chelates the silver in Flamazine the two ointments cannot be used as a mixture for thermal burns contaminated by arsenical agents.) In the case of systemic absorption it should be given parenterally by intra-muscular injection. Dimercaprol, as a 5 % solution in arachis oil with benzyl benzoate, is available for intramuscular injections; it should be administered by injection deep into the buttocks in doses of 2.5 mg/kg every 4 hours for four doses, followed by two doses per

day according to the casualty's condition. Intramuscular injection of BAL is very painful and causes muscle necrosis.

Phosgene oxime

0618 Phosgene oxime or its homologues may be used in a mixture in order to produce intensive irritation and pain in the face and eyes thus preventing the donning, or inducing the removal of, the respirator; it thereby causes a lethal or incapacitating agent to be inhaled.

Signs and symptoms

0619 The action on the skin is immediate with severe irritation or, in all but minor contamination, severe pain which increases rapidly and radiates from the area of direct contact. With 30—60 Seconds the affected area turns white and is surrounded by an erythematous ring. After about 1 hour the area appears swollen, often showing a central wheal. Within 24 hours the area acquiires a brown pigmentation and necrosis of the skin occurs, accompanied by blistering at the periphery. An eschar forms after about 1 week and sloughs off during the next 6 weeks. Complete recovery may taken up to 3 months. If the eyes are contaminated, intense irritaion and pain occur and corneal lesions may result in permanent blindness. Respiratory exposure results in painful coughing and bronchial hypersection. Pulmonary oedema may occur.

Treatment

0620 The affected skin and eyes should be flushed with water (2% sodium bicarbonate if available). Any necrotic lesions should be treated as deep thermal burns. Steroids are only likely to be of benefit after exposure of the respiratory tract, in which case the regime outlined in sub-paragraph 0501b should be used.

7 Incapacitating agents

Introduction

0701 Incapacitating agents are substances that impair the subject's ability to perform duties without causing serious risk of death or permanent injury. Lethal agents in sub-lethal doses and blister agents, both of which may cause permanent injury, and riot control agents are excluded from this category.

0702 Incapacitating agents are classified as physical incapacitants or psychotomimetic agents according to whether their action is predominantly upon the physical or mental activities of the victim.

Physical incapacitants

0703 Possible mechanisms of physical incapacitation are many, but the criterion that no serious risk of death or permanent injury should result means that no practical physical incapacitant is known at present, although the vomiting agent DM (see Chapter 9) is described as a physical incapacitant in the Service classification (see Table 1—1).

Psychotomimetic agents

0704 There are many drugs which act upon the central nervous system to produce incapacitation; few of these are sufficiently potent and safe, or possess the necessary chemical and physical properties, to make them potential chemical agents. Of these few, BZ, an atropine-like drug, is the most important but lysergic acid diethylamide (LSD 25) and other similar drugs merit consideration.

BZ

Physical and chemical properties

0705 BZ is a crystalline solid at normal temperatures and sufficiently stable to be disseminated as a smoke from a pyrotechnic device. Other members of this glycolic acid ester series may be liquid at usual temperatures and may be absorbed through the skin.

Detection

0706 There is no device at present available for detecting this agent.

Protection

0707 Protection is given by the Service respirator and Suit Protective NBC.

Mechanism of action

0708 BZ acts by blocking the activity of cholinergic synapses in a manner similar to that of atropine. Unlike atropine, BZ produces predominantly central rather than peripheral effects.

Signs and symptoms

0709 In 1—2 hours after exposure, BZ produces atropine-like effects such as dilation of the pupils, dry mouth and increased heart rate, followed later by ataxia and drowsiness. These effects, apart from the mydriasis, give way after 6 or 7 hours to a confused mental state in which delusions, hallucinations and aimless behaviour are common and may persist for several days. During this phase the subject may injure himself and others. Memory for the period of the intoxication may be lost or fragmentary. The mydriasis may persist for 3 days.

Treatment

0710 In most casualties, symptomatic treatment is all that will be necessary. Firm restraint when necessary and a friendly attitude are called for especially in dealing with those subjects who are capable of walking. All dangerous objects must be removed and anything likely to be swallowed should be kept away from the subject as bizarre delusions may occur. Body temperature should be observed as heat stroke may occur, especially in tropical climates.

Fluid intake must be maintained. Physostigmine, which is used as an antidote to BZ, should be reserved for casualties who appear to be in danger. Where this treatment is deemed to be necessary, an injection of 2—3 mg will be required to alleviate the condition. Repeated injections at intervals of 15—30 minutes may be required to avoid relapse. Neostigmine and pyridostigmine do not penetrate the blood brain barrier and are considerably less effective.

Lysergic acid diethylamide (LSD)

Physical and chemical properties

0711 LSD is solid at normal temperatures and is soluble in water. It is a very difficult agent to disseminate and consequently is likely to be used only in a clandestine manner.

Detection

0712 There is no device available for detecting this agent in the field.

Protection

0713 No protection is available against clandestine attack, but it seems that only small quantities of food or water could be contaminated.

Mechanism of action

0714 Very small doses (e.g., 50 μg per man) are capable of inducing a psychotic state in man, but the precise mechanism of action is not yet known.

Signs and symptoms

0715 The clinical manifestations of LSD intoxication often include an early stage of nausea followed 45—60 minutes after dosage by a confused state in which delusions and hallucinations are common, but not always experienced. There is some evidence that the effects may be held off, at least for a time, by determination to continue duty and that the presence of undrugged comrades enables affected subjects to maintain contact with reality. Recovery is spontaneous and is usually complete within 12 hours.

Treatment

0716 The best treatment known at present for LSD intoxication is the administration of diazepam 10—20 mg intravenously or intramuscularly, or sodium amytal 200—400 mg intravenously to sedate the patient until spontaneous recovery occurs. Chlorpromazine has also been suggested for therapy but does not appear to have any advantage over these drugs.

Other drugs

0717 The phenothiazines and cannabinols, although they seem to act primarily by depressing the central nervous system, are not considered likely to be used in warfare owing to the relatively large doses required to produce an effect.

8 Miscellaneous agents

Introduction

0801 There are a number of agents which do not readily lend themselves to pharmacological classification; these include cyanide-type agents and arsine. The cyanide-type agents are discussed in this chapter, arsine is discussed in Chapter 15. The use of cyanide agents was initiated by the French in 1916 with the employment of shells filled with hydrogen cyanide. Because of its extreme volatility and the fact that the vapour is lighter than air, it was found almost impossible to establish a lethal concentration in the field by this means of delivery. In an attempt to overcome this disadvantage the related substances cyanogen chloride and cyanogen bromide were produced, the vapours of which are several times heavier than air. With modern weapon systems, it is certainly possible to produce a lethal field concentration of hydrogen cyanide and knowledge of the effects of this agent is essential.

0802 The agents in this group, known also as blood agents, are:

(a) Hydrogen cyanide.

(b) Cyanogen chloride (CNCl).

(c) Cyanogen bromide.

The two latter compounds, after absorption from the lung, react with haemoglobin in such a way that hydrogen cyanide is eventually released. Their effects on the body, therefore, are essentially similar to those of hydrogen cyanide. The rest of this chapter deals with hydrogen cyanide but the main points of difference from the other two agents are indicated.

Hydrogen cyanide

Physical and chemical properties

0803 Hydrogen cyanide (AC) is a clear, colourless liquid with a boiling point of 26 °C. It is very volatile and the vapour, being lighter than air, disperses rapidly after release. It has a smell of bitter almonds which may be noticed in concentrations as low as 1 ppm. This is well below the danger level, but is unreliable as a means of detection. Hydrogen cyanide is soluble in water producing a weak acid solution.

Detection

0804 The Nerve Agent Inhibited Enzyme Alarm and Detector (NAIAD) is an automatic device available to the Services for detecting concentrations of these agents in the vapour state; a Draeger tube can also be used. Cyanide in water can be detected in a concentration of 20 ppm using the Water Testing Kit, Poisons.

Protection

0805 Protection against these agents is given by the Service respirator and the Suit Protective NBC. However, these agents seriously impair the effectiveness of the respirator filter which should be changed after 20 minutes exposure. The protective suit is necessary for full protection since the agents, in their liquid state, can be absorbed through the skin.

Decontamination

0806 Because of its physical properties the agent will not remain for long in its liquid state. Decontamination should not, therefore, be necessary.

Mechanism of action

0807 The cyanide ion forms a reversible complex with the respiratory cytochrome oxidase enzyme system which results in impairment of cellular oxygen utilization. The central nervous system, particularly the respiratory centre, is especially susceptible to this effect, and respiratory failure is the usual cause of death.

Pathology

0808 With exposure to high concentrations, sufficient hydrogen cyanide may be inhaled in a few breaths to cause immediate death by respiratory failure. In these cases no pathological changes are demonstrable. The blood remains well oxygenated and the skin has a flesh-pink colour, less bright than that of carbon monoxide poisoning, but may be grey or cyanosed. In cases where death is delayed following exposure to lower concentrations, small areas of haemorrhage and softening of the brain may be seen due to anoxic damage. Where exposure is to sub-lethal concentrations, cyanide is detoxicated in the body to harmless thiocyanate. This reaction is catalysed by intracellular trans-sulphurase enzymes, one of which requires thiosulphate as a substrate. The limiting factor in cyanide detoxication is the amount of available intracellular reducing sulphur which can serve as, or be transferred into, substrate. This reducing sulphur is available, *in vivo* in the form of thiosulphate, cystine and cysteine. In addition to the systemic effects outlined above, cyanogen chloride and bromide also have local irritant effects on the eyes and the respiratory tract similar to those of the choking agents. There may be severe inflammatory changes in the bronchioles with congestion and pulmonary oedema.

Signs and symptoms

0809 The more rapidly the tissue cyanide levels build up, the more acute are the signs and symptoms of poisoning and the smaller is the total absorbed dose required to produce a given effect. In high concentrations there is an increase in the depth of respiration within a few seconds. This stimulation of respiration may be so powerful that a casualty cannot voluntarily hold his breath. Violent convulsions occur after 20—30 seconds with cessation of respiration within 1 minute. Cardiac failure follows within a few minutes. With lower concentrations the early symptoms are weakness of legs, vertigo, nausea and headache. These may be followed by convulsions and coma which may last for hours or days depending on the duration of exposure to the agent. If coma is prolonged, recovery may disclose residual damage to the central nervous system manifested by irrationality, altered reflexes and unsteady gait which may last for several weeks or longer; temporary or permanent nerve deafness has also been described. In mild cases there may be headache, vertigo and nausea for several hours before complete recovery. With cyanogen chloride and bromide, the above systemic effects are modified by their irritant properties.

Exposure is followed by intense irritation of the eyes, nose and throat, with tightness of the chest and coughing. Severe lachrymation and blepharospasm may occur. Vertigo, headache and dyspnoea follow which may proceed to convulsions, coma and death. In non-fatal cases pulmonary oedema often develops which gives rise to a persistent cough with frothy sputum, severe dyspnoea and marked cyanosis.

Treatment

0810 (a) Successful treatment for acute cyanide poisoning depends upon rapid fixation of the cyanide ion, either by direct fixation with cobalt compounds or by methaemoglobin formation. Any casualty who is fully conscious and breathing normally more than 5 minutes after presumed exposure to cyanide agents has ceased will recover spontaneously and does not require treatment, cyanide being very rapidly detoxified in the body.

(b) The established treatment is dicobalt edetate which directly fixes the cyanide ion, but as this is toxic to liver and kidneys it must not be given to a patient who is still conscious 5 minutes after exposure has ceased, unless slow absorption from an ingested cyanide salt is suspected. It should be remembered that high concentrations of cyanide vapour and also solutions of cyanide salts are absorbed through the skin. The dose of dicobalt edetate is 300 mg in 20 ml of solution (Kelocyanor) given intravenously. Repeated doses may be needed if the casualty does not recover consciousness rapidly. Cobalt edetate should be followed by intravenous sodium thiosulphate given slowly in a dose of 25 g in a 50 % solution.

(c) An alternative treatment has been advocated using dimethyl aminophenal (DMAP), although this drug is not available in the UK and there is some concern over its possible mutogenic effect. This treatment induces methaemoglobinaemia with rapid fixation of the cyanide ion. DMAP is non-toxic and is given in doses of 250 mg in 5 ml intravenously: in an emergency it is effective intramuscularly at the expense of some muscle damage. DMAP should be repeated until the casualty is fully conscious and should then be followed by an intravenous injection of sodium thiosulphate 25 g in a 50 % solution. It should be remembered that methaemoglobinaemia which is essential to this method of treatment will itself cause a cyanotic appearance.

(d) If no other method of treatment is available amyl nitrite may be used, the ampoule being crushed and placed under the mask of the resuscitation device which is used according to instructions.

(e) Oxygen should be given if available, but amyl nitrite must not be used under an oxygen mask owing to the risk of explosion.

(f) Cyanogen halide poisoning should be treated in the same way as cyanide poisoning as regards its cyanide-like effects. Pulmonary irritation should be treated in the same way as phosgene poisoning (see Chapter 6).

Section 4

Agents likely to be encountered in riot control and/or warfare

9 Riot control agents (including vomiting agents)

Introduction

0901 Riot control agents are chemicals which produce irritating or disabling effects when in contact with the eyes or when inhaled. They are distinguished from all other chemical agents by their extremely short duration of action, minimal toxicity and their activity in low concentrations. Sensory irritant agents are used in riot control and also in training, but the arsenical vomiting agents may be used in war.

Sensory irritant agents

General

0902 Earlier agents such as chloracetophenone (CN) and chloropicrin, which were capable of producing corneal lesions and even pulmonary oedema in very high concentrations, have been superseded in the West by ortho-chlorobenzylidene malanonitrile (CS), although the earlier agents remain generally available. CS has a very high margin of safety. Another compound, dibenzoxazepine (CR) has been considered, but not used.

CS

Toxicity

0903 The toxicity of CS is very low, the estimated lethal concentration for man over 1 hour being 1,000 mg/m^3, whereas a concentration of 1 mg/m^3 is intolerable to most people.

