NERVE AGENTS

GUIDELINES FOR ACTION IN THE EVENT OF A DELIBERATE RELEASE

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1 BACKGROUND

1.1 Introduction

The nerve agents are a group of particularly toxic chemical warfare agents. They are chemically related to organophosphorus insecticides. The principle agents in this group are tabun (GA), soman (GD), sarin (GB), GF and VX.

A small drop of liquid nerve agent on the skin may be sufficient to cause death.[1]

Nerve agents may be absorbed by inhalation, ingestion or through the skin.

Protective clothing and full respiratory protection must be worn in contaminated areas or when handling casualties contaminated with liquid agent. Off-gassing of volatile nerve agents from casualties may be sufficient to cause symptoms. Casualties should be transported in such a way that emergency personnel do not become contaminated, or exposed to fumes.

Nerve agents inhibit the enzyme acetylcholinesterase, thus resulting in an accumulation of acetylcholine throughout the body which has effects on the central nervous system and nicotinic and muscarinic receptors peripherally. Accumulation at these sites may produce the following:

Muscarinic (parasympathetic effects)

- Copious secretions (salivation, bronchorrhoea, rhinorrhoea, lachrimation, sweating), bronchospasm, bradycardia, abdominal cramps, diarrhoea, constricted pupils (miosis).

Nicotinic (motor and post-ganglionic sympathetic)

- muscle fasciculation, weakness, respiratory paralysis, tachycardia, hypertension.

Central Nervous System:

- Emotional lability, confusion, ataxia, convulsions, coma and central respiratory depression.

Death is usually due to respiratory arrest.

1.2 Physical and chemical properties

Nerve agents are organophosphorus esters. They are liquids at room temperature, but may also be absorbed as an aerosol or vapour. In their pure state most are colourless, but impure agents may be yellow or brown liquids. Some agents have a fruity odour. Agents may be thickened with various substances to increase persistence and thus the total amount penetrating intact skin.

Sarin (GB) is a volatile liquid (high vapour pressure) and tends to be non-persistent as it evaporates readily. Soman (GD) and Tabun (GA) are also relatively volatile. VX is a relatively non-volatile liquid (low vapour pressure) and is therefore particularly persistent.

Vapour from volatile agents is readily absorbed by inhalation and they present both a respiratory and a percutaneous hazard. VX has a low vapour pressure and absorption occurs mainly through the skin, rather than by inhalation of vapour.

Nerve agent vapour is denser than air and therefore accumulates in low-lying areas.

Nerve agents are moderately soluble in water, but highly soluble in lipids. They hydrolyse slowly in water but are rapidly inactivated by strong alkalis and chlorine-containing bleaches[1].

Table 1

	TABUN (GA)	SARIN (GB)	SOMAN (GD)	V X
	Ethyl N-dimethyl phosphor amido cyanidate	Isopropyl methyl phosphono luoridate	Pinacolyl methyl phosphono fluoridate	O-ethyl S-[2-(diisopropylamino) ethyl] methylphosphono-thiolate
APPEARANCE	Colourless to brown liquid giving of a colourless vapour	Colourless to brown liquid giving of a colourless vapour	Colourless to brown liquid giving of a colourless vapour	Amber coloured liquid
Molecular weight	162	140	182	267
Vapour density	5.6	4.9	6.3	9.2
Vapour Pressure (mmHg at 25C)	0.07	2.9	0.4	0.007
Volatility (mg/m3 at 25C)	610	22,000	3,900	10.5

1.3 Summary of Human Toxicology

When dispersed as a spray or aerosol, nerve agents may be absorbed by inhalation, ingestion or through the skin. When dispersed as a vapour, the vapour is primarily absorbed through the respiratory tract. For volatile agents (eg GB), inhalation is considered the major hazard. Non-volatile nerve agents (eg VX) present a contact hazard with little respiratory exposure unless aerosolised droplets are inhaled.

Nerve agents exert their major action by causing prolonged inhibition of the enzyme acetylcholinesterase. Inhibited enzyme is unable to break down acetylcholine, which therefore accumulates causing excess stimulation at nicotinic and muscarinic and central nervous system sites.

Some nerve agents undergo a conformational change after binding to the enzyme. This process is known as aging. Once 'aged' the enzyme-inhibitor complex is not able to be reactivated by oximes. Soman (GD) ages very quickly, whereas the other nerve agents age more slowly and are responsive to oxime therapy.