Treatment

0904 Normally treatment is not necessary, since recovery is rapid and complete when individuals escape from the irritant atmosphere or are put on a respirator. To hasten recovery, victims should

whenever possible move to fresh air, separate from fellow sufferers, face into the wind with eyes open and breathe deeply. Very severely exposed casualties may require active treatment, which should be carried out as follows:

(a) *The pulmonary system.* Tracheitis and bronchitis may result in marked pain or severe cough. A cough sedative linctus may be used to control such symptoms. Individuals who suffer from pre-existing pulmonary or cardiac disorders may suffer from an exacerbation of these. Treatment should be along usual lines.

(b) *The cardiovascular system.* This is not affected directly, but the irritation and distress produced by CS may cause a rise in blood pressure which is usually slight and short-lived. Those already suffering from cardiovascular disorders should therefore be observed carefully with this transient hypertension in mind.

(c) *The central nervous system.* There is no direct effect on the central nervous system. Occasionally hysterical manifestations, such as hyperventilation, may occur and should be treated appropriately.

(d) *The skin.* CS should be removed at once with soap and water. Erythema alone will resolve in 24—48 hours without further treatment. Local vesication may occur after gross exposure to solid CS under moist conditions, and should be treated as a thermal burn. In very rare cases ulceration may develop; it should be treated with paraffin gauze dressings. A steroid-antibiotic cream may be helpful.

(e) *The eyes.* Severe exposure should be treated by irrigating at once with physiological saline or water. After irrigation, proxymetacaine hydrochloride 0.5 % (ophthaine) may be used to relieve pain. Amethocaine eye drops may also be used, but this is less satisfactory; in any case cocaine is better avoided because of its action on proteins. Tetracaine ointment will relieve severe blepharitis; antibiotics may be used to relieve secondary infection.

CR

0905 Dibenzoxazepine, commonly known as CR, is a yellow crystalline solid with a melting-point of 71 °C. Compared with CS, it is more stable in solution and is more potent. It is a highly

irritant compound, qualitatively similar in its effects and safety to CS. It is of low solubility in water, but aqueous solutions may be prepared with the aid of an appropriate organic co-solvent (such as polyethylene glycol or dipropylene glycol monomethyl ether). It is chemically stable in water. With the aid of a co-solvent, CR may be used in aqueous solution squirted from various types of liquid delivery devices. In solution, even 0.0025 % CR will produce marked irritant effects on the eyes and skin. The structures principally affected by liquid contamination are the eyes, skin, mouth and nasal cavity. CR may also be generated as an aerosol or a grenade smoke when its effects will be similar to those produced by CS aerosols or smokes.

(a) *The eyes.* Even small amounts of CR cause intense pain accompanied by blepharospasm and lachrymation. There is injection of the conjunctivae and slight blepharitis. The principal effects last for about 10 minutes, but injection of the conjunctivae and erythema of the lid margins may persist for some hours. A short-lasting rise in intra-ocular pressure is usually present during the acute phase, and those with established or incipient glaucoma may be at a risk. Structural damage to the eyes is unlikely to occur.

(b) *The skin.* Marked burning sensations and erythema occur within a few minutes of contamination; particularly sensitive areas are the face, shoulders, back of neck, trunk and external genitalia. Symptoms last for less than 30 minutes and die away spontaneously even in the presence of continued contamination; but they are readily reactivated on washing when they may be as severe as on initial contamination. The erythema is variable, but sometimes intense, and persists for a few hours. There has been no report of skin damage or allergic sensitization with CR. Cuts and abrasions may be affected more strongly than intact skin, but healing is unaffected.

(c) *The mouth and nose.* CR has an unpleasant taste and causes a burning sensation on the tongue and palate accompanied by a marked mucoid salivation. Quantities of material likely to be swallowed are small and do not constitute a toxicological hazard. Some individuals may complain of belching, short-lasting epigastric discomfort, nausea and, very occasionally, vomiting. There is usually a marked rhinorrhea and there may be transient, but sometimes distressing, shortness of breath.

(d) The principal systemic consequence of contamination by CR in solution is a rise in systolic and diastolic blood pressure. This is due to the uncomfortable nature of the experience and blood pressure usually returns to normal for the individual within 30 minutes. Those with established cardiovascular disease may constitute a risk group. The exact nature of the effects caused by CR, and their severity, depend on a number of factors including individual variation in susceptibility, the degree of contamination, and the solvent used. Devices intended to disseminate jets of CR in solution could be of many types, differing principally in the volume delivered and the operational range of projection.

Treatment

0906 In the acute phase, treatment consists principally of reassurance; little can be done to cut short the skin irritation but reassurance that it will abate spontaneously within minutes can be given. Pain in the eye can be relieved by proxymetacaine hydrochloride 0.5 % (ophthaine) or amethocaine eye drops. Decontamination should be by the removal of contaminated clothing which should be placed so that it cannot contaminate other individuals or objects, followed by thorough washing of the affected areas. It should be recognized that a temporary return of symptoms is usual on washing. Contact with the individual and his clothing should be limited; ideally gloves should be worn (the hands are not very sensitive to CR, but can readily convey irritant to other more sensitive areas). Residual contamination of roads and pavements should be washed away with liberal quantities of water; alternatively bleach may be used with subsequent washing. Clothes and contaminated objects should also be treated with bleach solution where possible, or burned. Because CR persists, contaminated articles should not be disposed of with normal rubbish. Unwrapped food contaminated with CR will not present a toxicological hazard but will have an unpleasant taste, and on this account should be disposed of.

Weapons systems

0907 The detailed description of the weapon systems used to dispense riot control agents is outside the scope of this manual, but it should be noted that systems at present in use for the purpose include the following:

(a) Cartridge, typically of large calibre (40 mm) and relatively low velocity (50—80 m/s), intended for firing from a Verey pistol or from specialized riot guns. These discharge a container of aluminium or cardboard which is itself a small pyrotechnic generator. Emission of the agent commences towards the end of the trajectory and continues on the ground for a further 8—20 seconds. In addition to the effects produced by CS there is also a remote possibility of ballistic injury which may result in contusions, or in extreme cases and at close range, in a severe penetrating wound. The healing process of either burns or open wounds is not likely to be adversely affected by the presence of CS.

(b) Grenades, which are of two types; those that discharge the agent as a smoke while the grenade remains intact, and those that discharge burning pellets of the agent in all directions when the rubber casing bursts. The pellets are small and do not adhere to skin or clothing, nor are they likely to produce impact injury to the eyes beyond half a metre or so.

Arsenical vomiting agents

General

0908 DM is typical of these agents and is the one discussed in this chapter. It is possible that arsenical vomiting agents may be used in conjunction with a lethal or incapacitating agent in order to hinder the donning of respirators or induce casualties to remove their respirators owing to persistent pain, sneezing or vomiting, and so causing exposure to the other agents.

Physical and chemical properties

0909 DM is a yellow crystalline solid with a melting-point of 195°C. It is odourless, insoluble in water, and stable on storage. It is disseminated as a smoke, which is canary yellow when concentrated but which gradually becomes colourless as it is mixed with air. DM is non-persistent and protection is given by the Service respirator.

Signs and symptoms

0910 DM is not immediately detectable by the senses and there is a delay of several minutes between exposure and the onset of symptoms. An effective dose is likely to be inhaled before the presence of the agent is suspected. Even if the respirator is put on as soon as the effects are noticed, symptoms will continue to increase in severity for several minutes. Unless he is aware of this possibility, the victim may believe his respirator to be ineffective and remove it resulting in further exposure to the agent. The symptoms consist of acute pain in the nose and sinuses with a sense of fullness in the head and headache. There is a burning sensation in the throat with tightness and pain in the chest, accompanied by violent sneezing and uncontrollable coughing. The eyes are affected with irritation, pain and lachrymation. Nasal secretions and salivation are greatly increased, and nausea and vomiting develop. Mental depression is marked in severe cases. Following exposure to field concentrations, symptoms disappear within several hours with no residual damage. Severe pulmonary injury and death have been reported following accidental exposure to high concentrations in confined spaces. For this reason DM is unsuitable for use during training.

Treatment

0911 Since the effects wear off completely within a short time, treatment will not normally be necessary. The respirator must be worn in spite of coughing, sneezing, salivation and nausea. When necessary the respirator should be lifted briefly from the face to permit vomiting or to drain accumulated secretions from the face-piece. Vigorous exercise will help to reduce the intensity and duration of the effects.

Section 5

Combined injuries

10 Combined injuries

Introduction

1001 In the chemical operations context, combined injuries are conventional injuries which are affected by chemical agents. Such injuries may be further complicated by the effects of nuclear or biological weapons: this aspect and that of conventional casualties contaminated by, but not suffering from the effects of, chemical agents are discussed in the *NATO Handbook on the Concept of Medical Support in a NBC Environment.* In this section we discuss the effects of poisoning by chemical agents, and the effect of drugs used to treat such poisoning, on the handling and treatment of conventional casualties. A summary of possible interactions is given in Table 10—1.

Table 10—1 Possible interactions chemical/conventional injuries

Chemical agent group	Conventional injury
Nerve agents (carbamate pre-treatment) (atropine)	Relaxant in anaesthesia Resuscitation Blood loss Shock
Vesicants (mustard and arsenicals)	Slow healing of wounds Haemopoietic depression Infection more likely
Lung-damaging agents	Resuscitation Blood loss Shock (latent period may be shorter)
Cyanogen agents	Resuscitation Blood loss Shock

Non-persistent agents

Injuries complicated by exposure to non-persistent nerve agents.

1002 The dangers presented by this form of combined injury are from the nerve agent itself, from the interaction of respiratory depression with the conventional injury and from the reduced cholinesterase activity upon the drugs used in anaesthesia during subsequent surgery: even carbamate pre-treatment may affect relaxants to a limited extent. Signs of nerve agent intoxication will call for treatment as described in paragraph 0414. Loss of blood will complicate respiratory failure so that the administration of oxygen, if available, and positive pressure resuscitation, if necessary, should be applied at the earliest indication of the need. The need for replacement of blood lost through conventional injury will be correspondingly greater if respiratory depression is present. Reduced cholinesterase activity will affect the use of relaxant drugs used during anaesthesia. On basic principles the action of anticholinesterases, including to a lesser extent pyridostigmine pre-treatment, may be expected to potentiate the action of depolarizing relaxants, such as succinyl choline, but to oppose the action of non-depolarizing relaxants of the current type.

Injuries complicated by exposure to lung-damaging agents

1003 This form of combined injury increases the stress element involved in the induction of pulmonary oedema. The latent period between exposure and the development of pulmonary oedema may possibly be shortened and the pulmonary oedema itself may be more severe. The casualty should be kept at rest as far as possible during evacuation and steroid treatment as described in sub-paragraph 1510b should be applied at the earliest opportunity. There is no contra-indication to the use of opiates or other systemic analgesics in order to treat pain or shock from the conventional injury. Oxygen therapy may be required but fluid replacement should be used with caution. The final decision on the necessity for fluid replacement must be made on the basis of the casualty's condition, bearing in mind the danger of increasing pulmonary oedema.

Injuries complicated by exposure to cyanogen agents

1004 Combined injuries of this type will present especial danger from respiratory depression and from the therapeutic reduction ofthe oxygen-carrying power of the blood, owing to the formation of methaemoglobinaemia in treatment. The need for treatment of

cyanide poisoning is urgent and treatment must be started in accordance with paragraph 0810 as soon as the airway has been secured and steps have been taken to arrest any severe haemorrhage. Oxygen therapy may be required, together with positive pressure resuscitation. This is particularly urgent in the presence of marked heamorrhage. If opiates are used, they must be used with caution.

Persistent agents

NB: Decontamination of any exposed surfaces is a matter of great urgency.

Injuries complicated by persistent nerve agents

1005

(a) Where the conventional injury is itself contaminated by a persistent nerve agent the danger of the casualty absorbing a lethal dose of nerve agent through the wound is very great and the prognosis is correspondingly bad. Although the wound track resulting from a conventional weapon injury is surrounded by devitalized tissue, there is rapid penetration of the tissues by nerve agent and a lethal dose may be quickly absorbed especially if a persistent agent contaminates the wound. Decontamination of the skin surfaces around the wound should be carried out using fuller's earth and then a surface dressing applied. The wound itself should be covered with material similar to that of the protective suit. Early surgical excision of the contaminated wound offers the best chance of success but autoject treatment should be started immediately the wound contamination is diagnosed and repeated as necessary. Surgery of the contaminated wound offers minimal danger to medical and nursing staff if gloves made of butyl rubber are worn. If these are not available then two pairs of latex rubber gloves should suffice if washed at short intervals in hypochlorite solution and changed frequently. The evacuation of casualties with combined injuries requires careful observation while on route to a surgical unit and autoject treatment continued if signs of poisoning persist or worsen. For conventional wounds not directly contaminated but with the

surrounding skin affected by a chemical agent, decontamination of the skin should be carried out and any poisoning treated as appropriate for the particular agent involved.

(b) Where the conventional injury is not directly contaminated, but skin absorption is thought to have occurred, skin decontamination should be carried out in the recommended way. Any signs of nerve agent intoxication should be treated as described in paragraph 0414. The casualty should be kept under as close observation as circumstances allow in case signs of delayed absorption of agent appear. If it is necessary to evacuate without medical supervision, consideration may be given to the use of one injection from the automatic injection device as a precaution against delayed absorption of nerve agent.

Injuries complicated by vesicant agents

1006 Vesicant agents will debilitate the casualty and will seriously delay the healing of any wound, even if the wound itself is not directly contaminated. A contaminated wound will be very slow to heal and will also lead to rapid systemic absorption of the agent. If a wound is contaminated with lewisite (immediate pain disproportionate to the severity of the wound is suggestive of this) therapy with dimercaprol (BAL) will be required at an early stage — see paragraph 0617. The area around the wound should be decontaminated and the wound dressed; the dressed wound should be protected with material similar to that of the protective suit. Opiates should not be withheld if the condition of the casualty calls for their use.

Treatment of wounds that are not contaminated

1007 Wounds that are not contaminated should be dressed in the usual way. They should then be covered with agent-proof material similar to that of the protective suit. Any pressure bandage considered necessary may then be applied over the protective covering.

Head wounds preventing use of the respirator

1008 These cases, once the wounds have been attended to and dressed, will require evacuation in a casualty bag or half bag (see paragraph 1212). In emergency, the casualty's head may be protected in a pervious blouse from a spare protective suit.

Section 6

Public Health aspects

11 Contamination of food and water

Introduction

1101 Food and water cannot be decontaminated easily. Supplies must be protected as much as possible. Food and water may be contaminated by chemical agents in the vapour, aerosol or liquid states. The most dangerous contamination is from nerve and blister agents since these are likely to be disseminated as liquids and are more readily absorbed by foodstuffs. Exposure to high concentrations of the vapours of other agents may make food unpalatable or unfit for consumption.

Food

Susceptibility to contamination

1102 Nerve and blister agents are readily soluble in oils and fats. Food with a high fat content can absorb large quantities of these agents when exposed to liquid or vapour which may diffuse throughout the material. Liquid agents will also penetrate foods of low fat content, rendering them dangerous, but foods of this type would probably not absorb significant quantities of vapour. Foods of high water content, contaminated by agents that are easily hydrolysed, may be made unpalatable by the formation of acid products of hydrolysis.

Protection of food

1103 Liquid or vapour may penetrate wooden and cardboard boxes, or paper wrappings, in sufficient quantities to make consumption of the contents dangerous. Sealed polythene will give good protection against vapour but is penetrated by liquids in minutes to hours depending on the thickness of the material. Only food sealed in impervious containers, such as tins, glass or glazed earthenware jars, and foil wrappings, is completely protected against chemical agents.

Decontamination

1104 When it is suspected that impervious containers have been contaminated, they must be thoroughly decontaminated before being opened (see Chapter 12). If it is suspected that other types of container have been contaminated the contents must be assumed to be contaminated. Where contamination is with liquid nerve or blister agent, the whole contents must be condemned. Certain food contaminated by chemical agent vapours can be rendered safe by exposure to the air followed by cooking (see Table 11—1). If there is any doubt that a particular food is safe it must be condemned.

Water

1105 Open water sources may become contaminated by direct chemical attack on an area or by the catchment of water from such an area. In either case, concentrations sufficient to produce casualties may result. Water from deep sources, such as springs or wells, is less likely to be contaminated. At present there is no practicable means of decontaminating water in the field. Military supplies must be protected whenever possible by storage in closed containers and by covering storage tanks. All known lethal chemical agents can be detected in water by using the Water Testing Kit, Poisons. Full instructions for the use of this equipment are contained within it.

Table 11—1 Effects of chemical agents on food

	High fat content (butter, fats, milk, cheese, meat, bacon, etc., and shell eggs)	Low fat, high moisture content (fruit, vegetables, sugar, salt, etc.)	Low fat, low moisture content (cereal, tea, coffee, flour, bread, rice, etc.)
Nerve agents *Liquid*	All foods to be condemned		
Vapour	To be condemned	Dry foods should be exposed to the air for 48 hours. Other foods should be washed with 2% sodium bicarbonate solution, peeled where applicable, and cooked by boiling	
Blister agents *Liquid*	All foods to be condemned		
Vapour	To be condemned	As for foods contaminated with nerve agent vapour	
Choking agents	Agents decompose rapidly on contact with water. Food should be washed with water where possible and exposed to the air for 24 hours. Food may be made unpalatable by acid products of hydrolysis		
Cyanide-type agents	Unlikely to produce dangerous contamination of foodstuffs		
Riot control agents	Food may be made unpalatable to the extent of being inedible		

Section 7

Decontamination

12 Decontamination

Introduction

1201 Decontamination is a difficult and lengthy process, so the risk should be minimized by keeping personnel and equipment under cover whenever possible. This will reduce the chance of contamination by direct attack or by pick-up from contaminated objects.

1202 Chemical agents of low volatility which have been disseminated in the liquid state can continue to present a hazard and cause casualties for days, weeks or even months. To minimize this hazard, decontamination of personnel and equipment must be carried out as soon as possible.

Decontamination of personnel and equipment

Stages of decontamination

1203 There are three stages:

(a) *Immediate decontamination.* This is the removal of chemical agent from exposed parts of the body after a liquid attack, and from those items of personal equipment which come into contact with the body. To be fully effective, decontamination of the skin must be completed within 5 minutes of contamination. The drill for carrying out immediate decontamination as soon as the operational situation allows is described in Annex A to this Chapter.

(b) *Operational decontamination.* The aim of this stage is to reduce the hazard from severe contamination of protective clothing caused by contact with contaminated equipment. It is a continuing process whenever time and the

operational situation allow. It consists particularly of decontamination of those parts with which contact is probable during use of the equipment.

(c) *Unit decontamination.* Here the aim is to remove the contamination completely from all the unit's equipment. This is a major task which cannot be performed while maintaining an operational role.

The usual policy will be to rely on personal protection and continue to fight using contaminated equipment (*fighting dirty*).

Methods of decontamination

1204 Decontamination can be effected by:

(a) Destroying the agent by chemical or physical means;

(b) Removing the agent by using solvents or absorbents, or by washing;

(c) Weathering.

In addition, the agent can be rendered harmless by sealing contaminated articles in impermeable containers or by burying them deep in the ground.

Destruction

1205

(a) *Chemical*

(1) Bleaching powder is issued as a general purpose decontaminant. It destroys all known chemical agents. It should be mixed to a slurry with water and applied by brushing on to the surface to be decontaminated. It must not be used dry because it catches fire if it comes into contact with certain agents.

(2) The Decontamination Apparatus NBC, Portable contains a decontaminant mixture composed of 5 % sodium dichloro-isocyanurate, 2.5 % sodium hydroxide and 0.9 % boric anhydride in water. This mixture destroys chemical agents and enables decontamination to be carried out with a great saving of time.

(b) *Physical.* Chemical agents are destroyed by heat, and burning is the best method of disposing of combustible items which are contaminated. Clothing, blankets and certain other items can be decontaminated by boiling. Both

these processes will cause toxic vapours and people in the vicinity and downwind must be adequately protected.

Removal

1206 Where the methods of decontamination outlined above are unsuitable, chemical agents can be removed by absorbents, by solvents or by washing.

(a) *Absorbents.* The Decontamination Kits Personal (DKP) Nos 1 and 2 contain fuller's earth which is a highly absorbent powder. When applied to contaminated surfaces, fuller's earth absorbs and retains chemical agents, rendering them harmless. These kits are issued to all personnel for carrying out immediate decontamination.

(b) *Solvents.* Petrol, kerosene and other organic solvents can be used to dissolve agents and remove them from contaminated surfaces. If this method is employed, disposal of the solvent must be such as to ensure that the agent does not present a further hazard as the solvent evaporates.

(c) *Washing.* When other methods of decontamination cannot be used, much of the contamination can be removed from surfaces by washing down with water. Hot water and detergent are more effective than cold water alone. Again it must be remembered that agents removed in this way are not destroyed, and the water must be disposed of by a method that does not lead to dangerous spread of contamination.

Weathering

1207 Items of equipment not required for immediate use may be left exposed to the elements to allow the contamination to decay. Decay is due to evaporation, washing by rain and hydrolysis, and may not be complete for days, weeks or months, depending on the weather. Equipment left in this way must be clearly marked with gas warning signs.

Sealing

1208 Small items that are contaminated and are destined for eventual destruction or decontamination can be rendered harmless, temporarily, by sealing them in an impermeable container. Gasproof paper sacks are issued for this purpose. Where items are

no longer required, they can be disposed of by burying. A liberal quantity of bleach slurry should be buried with the articles to ensure destruction of the agent.

Decontamination of casualties

General

1209 Casualties must be assumed to be contaminated if they have been evacuated from an area in which persistent agents have been used. They must be decontaminated thoroughly before they can enter collective protection or receive medical treatment, apart from first aid, oral therapy, or through-clothes injections. Without thorough decontamination there is a continuing hazard both to the casualty and to medical personnel.

1210 Where casualties are contaminated with liquid nerve agent, speed in decontamination is of the first importance, as is the administration of atropine and oxime if signs or symptoms of poisoning are present. These should precede even life-saving measures for traumatic injury, although the latter can be commenced before decontamination is complete.

1211 There is less urgency for decontamination when casualties are contaminated with liquid blister agent as there is no immediate danger to life. However, any undue delay will increase the severity of the chemical injury. Life-saving measures for traumatic injury can precede decontamination after due consideration has been given to the hazard to medical personnel and to the possible spread of contamination. In such cases thorough decontamination of medical personnel and the casualty must be carried out as soon as possible afterwards.

Casualty bags

1212

(a) The casualty bag is a full-length protective bag in which stretcher casualties who are unable to wear their respirators can be safely evacuated in a contaminated state or safely pass through a chemical environment during evacuation.

(b) The casualty half bag is a protector hood covering the head, face and upper body for similarly affected walking casualties.

(c) Both bags have transparent face-pieces.

Handling of contaminated casualties

1213 In all medical units concerned with the reception of contaminated casualties there must be a clear demarcation between dirty areas where they are first received, and clean areas to which they are moved when decontamination is complete. In dirty areas all personnel must wear full protective clothing and respirators. Decontamination of the casualty will involve:

(a) Removal of all contaminated blankets, clothing and equipment which should be set aside for decontamination;

(b) Decontamination of the skin by the application of fuller's earth, bleach paste or other suitable decontaminant, followed by washing with soap and water.

Annex A
Immediate action and decontamination drills

1. **General.** It is emphasized that individual action on attack must be without hesitation and entirely automatic. All the actions listed in the following paragraphs may be necessary when a NBC attack is suspected.

2. **Immediate action drill**

Action	Explanation
(a) **Stop breathing** – Remove spectacles if worn	It is imperative that breathing is stopped immediately there is the suspicion of an attack. Avoid taking an extra breath before beginning the masking drill
(b) **Put on the respirator** leaning forward and bending the head downward	(1) The respirator should be put on in 9 seconds (2) The amount of liquid contamination received on the face or on the inside of the respirator face-piece will be reduced by leaning slightly forward and bending the head downward (3) Shouting 'Gas! Gas! Gas!' will warn others and will help to expel any vapour that may be trapped in the respirator face-piece
(c) **Check the correct adjustment of all clothing and equipment**	Make sure your personal protection is complete. Check to see that all fastenings are closed and that no skin is exposed. Pay particular attention to the fit of the NBC hood. This detailed check is best completed by men working in pairs.

(d) **Check the detector paper** and look for any other indication of a liquid attack	Check the detector paper on your NBC suit and nearby equipment. Watch others for their reaction, for example, the local commander, gas sentry or nearby persons. This is necessary in case your personal detector was shielded in some way

The above action (sub-paragraphs 2a-d) must be taken for every chemical attack unless individuals are already fully protected.

3. **Immediate self-aid.** The effects of some chemical agents on the body develop very rapidly. Therefore, it is imperative that an individual takes immediate steps to avoid becoming a casualty if he recognizes that he has been exposed directly to the effects of chemical agents. Only masking takes priority over the following steps:

Action	Explanation
If symptoms occur:	After masking, if any symptoms of nerve agent poisoning are apparent or if they appear at any later time, immediately give yourself one injection (see note below)
(a) Inject the Combopen autoject	
(b) Take one dose of the diazepam tablet as follows:	
(1) Decontaminate the gloves and remove the container autoject safety cap	The autoject should be shielded by the body from falling spray. Care must be taken not to lose the tablet. The eyes are to be kept closed as far as is possible. The face-piece should not be displaced entirely from the face but moved only far enough to permit the swallowing of the tablet
(2) Take a few deep breaths to steady the breathing and hold the last one	
(3) Grasp the respirator outlet valve and pull the face-piece away from the face, upwards to give access to the mouth	
(4) Swallow the tablet	

(5) Quickly replace and secure the respirator

Note: If the symptoms still persist 15 minutes after the first injection, repeat injections and tablet swallowing up to a total of three at 15 minute intervals.

4. **Immediate decontamination.** After a liquid attack has taken place, and when freshly exposed paper remains unspotted, carry out immediate decontamination. Using the Decontamination Kit Personal (DKP) the procedure is best done in pairs. This method is more efficient, and results are obtained sooner, than if each man works alone. If, however, individuals are protected at the time of a liquid attack, the drills in sub-paragraphs 4b, g and i only should be carried out as soon as possible. This will prevent casualties occurring later due to penetration of protective equipment by liquid agents.

Action	Explanation
(a) Take cover from further liquid contamination	If no suitable cover is available continue to expose fresh pieces of detector paper at frequent intervals until one remains unspotted, showing that liquid has stopped falling
(b) Take out a DKP pad and decontaminate the NBC gloves	
(c) Remove the helmet and push the NBC hood on to the back of the neck	The helmet should not be placed on a contaminated object or the ground. It can be hung from the arm by the chin-strap
(d) Take a few deep breaths to steady the breathing; hold the last one and remove the respirator	Do not breathe unless the respirator is on. It may be necessary to replace the respirator on the face in order to take further breaths while performing the drills in sub-paragraph 4e. This may be done as often as is necessary, but it is essential to blow out hard each time the respirator is replaced on the face

(e) Rapidly but thoroughly decontaminate the following in the order stated	This order is specified to maintain the highest possible level of protection during the drill. Close eyes as protection against vapour when the respirator is off the face
(1) If liquid agent has entered the eye — pour water from the water-bottle into the eyes	Liquid chemical agents will penetrate the eyes very rapidly. If it is suspected that liquid droplets may have fallen into the eyes, wash out each eye separately taking care that water from one eye does not flow into the other. Use plenty of water
(2) The face	Pay particular attention to the area immediately surrounding the eyes, nostrils and mouth
(3) Ears, neck and hair	Take special care of the back of the neck, behind the ears and the throat
(4) Inside of respirator face-piece	Pay particular attention to the eyepieces, Mk. 5 spectacles if used, and the outlet valve. If the eyepieces become covered with fuller's earth during the process do not clean them at this point (see Notes 1, 2 and 3)
(f) Replace the respirator, blow out hard, apply fuller's earth to the head harness and replace the hood	Decontamination of the head harness is more effectively done by men working in pairs
(g) Decontaminate the outside of the respirator	Pay particular attention to the outlet valve and the bottom and side of the canister nearest the face-piece. This is more effectively done by men working in pairs
(h) Decontaminate the inside of the helmet and the chin-strap and replace	The puffer bottle is to be used for the inside of the helmet. If insufficient fuller's earth remains in the used DKP pad to complete proper decontamination of the boots, an additional supply may be obtained from the puffer bottle
(i) Decontaminate the boots by opening a used DKP pad so that the powder pours over the boots. Rub and dab the powder well into the welts and any other difficult places	

Note 1: To clean the respirator eyepieces use the disinfecting cloth and the drill in sub-paragraph 4d.