The clinical effects or nerve agent poisoning are dependent upon the concentration, duration and route of exposure. Local effects eg miosis may occur in the absence of systemic poisoning.

The main cause of death is respiratory failure due to a combination of CNS depression and muscle weakness.

Other symptoms and signs include:

Mild:

- headache, miosis, conjunctival injection, lachrimation, rhinorrhoea

Moderate:

- wheezing, drooling, vomiting, diarrhoea, fatigue, fasciculation, muscle weakness

Severe:

- respiratory depression, convulsions, cardiac arrhythmias

Three types of antidote are of use in the treatment of nerve agent poisoning and have a synergistic effect:

ATROPINE

- antagonises the effects of acetylcholine at muscarinic receptors. It is particularly effective in decreasing secretions and treating bradycardia.

OXIMES

- reactivate inhibited enzyme, thereby decreasing the amount of excess acetylcholine

DIAZEPAM

- CNS protection.

The mainstay of treatment is early decontamination, respiratory support and treatment with antidotes, particularly atropine.

In the event of an incident causing release of nerve agent, additional resuscitation equipment and antidotes will be available. These should be requested EARLY in the course of the incident. The 'trigger' for obtaining these supplies will be released by DH separately.

Sarin has been used twice against civilian populations in Japan. In the first incident, 7 people died and there were over 200 casualties[2], in the second at Attack there were a total of 11 deaths and over 5,000 casualties[3] in both cases casualties suffered symptoms of typical organophosphorus poisoning. Hospital staff suffered effects of sarin toxicity, either from residual vapour exposure or secondary contamination.

1.4 Clinical features

1.4.1 Acute

The order in which signs and symptoms appear and their relative severity depend upon the route of exposure and whether the casualty has been exposed to liquid or vapour. Systemic effects may occur after significant absorption via any route.

Inhalation

- Chest tightness, rhinorrhoea and increased salivation may occur within minutes.
- Systemic features may develop.

Dermal exposure

- Contact with liquid nerve agent may produce localised sweating and fasciculation, which may spread to involve whole muscle groups.
- Systemic features may develop (see below), though onset is slower than following vapour inhalation.

Ingestion

- Ingestion of contaminated food or water may cause abdominal pain, nausea, vomiting, diarrhoea and involuntary defecation.
- Systemic features may develop..

Eyes

- The eyes are particularly sensitive to nerve agents. Miosis (small pupils), hyperaemia and ciliary body spasm may occur together with a headache.
- Exposure to low concentrations of vapour may produce miosis in the absence of significant systemic poisoning. It is not necessarily an indication for systemic therapy.
- Miosis may last several days.

Respiratory tract

- Stimulation of muscarinic receptors results in copious secretions and bronchospasm may occur. Dyspnoea, chest tightness and pulmonary oedema may occur.

Respiratory failure (central and muscular) is the major cause of death.

Muscles

- Systemic poisoning results in stimulation of nicotinic receptors and results in fatigue, muscular weakness, twitching, fasciculations, dyspnoea and cyanosis.
- Dermal exposure may cause local fasciculation at the site of exposure.

CNS

- CNS effects include coma, convulsions, respiratory depress and resultant hypoxia and cyanosis.
- Neurological sequelae including poor memory and insomnia have been described following severe exposure.

CVS

- CVS effects include hypotension, bradycardia, tachycardia and cardiac arrhythmias including torsade de pointes

Systemic effects

- Casualties will experience some or all of the following symptoms:

Mild:

- Headache, miosis, dim vision, conjunctival injection, lachrimation, rhinorrhoea, drooling, tight chest, localised sweating or fasciculations, nausea, bradycardia or tachycardia.

Moderate:

- As above with increased wheezing, drooling, vomiting, diarrhoea, fatigue, fasciculation, muscle weakness or vomiting

Progress of symptoms from mild to moderate indicates either inadequate treatment or continued exposure to the agent

Severe:

- Casualties will experience the above symptoms together with strange or confused behaviour, difficulty breathing, respiratory depression, convulsions, involuntary urination or defecation, cardiac arrhythmias, severely pinpoint pupils or coma.

1.4.2 Intermediate syndrome

Following poisoning with some organophosphorus compounds patients may develop an intermediate syndrome consisting of acute respiratory failure, with paralysis of the proximal limb muscles, motor cranial muscles and respiratory muscles 24 to 96 hours after poisoning, despite initial recovery from the cholinergic phase of poisoning.