Note 2: Ten to twelve bangs with the DKP pad are sufficient to decontaminate the interior of the face-piece. This amount of fuller's earth powder, even when mixed with perspiration and condensation on the inside of the face-piece, will not affect the working of the respirator. If too much powder is used it may cause coughing and irritation of the eyes and may clog the outlet valve.

Note 3: After completing immediate decontamination, if a wet respirator is put away and allowed to dry out, fuller's earth in the outlet valve may clog, and cause leakage the next time the respirator is worn. To obviate this the outlet valve should be flushed with at least 150 ml of water (approximately 1/6 of water-bottle contents).

Section 8

Other Service toxic hazards

13 Carbon monoxide and carbon dioxide

Introduction

1301 Carbon monoxide and carbon dioxide are well known as products of combustion. Carbon monoxide is produced when combustion of substances containing carbon is incomplete, such as where stoves or cookers are used in confined spaces. It occurs in coal gas and natural gas, in exhausts from internal combustion and jet engines and in fumes from coke or charcoal fires; it may also be encountered in bomb or shell craters and camouflets.

1302 Carbon dioxide is produced by the combustion of any substance containing carbon and is excreted in the breath; it may occur in explosion craters or in badly ventilated tunnels, and may also build up to dangerous levels where men are crowded into inadequately ventilated spaces.

Carbon monoxide

Physical and chemical properties

1303 Carbon monoxide is a colourless, odourless gas that is slightly lighter than air, and consequently will not persist in any dangerous concentration in the open.

Detection

1304 Carbon monoxide may be detected and its concentration measured by the use of the appropriate Draeger tube.

Protection

1305 The Service respirator does not give protection against carbon monoxide. The only satisfactory protection is a respirator with an integral or external air supply.

Mechanism of action

1306 The affinity of haemoglobin for carbon monoxide is many times greater than that for oxygen, and the carboxyhaemoglobin so formed is more stable that the oxyhaemoglobin formed normally. Oxygen transport is therefore blocked if an effective dose of carbon monoxide is absorbed, and death results from anoxia. Although 100 ppm of carbon monoxide may be breathed for several hours by a healthy man, 600 ppm for 1 hour is sufficient to cause symptoms and 2000 ppm will cause death in 1 hour.

Signs and symptoms

1307 The onset of signs and symptoms of carbon monoxide poisoning is insidious. Mild exposure results in severe headache, but severe exposure causes unconciousness within minutes and death shortly afterwards. A bright pink coloration due to the presence of carboxyhaemoglobin rather than that of oxyhaemoglobin is characteristic. The flesh-pink colour of cyanide poisoning is less bright.

Treatment

1308 The casualty should be removed immediately from the carbon monoxide-laden atmosphere. If breathing is absent or weak, positive pressure resuscitation should be commenced without delay. Oxygen, especially under pressure, as in a hyperbaric chamber, increases the speed of carbon monoxide displacement. Absolute rest is essential until all acute symptoms have disappeared.

Carbon dioxide

Physical properties

1309 Carbon dioxide is a colourless and odourless gas that is considerably heavier than air and may therefore collect and persist in craters and other depressions in the ground under conditions of low air turbulence.

Detection

1310 Carbon dioxide may be detected and its concentration measured by the use of the appropriate Draeger tube.

Protection

1311 The Service respirator does not give protection against carbon dioxide. The only satisfactory protection is a respirator with an integral or external air supply.

Mechanism of action

1312 Carbon dioxide is dangerous when present in sufficiently high concentrations (of the order of 10 %) to cause respiratory acidosis. In high concentrations it also has a depressant action on the central nervous system, and by displacing oxygen from the air may produce hypoxia.

Signs and symptoms

1313 Exposure to concentrations of about 5 % carbon dioxide produces a sensation of stuffiness accompanied by headache. After some time the casualty may develop air hunger. High concentrations of carbon dioxide produce rapid unconsciousness and death within minutes.

Treatment

1314 Removal to the open air is all the treatment necessary for mildly poisoned casualties. If breathing is absent or weak, positive pressure resuscitation should be commenced without delay. Oxygen should be used if available.

14 Operational chemical hazards

Introduction

1401 Chemical hazards associated with operational activities arise from smokes and incendiary substances; from fumes of such things as explosives, fuels, lubricants, solvents and missile propellants; and from the fumes encountered in fires and fire-fighting chemicals.

Nitro-explosive fumes

Physical and chemical properties

1402 Nitro-explosive fumes contain oxides of nitrogen of which nitrogen dioxide is the most abundant. It must be remembered that carbon monoxide and carbon dioxide will also be present (see Chapter 13). These fumes may be encountered in gun-pits, armoured vehicles, ships' magazines and turrets and in mining or tunnelling operations. Unexploded cordite may give fumes of nitroglycerine.

1403 Nitrogen dioxide is a pungent brown gas; it is heavier than air, and at temperatures below 20 °C gradually polymerizes into nitrogen tetroxide which is a yellow solid. It is soluble in water with the formation of nitrous and nitric acids.

Detection

1404 There is no device available for detecting this substance.

Protection

1405 Only limited protection is given by the Service respirator.

Mechanism of action

1406 Nitrous fumes act in the same way as lung-damaging agents (see Chapter 5) by causing irritation of the lower respiratory passages and subsequently pulmonary oedema. Concentrations over 100 ppm are dangerous.

Signs and symptoms

1407 These are very similar to those of phosgene (see Chapter 6). The onset is insidious, and although lachrymation, choking and nausea may occur, a latent period of between 1 and 24 hours is common. This is followed by shallow breathing, cyanosis and a painful productive cough, dyspnoea and apprehension and signs of pulmonary oedema. Fumes of nitroglycerine, although not dangerous to life, cause headache and general malaise; hypotension may occur if nitroglycerine fumes are breathed in high concentration.

Treatment

1408 Early treatment with steroids both by inhalation and systemically is life-saving. Treatment with a steroid inhaler should be commenced as soon as possible after exposure; ideally within 15 minutes and this should be supplemented by early systemic use of steroids. Details of this treatment and of supportive measures are given in Chapter 5.

Fumes of fuels and lubricants

Introduction

1409 The unburnt fuels themselves consist largely of hydrocarbons which usually have narcotic effects. Because of their lower volatility, diesel and kerosene are less dangerous than petrol. Fumes from the combustion of these fuels in internal combustion or jet engines contain a certain proportion of carbon monoxide which varies with the characteristics of the engine and its running speed (see Chapter 13). It should be noted that compressed air not specifically intended for breathing may contain dangerous quantities of lubricants. The purity of air for breathing must conform to standards laid down in the British Standard BS 4275/68, *The Selection, Use and Maintenance of Respiratory Protective Equipment.* The overheating of lubricant oils may result in the production of acroleins which are aldehydes with intensely irritant properties. A concentration of 1 ppm is immediately detectable, but

a concentration of 10 ppm causes death in a short time from pulmonary oedema.

Physical and chemical properties

1410 Petrol, diesel and paraffin vapours are heavier than air and may be encountered in fuel tanks, in vehicles or in spaces where fuels have been stored.

Detection

1411 No device for detecting these vapours is available.

Protection

1412 Although the Service respirator gives full protection against hydrocarbon fumes, a respirator with an integral or external air supply is needed in most practical situations where this hazard exists, e.g., in confined spaces.

Mechanism of action

1413 These hydrocarbon fumes act by preferential absorption into the central nervous system. Their action is narcotic and in the case of petrol fumes, they produce unconsciousness and death in concentrations over 1 % (10 000 ppm) in the case of petrol fumes. The exact danger levels for concentrations depend on the volatility of the hydrocarbons in question. Swallowed fuels have a narcotic effect and permanent brain damage has been reported. Aspiration pneumonia may follow as a complication.

Signs and symptoms

1414 Drowsiness and narcosis proceeding to death are encountered in severe poisoning. Less severe exposures may cause dizziness, headache, nausea, vomiting and muscular incoordination. Acute emotional disturbances following hydrocarbon poisoning have been reported. Poisoning from tetratethyl-lead additives is very rare but has been seen in people cleaning storage tanks and not wearing full protective clothing and air-line respirators.

Treatment

1415 Removal to fresh air is the only treatment necessary in cases of mild exposure. When severe poisoning has occurred, oxygen should be administered and positive pressure resuscitation may be called for. Swallowed fuel should be removed by gastric lavage.

Smokes and incendiary substances

Smokes

1416 Smokes produced for operational purposes are not toxic in the concentrations encountered in the field, but exposure to high concentration (e.g., if a smoke weapon is let off in an enclosed space) may result in serious broncho-pulmonary irritation.

1417 Personnel working in closed compartments in ships, tanks and aircraft are at special risk in the event of fire or explosion. The initial threat is from intoxication with carbon monoxide, hydrogen cyanide or phosgene released by burning synthetics and from hypoxia or burns; such casualties require the application of standard resuscitation procedures which include the establishment of a clear airway, ventilation and external cardiac massage as necessary. In carbon monoxide intoxication, which is the most common threat to life, the administration of 100 % oxygen will achieve half-time clearance of carbon monoxide four times quicker than breathing air.

1418 When victims are rescued alive from fire, fluid replacement is the primary requirement for patients with extensive burns. Those who have inhaled smoke may have suffered damage to the large and small airways and alveoli owing to the presence of irritant vapours in some smokes rather than to their carbon content; important constituents of smoke in this respect being chlorine, oxides of nitrogen, phosgene and aldehydes that are generated when plastics and wood are heated to high temperatures.

1419 Zinc chloride is a highly damaging pulmonary agent found in certain smokes, including those used for field training, especially if inhaled in an enclosed space or when the ambient humidity is low.

1420 Damage to the larynx is most likely when there are burns of the head and face; the presence of stridor and cyanosis is then an indication for the insertion of one or two large-gauge plastic cannulae through the cricothyroid membrane and for oxygen administration until a formal tracheostomy can be performed.

1421 When significant smoke inhalation has occurred, there is a risk not only of bronchospasm but also of pulmonary oedema which may occur up to 48 hours after exposure. Reports suggest that repeated inhalations of steroid aerosols at half-hour intervals may be protective. Animal experiments have shown that massive doses of certain glucocorticoids, e.g., methyl prednisolone 2 g repeated at

6 or 12 hour intervals, improve survival rates. The experience gained in 1982 during the Falklands campaign supports the value of such treatment. There is also experimental evidence that humidification of the oxygen administered improves the outcome. Broad-spectrum antibiotics should be prescribed to prevent secondary infections and broncho-dilator drugs should be given when bronchospasm is present. When such victims follow an unfavourable course, positive and expiratory pressure ventilation is indicated.

1422 Although the Service respirator gives no protection against carbon monoxide it does help to preserve vision in a smoke-filled environment and gives short-term protection against hydrogen cyanide and the irritant constituents of smoke. Emergency Life Support Apparatus (ELSA) provides a few minutes' supply of air to facilitate escape from smoke.

1423 A late complication of both burns and inhalation injury is the development of pulmonary fibrosis. There are theoretical grounds for the administration of colchicine or penicillamine as these drugs have been shown to suppress the exudation of collagen from fibroblasts is tissue cultures.

Incendiary substances

1424 Phosphorus, which is present in smoke weapons, may cause severe skin burns if it is scattered while still burning. As a first aid measure, phosphorus-contaminated clothing should be cut away and the burns covered with a dressing kept soaked with water. When conditions permit, the burn should be soaked with sodium bicarbonate solution then washed with a 2 % solution of copper sulphate. This solution must not be used as a soak as the copper would then be absorbed. The particles of phosporus, made black by the copper sulphate, should then be removed with forceps and the burn dressed with a pack of sodium bicarbonate.

Fumes from missile propellants

1425 Missile propellants may include fuming nitric acid, hydrogen peroxide, boranes and hydrazines. Hydrogen chloride occurs in the exhaust gases from many types of missile.

Fuming nitric acid

1426 Fuming nitric acid is irritant and gives rise to nitrous fumes (see paragraphs 1402-1408). Apart from the nitrous fumes themselves, the acid fumes are intensely irritating. The Service

respirator gives very limited protection. Nitric acid fumes produce symptoms essentially similar to those of nitrous fumes.

Hydrogen peroxide

1427 Hydrogen peroxide does not produce any vapour hazard.

Boranes

1428 These substances, which are boron analogues of simple hydrocarbons, evolve heavy vapours which have different actions depending upon their chemical nature. Diborane in mild exposure causes tightness of the chest, cough and raised body temperature; higher concentrations cause pulmonary oedema. Pentaborane and decaborane cause nervous tension and dizziness at low concentrations and convulsions at higher exposure levels. The higher boranes are absorbed through the skin. The Service respirator gives limited protection against borane vapour, but the Suit Protective NBC is needed for protection against liquid splashes. The precise levels of tolerance are not known, but 50 ppm of decaborane vapours may be fatal after some 2 hours exposure. As no specific treatment for borane intoxication is known, therapy should be along symptomatic lines.

Hydrazines

1429 Hydrazine, and its methyl and dimethyl derivatives, may be present in missile exhausts. Acute toxic levels of these fumes are not known, but from the results of animal experiments it seems that the absorption of 1—2 g of hydrazine may be expected to be fatal to man. Chronic exposure to fumes causes weakness, muscular incoordination and later, fatty degeneration of the liver. The Service respirator gives adequate protection.

Hydrogen chloride

1430 Hydrogen chloride is a colourless gas somewhat heavier than air; it is readily soluble in moisture droplets so that it is usually seen as a steamy cloud. The action of the gas is irritant and it is all the more irritant on the rare occasions when the anhydrous gas is inhaled. Mild exposure produces irritation of the throat and lower respiratory passages and concentrations of over 1000 ppm are dangerous owing to the production of pulmonary oedema: such concentrations are unlikely to be encountered on operations. The Service respirator gives adequate protection.

Propylene glycol dinitrate (Otto fuel)

1431 Propylene glycol dinitrate is a potent vasodilator which may produce harmful effects from inhalation of the vapour, ingestion or absorption through the skin. The earliest symptoms are nasal congestion and headache which may persist for several hours. Nausea may develop after prolonged exposure. Death in experimental animals usually results from methaemoglobinaemia but in man the predominant effects are cardiovascular. Organic solvents must not be used for skin decontamination since these markedly facilitate the penetration of propylene glycol dinitrate. A ceiling threshold limit value of 0.2 ppm is recommended. Tolerance to the effects of the vapour generally develops with repeated exposure. Long-term chronic effects are unknown. The Service respirator should give adequate protection in unconfined areas but in confined spaces self-contained breathing apparatus is required. Fresh air and black coffee usually alleviate the headache from vapour inhalation.

Fumes encountered in fire fighting

Smoke

1432 Smoke from fire is irritating to the bronchial mucosa and may, in heavy concentrations, cause pulmonary oedema. The treatment of this condition is discussed in Chapter 5.

Carbon monoxide and carbon dioxide

1433 Carbon monoxide and carbon dioxide may be encountered in fire fighting. These substances are fully discussed in Chapter 13.

Carbon tetrachloride

1434 Carbon tetrachloride (tetrachloromethane) is contained in certain obsolescent fire extinguishers, and this substance is toxic. Acute exposure to carbon tetrachloride at concentrations of 20 000 ppm (2 %) will produce deep narcosis within 30 minutes. The treatment of this condition will be along the lines of that for the recovery from deep anaesthesia. Liver and kidney damage will occur after any heavy exposure and may also be seen after prolonged or repeated exposure to lower concentrations of carbon tetrachloride. These effects may be potentiated by alcohol. The Service respirator gives only limited protection and, furthermore, carbon tetrachloride may be absorbed in lethal quantities through the unbroken skin.

1435 Phosgene and chlorine are formed if carbon tetrachloride is used to extinguish incendiary materials, such as burning magnesium or thermite, or if it comes into contact with hot metal (see Chapters 5 and 15).

Methyl bromide

1436 Methyl bromide may still be found in obsolescent extinguishers intended for petrol and electrical fires. The liquid has a vesicant action when it comes into contact with the skin and the vapour causes delayed lung damage. Concentrations of the vapour over 100 ppm may cause depression and irritability proceeding to unconsciousness with epileptiform seizures; residual paralysis may follow recovery. No specific therapy is known. Limited protection against the vapour is given by the Service respirator.

Modern extinguishers

1437 Non-aqueous fluids currently used in fire extinguishers contain bromo-chlorodifluoromethane (BCF) and freons, which are halogenated compounds of hydrocarbons. These substances are normally of low toxicity, but phosgene and other lung irritants may be formed if such extinguishers are used on fires containing incendiary materials.

Burning plastics

1438 Certain plastics, especially polystyrene, form phosgene and chlorine when burning. Hydrogen chloride is formed when polyvinyl chloride burns. There is considerable evidence that polyurethane foams, which are widely used in building construction and for thermal insulation, can release hazardous quantities of cyanide-related vapours with high toxicity when they burn.

15 Other toxic hazards

Introduction

1501 Toxic hazards may occasionally arise from industrial chemicals which may be encountered accidentally as the result of blast damage to installations, or as unofficial agents in the hands of the enemy. Medical officers are advised to consult *Poisonous Chemicals on the Farm,* HMSO Health and Safety Series (HS(G)2) 1980, and relevant Service manuals for fuller details of many of these substances. There are numerous possible substances and a few of these have been selected for discussion here because they are widely used.

Herbicides

Phenoxyacetates (e.g, 2,4-D and 2,4,5-T).

1502 These have been widely used in agriculture and also as defoliants and anti-crop agents in war. Although these compounds are toxic to plants, they are only slightly toxic to man and serious poisoning, which is rare, has only been observed following ingestion of these compounds. Such poisoning results in hypersalivation, stomach cramps, diarrhoea and vomiting, and myotonia followed by a flaccid paralysis. Recovery may be complicated by liver and kidney damage. In fatal cases, death is preceded by coma. No specific antidote is known, and treatment must be along general lines; electrocardiogram (ECG) monitoring is advised in severe poisoning, since ventricular fibrillation has been reported.

Bipyridilium compounds (diquat and paraquat).

1503 These are non-selective weed-killers. They present little hazard in use but are irritant to mucous membrances and systemic poisoning may result if accidentally swallowed. In the case of paraquat, fatal quantities may be absorbed from the mucosa of the mouth. Systemic poisoning by these compounds results in diarrhoea

and vomiting followed by liver and renal failure, and in the case of paraquat, proliferative pulmonary lesions that commence 5 - 10 days after absorption and become fatal in 14 - 21 days; these effects have not been reported in diquat poisoning. No specific antidote is known, and treatment should be along general lines. Dialysis should be considered as a means of treatment in paraquat poisoning, but should be instituted without delay.

Insecticides

1504 Organophosphorus and carbamate insecticides have actions similar to those of the nerve agents, although some (those of the parathion type) require metabolic conversion before becoming toxic to mammals. Chapter 4 should be consulted if such poisoning is encountered.

Chloropicrin

1505 Chloropicrin is an oily liquid that evolves a pungent irritating vapour heavier than air. It is used as a soil fumigant and disinfectant in agriculture but was used in World War I as a chemical agent because of its irritant action on skin and mucous membranes. Exposure to 1 ppm of the vapour causes irritation of the eyes, and at higher concentrations lachrymation, blepharospasm and skin irritation also occur. When the vapour is inhaled bronchospasm and vomiting may occur. Severe exposures may cause delayed pulmonary oedema which may prove fatal in a similar manner to the oedema produced by lung-damaging agents. Treatment for severe exposure includes the early use of steroids as recommended in sub-paragraph 0510b.

Chlorine

1506 Chlorine is used in water purification and many other industrial processes. It was the first lethal chemical agent to be used in World War I, but was later superseded by phosgene (see Chapter 5). Chlorine is a greenish gas at normal temperatures, with a distinctive smell. Although it is less toxic than phosgene its action and the treatment of its effects are similar. The Service respirator gives full protection.

Arsine

1507 Arsine is a colourless gas with a sweetish smell, and occurs in industrial processes when nascent hydrogen is produced in the presence of soluble compounds of arsenic. Arsine was at one time

considered for use as a chemical agent. The principal action of arsine is to produce haemolysis. Symptoms may be delayed from 1 to 24 hours, depending upon the dose absorbed. Malaise and weakness occur, followed by nausea and vomiting. Blood pigments appear in the urine and haemolytic jaundice occurs in severe cases. It is estimated that a 30 minute exposure to 250 ppm of arsine would eventually prove fatal, death resulting from renal failure. The maximum allowable concentration (MAC), or threshold limit value (TLV), for prolonged exposure is 0.05 ppm. Treatment should be along general lines. Dimercaprol (BAL), though ineffective in preventing haemolysis, may be used to chelate arsenic. The Service respirator gives full protection.

Ammonia

1508 Ammonia, as a liquefied gas, is used in refrigeration plants, in other industrial processes and in agriculture. The vapour is colourless with a distinctive smell and is lighter than air. Ammonia is intensely irritant to the eyes and respiratory tract, and exposure to high concentrations for a long time may prove fatal by producing pulmonary oedema (see Chapter 5) or by reflex inhibition of the heart. It is estimated that a 30 minute exposure to a concentration of 2500 ppm would be fatal and the MAC (or TLV) for prolonged exposure is 50 ppm. Exposure to very high concentrations or swallowing a strong solution may result in oedema of the larynx. If a strong solution of ammonia is sprayed into the eyes, severe corneal injury with subsequent scarring may result. Such an injury should be treated by immediate irrigation of the eyes with water or saline solution followed by amethocaine drops to control the pain. Steroids may be used locally to prevent scarring, and systemically for laryngeal oedema. Cricothyroid puncture or tracheostomy may be required. The Service respirator does not protect against ammonia.

Sulphur dioxide

1509 Sulphur dioxide is a colourless gas with a distinctive smell. It is heavier than air and is used in a liquefied state in refrigeration plants and in other industrial processes. It is reported to have been used by the Spartans as a chemical agent and may, therefore, be the *fons et origo* of chemical operations. The actions of sulphur dioxide are irritant to the eyes and respiratory tract, and exposure to high concentrations may result in pulmonary oedema (see Chapter 5). It is estimated that a 30 minute exposure to 400 ppm of

sulphur dioxide will prove fatal. The MAC (or TLV) for prolonged exposure is 5 ppm. The Service respirator gives full protection.

Hydrogen sulphide

1510 Hydrogen sulphide occurs in the putrefaction of organic material and in many industrial processes; it is a colourless gas with a smell of rotten eggs. It is irritant to the eyes and respiratory tract and may cause corneal erosions. When absorbed, hydrogen sulphide in high concentrations has a cyanide-like action on the cytochrome oxidase system and its toxicity is approximately equal to that of hydrogen cyanide. When inhaled in lower concentrations, hydrogen sulphide first stimulates and then depresses the higher nervous centres, causing death from respiratory paralysis. It is estimated that exposure for 30 minutes to a concentration of 600 ppm would be fatal. The MAC (or TLV) for prolonged exposure is 10 ppm. Conjunctival irritation is noticeable at 50 ppm, but the distinctive smell, although detectable at first at about 0.03 ppm is not noticed after a short time owing to the onset of olfactory fatigue. The Service respirator gives only short-lived protection.

Section 9

Hypoxia and oxygen therapy

16 Hypoxia and cyanosis

Hypoxia

1601 Hypoxia exists when the supply of oxygen reaching the cells is inadequate for aerobic metabolism and is assessed under hospital conditions by measurement of the partial pressure of oxygen in arterial blood. Different tissues vary in their vulnerability to temporary oxygen deprivation. Oxygen is transmitted to the cell from the atmospheric air by the proper function of the respiratory and cardiovascular systems. Oxygen supply to tissues may generally be improved by:

(a) Increasing the partial pressure of oxygen in the inspired air (including hyperbaric methods);

(b) Ensuring adequate ventilation by relieving obstruction, and if necessary by intermittent positive pressure ventilation;

(c) Ensuring adequate blood flow, if necessary by intravenous infusion;

(d) Any appropriate combination of the above measures.

The existence of hypoxia may be indicated by cyanosis, although a cyanotic appearance may be due to other causes, and its absence does not exclude hypoxaemia (see paragraph 1603). The first effect of severe hypoxia is loss of consciousness; the effects of moderate hypoxia may include restlessness, anxiety, air-hunger and confusion. Some special causes of hypoxia include carbon monoxide poisoning and poisoning with cyanides. In both conditions, the skin has a pink colour. The treatment of carbon monoxide poisoning is described in paragraph 1308 and that of cyanide poisoning in paragraph 0810.

Cyanosis

1602 Cyanosis is due to the pressure in the capillary blood of reduced haemoglobin (or sometimes other blood pigments) in

replacement of oxyhaemoglobin. Cyanosis will occur when for any reason there is more than 5 g of reduced haemoglobin per 100 ml of blood in the capillaries. Blood normally contains about 15 g of haemoglobin per 100 ml so that if the blood is of normal corpuscular content and composition, cyanosis would become apparent at an oxygen saturation of about 65 %, i.e., with 35 % or 5 g per 100 ml of haemoglobin in the reduced form. Cyanosis appears, for instance, when the capillary blood is reduced to the requisite level of desaturation.

1603 The determining condition for cyanosis is thus not the degree of desaturation so much as the absolute amount of reduced haemoglobin in the blood. Cyanosis will therefore be apparent at a small percentage desaturation of the blood if for any reason the total haemoglobin content is raised. This circumstance is encountered when there is haemoconcentration, as, e.g., in phosgene poisoning. At the same time, owing to the increased viscosity of the blood, the rate of flow through the capillaries is reduced and an increased percentage of desaturation of the blood results. For these two reasons, namely the increased haemoglobin content of the blood and the increased percentage desaturation of the blood, there is a considerable increase in the absolute amount of reduced haemoglobin per unit volume of blood, so that the cyanosis occurs even when the oxygen supply to the tissues is adequate. Polycythaemias in general show cyanosis for a similar reason, since, as in phosgene poisoning, the circulation rate may be so slowed by reason of the increased viscosity of the blood that even if oxygen is administered and the oxygen supply to the tissues is adequate, the cyanosis may not completely disappear.

1604 In anaemic conditions the reverse state of affairs obtains, so that cyanosis may not show itself until the degree of oxygen desaturation of the blood has reached such a level as to entail severe oxygen lack.

1605 Methaemoglobinaemia and sulphaemoglobinaemia constitute other causes of cyanosis, but the colour of the skin is less blue and more leaden. Although the number of drugs and chemicals reported to cause these conditions are legion, four groups merit mention. These are: nitrites (and nitrates), aniline and its derivatives, certain sulphonamides, and the local anaesthetic prilocaine. Mild cases manifest only cyanosis, but in severe instances symptoms of hypoxia occur.

17 Artifical ventilation and administration of oxygen

Administration of oxygen

1701 When hypoxia is suspected or is evident as shown by cyanosis, oxygen should be given if it is available even when there is adequate lung ventilation. Causes of hypoxia associated with toxic agents include:

(a) Respiratory paralysis;

(b) Transient or persistent lung dysfunction from oedema, bronchial secretions and constriction; pulmonary collapse and consolidation;

(c) Carbon monoxide poisoning;

(d) Hypovolaemic shock in injured casualties;

(e) Cardiac arrest when the circulation is being supported or has been restarted.

Equipment

1702 Common systems of giving oxygen include masks and nasal catheters. No rebreathing occurs with the latter and they are relatively comfortable; if there is consistent mouth breathing they are ineffective unless the tip lies in the nasopharynx. There are two main groups of mask; the controlled concentration types such as the Ventimask and the simple face-piece variety such as the Inspiron. The former are designed for patients with carbon dioxide retention, although they are useful in any patient where the desired oxygen concentration is critical; the latter produce extremely variable inspired concentrations which depend on respiratory frequency, peak inspiratory flows and mask fit. In practical terms any oxygen is better than none for hypoxic casualties; because of the shape of the oxygen dissociation curve, small increases in inspired tension produce considerable improvement in oxygen saturation. Some types of inflating bag and mask (see paragraph 1706b) in conjunction with a reservoir bag or tubing may be used during

spontaneous breathing to give variable concentrations. With this system a 2 litre flow will produce 40% in an average adult male. With flows approaching the minute volume of the casualty, concentrations up to 100% may be obtained.

Oxygen toxicity

1703 It is known that concentrations of 40—60% and higher given over a period of several hours or longer may produce lung changes; these may be worse if there is prior lung damage. Even the inefficient face mask although providing low mean concentration may give high peak inspiratory levels which are harmful. However, under field conditions it is difficult to monitor inspired oxygen levels, and although the dangers of high concentrations for prolonged periods should be borne in mind, the practice of administering 4—6 litres/minute through a standard face mask is unlikely to be harmful during the early resuscitation period, and it is important that casualties with evidence of hypoxia should not be left untreated for fear of oxygen poisoning.

Artificial ventilation

1704

(a) *The airway.* With spontaneous ventilation and apparent upper respiratory obstruction, an airway may be maintained with proper positioning of the head and jaw or the insertion of an oropharyngeal airway; or both. Artificial ventilation may be undertaken with a simple device of this type but there is no protection against inflation of the stomach or regurgitation of stomach contents. Casualties left unattended with these airways in position during automatic ventilation tend to develop respiratory obstruction very quickly. The most effective airway is a cuffed endotracheal tube; when prolonged ventilation is anticipated, insertion of these tubes should be considered a high priority. Where endotracheal intubation is not possible, either because of the situation of the casualty or lack of trained personnel, other devices currently being developed may be used, such as the oesophageal obturator airway and the pharyngeal tracheal lumen airway. None of these devices is fully accepted, and further work is in progress to evaluate them.

(b) *Suction.* With any casualty where the integrity of the airway is at risk efficient suction equipment is essential. This is particularly so with victims of chemical agents; both bronchospasm and profuse bronchial, salivary and nasal secretions occur. Prompt and effective clearance of these is the major factor in resuscitation and urgent attention to this aspect of proper airway management cannot be over emphasized.

Intermittent positive pressure ventilation of the lungs (IPPV)

1705 The purpose of IPPV is to ensure gas movement (air, oxygen or a mixture of these) in the lungs. It cannot always ensure gas exchange which depends on the integrity of alveolar function and normal pulmonary blood flow.

Methods of IPPV

1706

(a) *Expired air.* A first aid technique that in a toxic environment is difficult to supply and maintain, but subject to effective decontamination of the casualty should be attempted if no equipment is available. This method cannot be used in a contaminated environment.

(b) *Manual bag or bellows.* The Resuscitator NBC No 1 Mk II is a bellows inflating device with a face mask attached. At present, this is the only equipment constructed from chemically resistant material and with a suitable chemical filter capable of being decontaminated. If automatic ventilators are not available modern clinical practice favours the use of the self-inflating bag for casualties who need IPPV because of respiratory insufficiency from any cause, as these bags are simple to use and are compatible with airway equipment such as endotracheal tube mounts.

(c) *Automatic ventilators.* Small portable devices have been developed for automatic ventilation using either a mask or endotracheal tube. The Joint Service standard machine is the Pneupac, a fluidic-controlled ventilator using air or oxygen from compressors or cylinders. Multi-outlet versions of this ventilator are in service in field ambulances. Larger machines driven by mains power or medical gases are used only in operating theatres and intensive care units.

Current developments

Positive end expiratory pressure (PEEP)

1707 The principle of maintaining expiratory intrathoracic pressure at a few centimetres of water above atmospheric has been used in hospital practice for some years. It is a valuable procedure where stability of the alveoli is uncertain, such as in loss of surfactant or where an increase in functional residual capacity produces a more favourable compliance. Equipment for use in the field has been developed consisting of a small valve on the expiratory part of a self—inflating bag; it should be used with an endotracheal tube in place.

Intermittent mandatory ventilation (IMV)

1708 This is a technique where the patient or casualty is allowed to take spontaneous breaths as the ventilation frequency is decreased. It is normally employed in weaning patients from ventilators but it can be useful when a casualty attempts to breathe on his own while still needing ventilatory assistance.

High-frequency positive pressure ventilation (HFPPV)

1709 Recent work has shown that low tidal volumes at high frequency (1—10 Hz) can maintain normal gas exchange, the main advantage being that cardiac output is affected less than in conventional IPPV. While the technique has still to be used routinely, it may be of value in field conditions by the method of ventilating a casualty with a needle or cannula passed through the cricothyroid membrane, an effective technique where severe obstruction such as oedema of the glottis precludes endotracheal intubation. This technique is still being evaluated.

Section 10

Toxins

18 Toxins

Introduction

1801 It is not within the scope of this manual to discuss at length the problems connected with the use of biological agents. This is covered in JSP 380 *Medical Manual of Defence Against Biological Agents*. However, in view of the ambiguous status of some toxins it is felt that some explanation of this problem, with reference to biological operations, should be included here.

Toxins

1802 These may be defined as poisonous chemical compounds produced in nature by some species of living organisms, many of which are extremely toxic. Some toxins can be produced cost effectively on a large scale either by synthetic chemical manufacture or by natural means. They can be disseminated in the same way as live biological agents or chemical agents.

1803 Toxins present a problem of classification because, although they are simply non-living chemical compounds, they are found naturally as products of a variety of micro-organisms, in plants, reptiles, fish, corals and so forth. Moreover modern synthetic organic chemistry has allowed the synthesis of these compounds without reference to living biological systems.

1804 Some guide to the place that toxins hold in the spectrum of possible chemical and biological agents may be found by referring to the wording of the 1925 Geneva Protocol and the 1972 Biological and Toxin Weapons Convention:

(a) Article I of this Convention states that:

Each State, party to this Convention, undertakes never in any circumstances to develop, produce, stockpile or otherwise acquire or retain:

(1) Microbial or other biological agents, or toxins whatever their origin or method of production of types and in quantities that have no justification for prophylactic, protective or other peaceful purposes;

(2) Weapons, equipment or means of delivery designed to use such agents or toxins for hostile purposes or in armed conflict.

(b) The Geneva Protocol of 1925 prohibits 'the use in war of asphyxiating, poisonous or other gases and of all analogous liquids, materials or devices . . .' and 'the use of bacteriological methods of warfare . . .'

No single definition of what a toxin is appears in the 1972 Convention but during negotiations of the Convention no participant dissented from the definition: 'Toxins are poisonous substances produced by biological organisms including microbes, animals and plants.'

1805 Thus in common with all other poisons and pathogenic micro-organisms the use of toxins is prohibited by the Geneva Protocol of 1925; but the development, production, stockpiling, acquisition or retention of toxins is specifically proscribed by the 1972 Biological and Toxin Weapons Convention.

1806 Presently then, the toxins, because of their commonly assumed natural biological origins, appear to be placed in the category of biological agents. However, having regard to their purely chemical nature, and to future advances in synthetic organic chemistry, they may come to be regarded more and more as essentially agents of chemical operations in which case they will become a subject for more detailed discussion in this manual.

Glossary

Agent	*See* Chemical agent
Artificial ventilation — resuscitation	The practice of maintaining respiration by artificial means
Anti-plant agent	A chemical agent that acts against plant life (e.g., defoliants)
Automatic injection device	A small automatic hand device for the self-injection of antidotes
Benzilates	*See* Glycollates
Binary agent	An agent formed by the reaction between two relatively harmless chemicals during delivery of the agent
BZ	A typical glycollate (*qv*)
Blister agent	*See* Vesicant agent
Chemical agent	A chemical substance that is intended for use in military operations to kill, seriously injure or otherwise incapacitate man through its pathophysiological effects. Excluded from consideration are riot control agents, herbicides, smoke and flame
Chemical defence	The methods, plans and procedures involved in establishing and executing defensive measures against attack utilising chemical agents (NATO Glossary)
Chemical operations	Employment of chemical agents (*qv*) to kill, injure or incapacitate man or animals for a significant period of time and to deny or hinder the use of areas, facilities or materiel; or defence against such employment (paraphrase of NATO Glossary)
Choking agent	*See* Lung-damaging agent
Concentration-time (Ct)	The dosage of a vapour received, being the product of the concentration and of the duration of exposure in minutes (mg min/m^3)
CS	A riot control agent: orthochlorobenzylidene malanonitrile
Cyanide-type agent	A lethal agent which poisons the mechanism of tissue respiration (e.g., hydrogen cyanide)
Cyanogen chloride	A cyanide-type agent (*qv*) which also has lung-damaging properties
DA, DM and DC	Arsenical vomiting agents

Decontamination	The process of making any person, object or area safe by absorbing, neutralizing, making harmless, or removing chemical (or biological agents, or by removing radioactive) material clinging to or around it (NATO Glossary)
Detection	The process of finding out whether a chemical (or biological or radioactive) agent is present
Dicks	Chemical agents of the vesicant or skin damaging or blister type chemically related to dichlorarsine
G-agent	A nerve agent. Usually non-persistent, but which may be thickened to confer persistency
GA	Tabun
GB	Sarin
GD	Soman
Glycollates	Glycollic acid esters having an anticholinergic action like that of atropine, but with a preponderance of central over peripheral effects, so that a toxic psychosis is produced by a very small dose
Hallucinogenic agent	A mental incapacitant
Herbicides	*See* Anti-plant agent
Immobilizing agent	*See* Physical incapacitants
Incapacitant	A chemical agent employed primarily to render an enemy incapable without permanent after-effects. Larger doses of an incapacitant may, however, be lethal
Incapacitating dose	The dose of an incapacitant which will incapacitate. Usually expressed as the incapacitating dose for 50 % of those exposed (ID.50 or ICt.50)
Lethal agent	A chemical agent employed primarily to kill, although it may have incapacitating effects at lower doses
Lethal dose	The dose of a lethal agent which will kill. Usually expressed as the lethal dose for 50 % of those exposed (LD.50 or LCt.50)
Lewisite	An arsenical vesicant agent
LSD 25	A mental incapacitant of the indole type
Lung-damaging agent	A lethal agent which acts against the respiratory system (e.g., phosgene (*qv*))
Mustard gas	A vesicant agent (*qv*) (e.g., sulphur mustard)

NBC	Nuclear, biological and chemical
Nerve agent	A lethal agent which interferes with the transmission of nerve impulses by blocking the action of cholinesterase: the lethal effect being due to respiratory paralysis
Nitrogen mustards	Nitrogen derivatives of mustard gas (*qv*) having a similar effect
Oximes	Compounds which reactivate inhibited cholinesterase (e.g., pralydoxime mesylate)
Persistence	The ability of a chemical to remain in the vicinity in which it has been dispersed
Phosgene	A lethal lung-damaging agent (*qv*)
Phosgene oxime	A skin-burning or vesicant agent (not to be confused with phosgene (*qv*) which is a lung-damaging agent, nor with oximes (*qv*) which are used in therapy of nerve agent poisoning)
Physical incapacitants	Chemical agents which are used to interfere with a physical function (e.g., mobility or sense of balance)
Pralydoxime mesylate	*See* Oximes
Protection	The process of preventing the access of a chemical agent to the people attacked
Psychotomimetic agent	A chemical employed primarily to derange the mind. *See* also Incapacitant
Riot control agent	*See* Sensory irritant agent
Sensory irritant agent	An agent which is reversible and shortlasting (e.g., CS)
Skin-damaging agent	*See* Vesicant agent
Stability	The ability of an agent to remain active despite weather or other conditions
Sulphur mustard	Mustard gas (*qv*)
Tear agent	Sensory irritant agent
V-agent	One of a group of persistent nerve agents (e.g., VX)
Vesicant agent	An agent employed to produce skin lesions which may vary from erythema to severe burning of the skin: (e.g., sulphur mustard (*qv*) and phosgene oxime (*qv*))
Vomiting agent	An agent which is sensory irritant in action, but in the effect of which lasting pain and vomiting occur (e.g., DA and other organic arsenicals)

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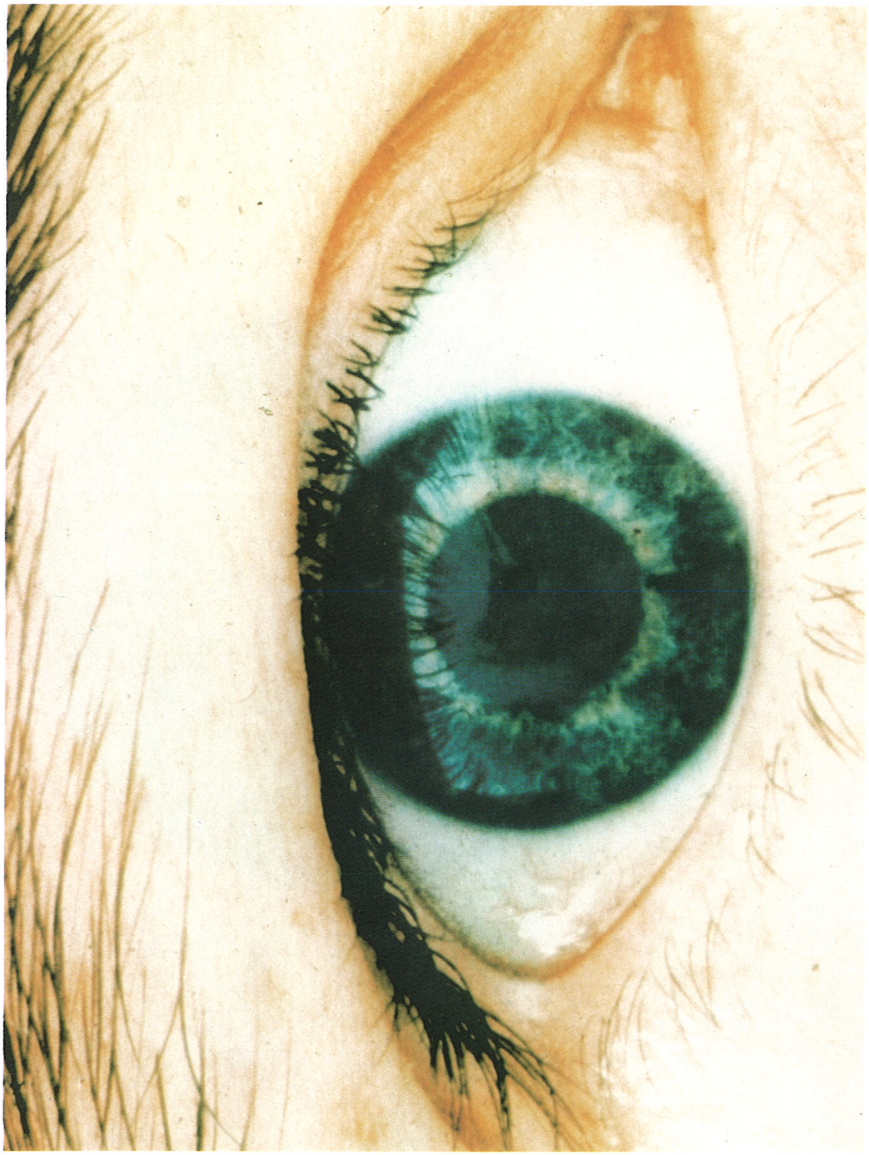

Plate 1a Normal eye
See also page 4-5

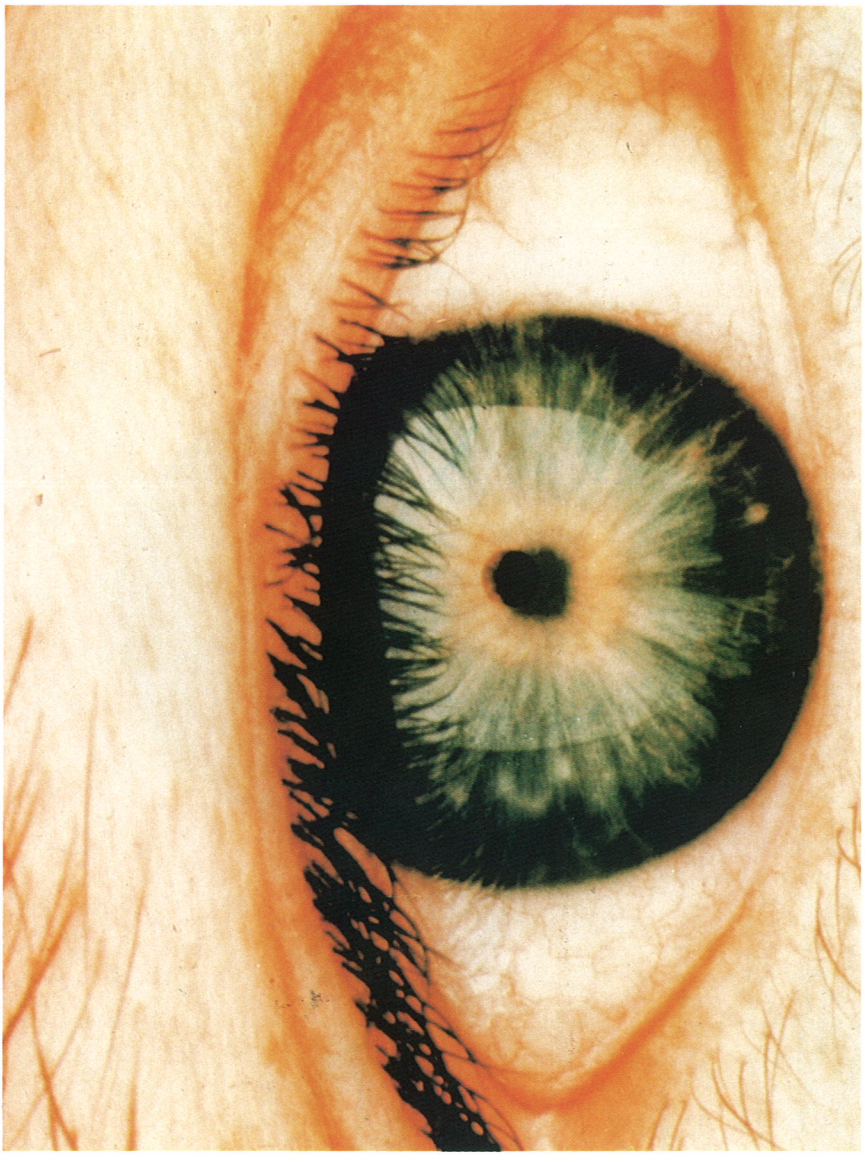

Plate 1b Eye after exposure to nerve agent
See also page 4-5

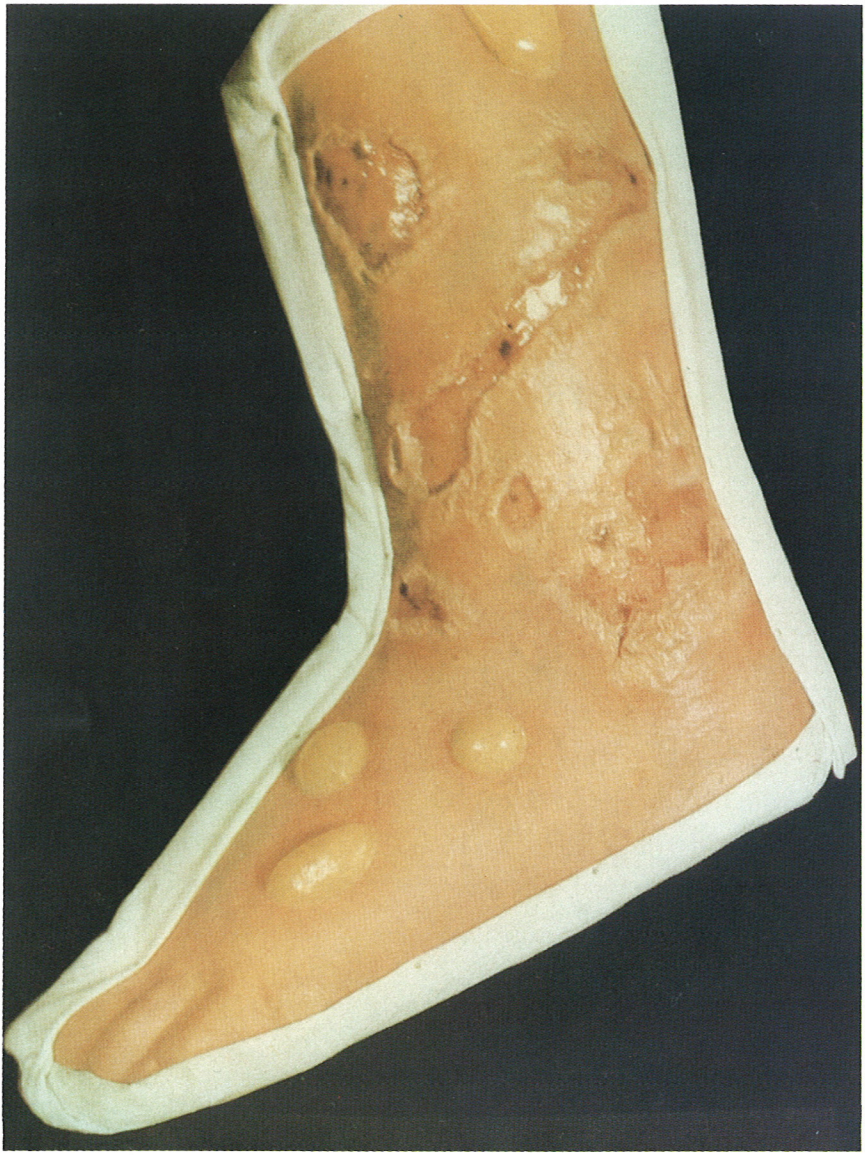

Plate 2 Effect of liquid sulphur mustard on the skin (5 days)
Destruction of outer layers of the skin following blister formation 5 days after exposure to liquid mustard.
See also page 6-6

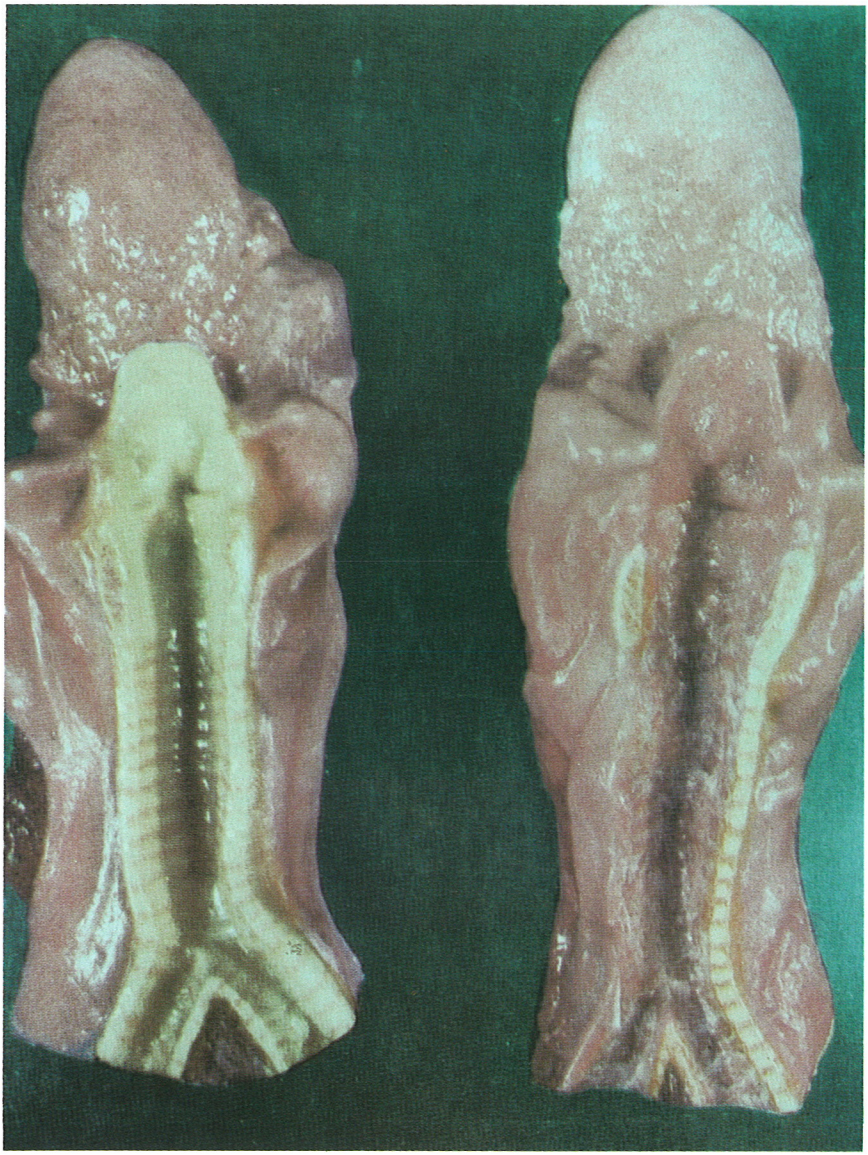

Plate 3 Effect of sulphur mustard on the respiratory tract
Left: Normal human trachea. **Right:** Trachea after exposure to sulphur mustard.

See also pages 6-4 and 6-6

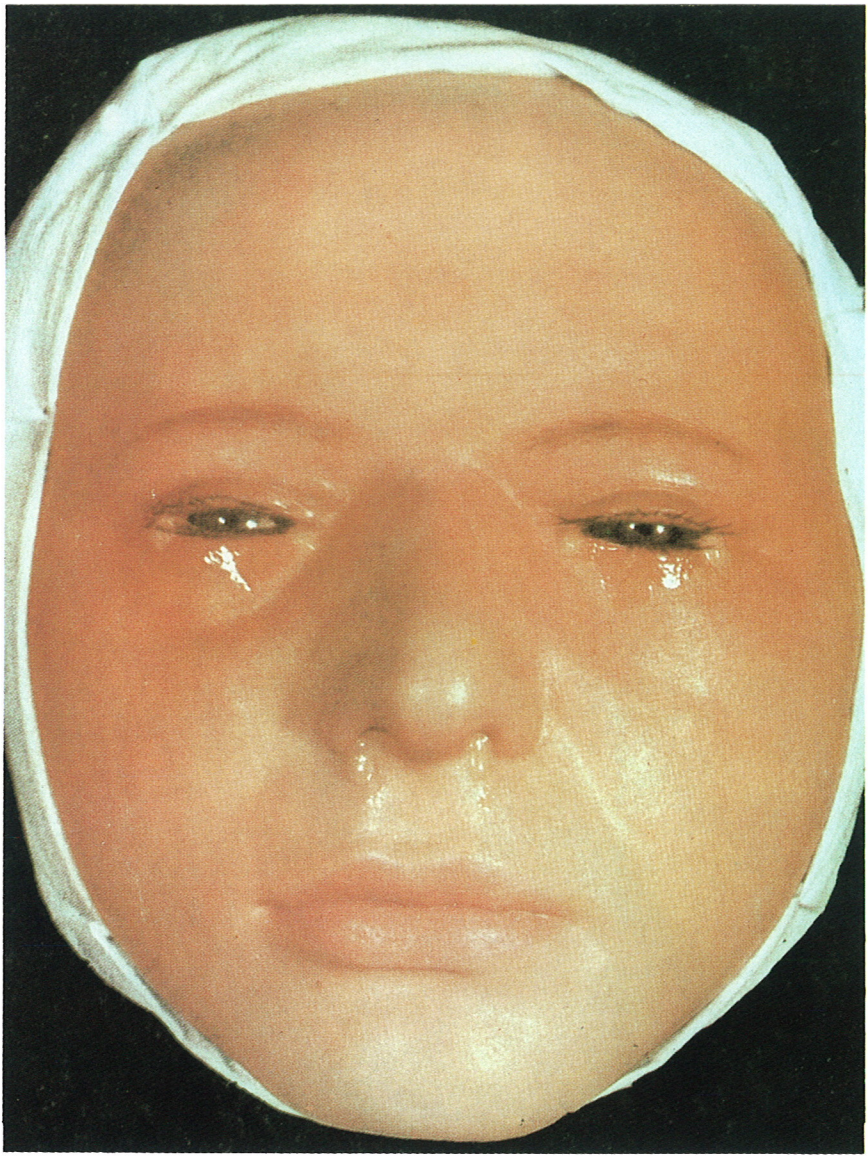

Plate 4 Effect of sulphur mustard on the face
Typical appearance of the face 24 hours after exposure to sulphur mustard vapour.
See also page 6-5

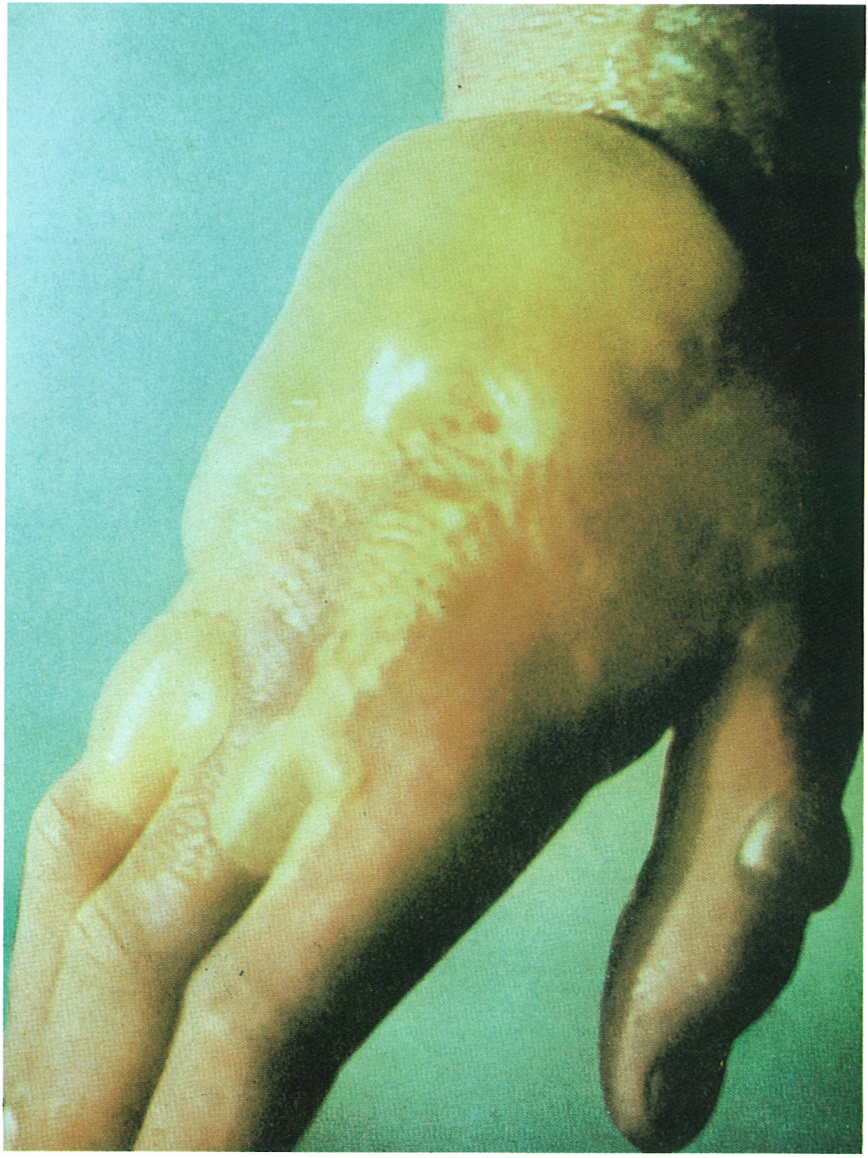

Plate 5 Effect of liquid sulphur mustard on the skin
Blisters appearing on the skin 24 hours after exposure to liquid sulphur mustard
See also page 6-6

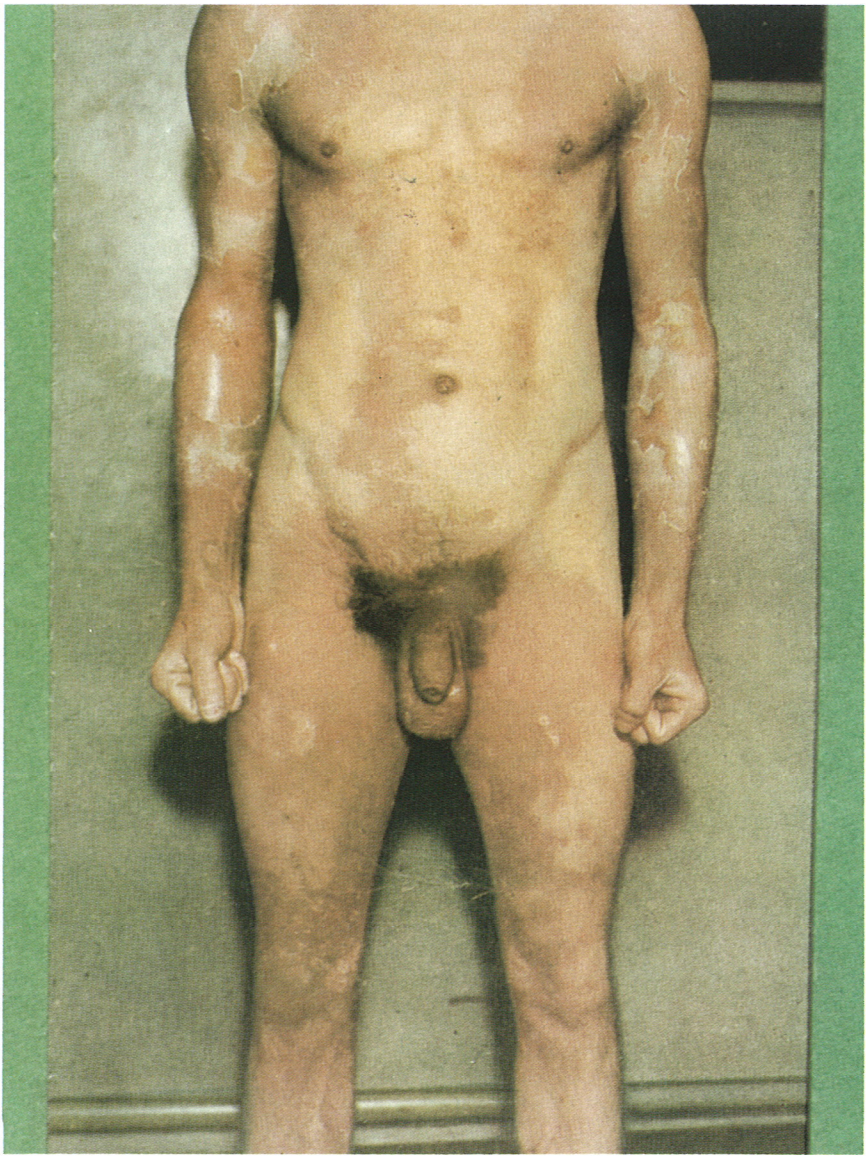

Plate 6 Effect of liquid sulphur mustard on the skin (7 days)
Scarring and coppery pigmentation of the skin 7 days after exposure to liquid sulphur mustard.
See also page 6-3

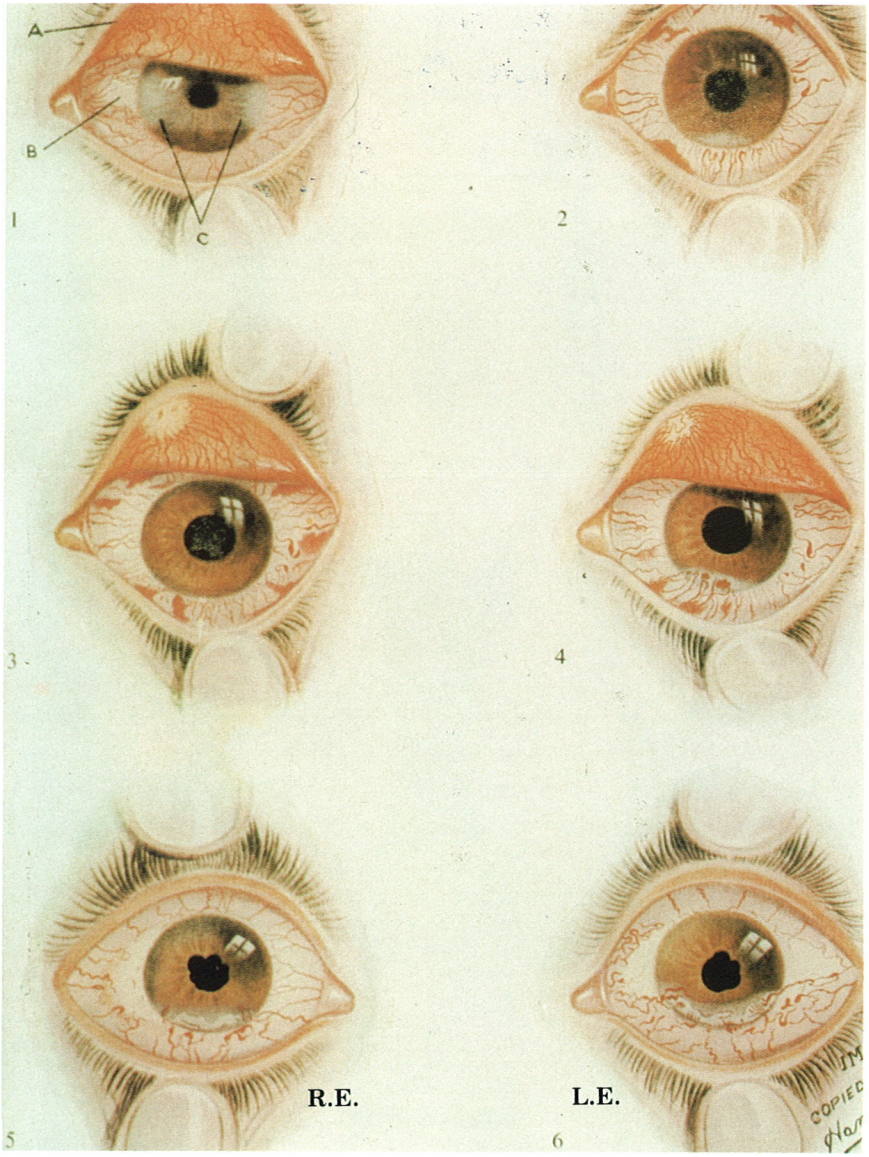

Plate 7
See caption opposite

Plate 7 Effects of sulphur mustard on the eye

(1) Severe lesions 3 weeks after exposure to sulphur mustard vapour showing a small patch of hyperaemia in the palpebral conjunctiva (A), a triangular white necrotic patch at the inner side of the limbus (B), and a slight corneal haze in the palpebral aperture (C).

(2) Severe lesion after exposure to a spray of liquid sulphur mustard, showing subconjunctival haemorrhages, dilated thrombosed vessels and partial destruction of the limbal capillaries in exposed areas which were formerly necrotic and opaque; and in the cornea, epithelial bedewing with oedema of the substantia propria below.

(3) Severe lesions 5½ weeks after exposure to a spray of sulphur mustard (the same eye as Fig 2), showing an area of necrosis on the palpebral conjunctiva, haemorrhages in the ocular conjunctiva with an area of necrosis at the limbus below, some bedewing of the corneal epithelium and clear substantia propria (stage of subsidence of primary oedema).

(4) Severe lesion 8 weeks after exposure to a spray of sulphur mustard (the same eye as Figs 2 and 3), showing partial vascularization of the necrotic patch in the palpebral conjunctiva, hyperaemia of the ocular conjunctiva around and in the former necrotic white area, reappearance of corneal oedema below, and vascular invasion of the cornea with thrombosis and heamorrhage in this region.

(5) and (6) Very severe lesions (so-called delayed keratitis) in both eyes 23 years after exposure to sulphur mustard, and 13 years after the first attack of delayed ulceration.

R.E. Fibrous plaque of ocular conjunctiva at outer side; abnormal varicose conjunctival vessels in outer and lower part; gross scarring of lower part of cornea and limbus. Posterior synechiae.

L.E. Triangular fibrous plaques at inner and outer side of limbus, tortuous and varicose conjunctivae and posterior synechiae.

See also page 6-5

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