The Intermediate Syndrome is refractory to atropine and pralidoxime treatment and ventilation is required; it is thought that the Intermediate Syndrome may not occur in patients who have received adequate pralidoxime therapy during the acute cholinergic phase.

1.4.3 Chronic effects

Sarin exposure has resulted in persistent changes in the electroencephalogram (EEG) in humans. Anxiety, irritability, impaired concentration and memory, confusion, slurred speech, drowsiness, depression and nightmares may be prolonged. Some organophosphorus compounds have been reported to cause peripheral neuropathies but this is thought not to occur with nerve agents.

2 CLINICAL PROCEDURES

Personnel must not enter a contaminated area without full personal protective equipment including respiratory protection.

2.1 Decontamination and first aid

2.1.1 Triage

Primary (first look) triage should be carried out using the standard triage sieve. In addition to normal discriminators, secondary triage should include the following:

Immediate

- Respiratory arrest, flaccidity, unconscious, currently fitting

Urgent

- Shortness of breath, bronchorrhoea, history of fitting

Delayed

- Known exposure to organophosphorus compounds, with or without symptoms.

2.1.2 Mass Decontamination

Mass casualties may be decontaminated by the Fire Service, using high volume, low pressure hosing or related methods, prior to handing over to the ambulance service.

Ambulance and medical staff should not enter the hot zone, except under exceptional circumstances. In the warm zone, an appropriate personal protective ensemble, including a respirator, must be worn.

Patients should be removed from the source of exposure. All the patient's clothing and personal effects should be removed. Skin decontamination should be carried out using a rinse-wipe-rinse regime with dilute detergent (10ml washing up liquid to a 10litre bucket of water).

Contaminated clothing should be placed in clear, labelled, sealed bags to prevent further contamination

Casualties should subsequently don clean clothing eg paper suits.

2.2 Sample collection and monitoring

Sample collection is useful in confirming the nature of the incident and in monitoring treatment. Erythrocyte cholinesterase correlates better with the severity of exposure than plasma cholinesterase, but is less widely available. Clinical samples eg electrolytes, glucose and blood gases should be taken according to the patient's condition.

Details of sampling requirements are outlined in section 3 of this document.

2.3 Treatment

2.3.1 Atropine and ventilatory support are the mainstay of treatment. High dose oxygen should be administered. Early ventilation may be required. Secretions may be copious and require regular respiratory toilet. Patients with more severe poisoning require ECG monitoring.

2.3.2 Atropine

Atropine antagonises the effects of acetylcholine at muscarinic receptors and is useful in controlling parasympathetic symptoms eg copious secretions and bradycardia

- If bronchorrhoea develops, administer atropine (0.6-2 mg in an adult; 20 microgram/kg in a child) intravenously every 10-30 minutes until secretions are minimal and the patient is atropinized (eg dry mouth and pulse rate 80-90bpm). In severe cases very large doses of atropine over a prolonged period may be required.

- Hypoxia should be corrected, as administration of atropine to hypoxic patients my precipitate arrhythmias.

2.3.3 Oximes

Oximes act by reactivating inhibited enzyme, thereby decreasing excess acetylcholine.

- Moderately or severely poisoned patients should be given pralidoxime mesylate 30 mg/kg body weight (2 g in an adult) intravenously over four minutes to reactivate phosphorylated enzyme. **Early administration is a priority**. In severe cases, pralidoxime mesylate 30 mg/kg body weight will be required intravenously every four to six hours, depending on the clinical features. Alternatively, an infusion of pralidoxime mesylate 8-10 mg/kg/hr may be administered as maintenance therapy (adult dose).

- Patients may require prolonged therapy.

Nerve agents may become resistant to reactivation by any oxime due to a process known as aging. For Soman (GD) this process occurs quickly and significant reactivation by oximes does not occur. Other agents age more slowly. Oximes should be administered promptly to have the greatest effect.

Some nerve agents (eg Tabun [GA]) are not significantly reactivated by pralidoxime. Patients who are slow to respond despite treatment with adequate doses of atropine may benefit from administration of obidoxime.

Even though the agent may be identified clinically as a nerve agent, the exact identification eg GA, GB, GD, VX may not be known initially. In the absence of exact identification, failure to respond to treatment, or failure to reactivate cholinesterase may be an indication to change to obidoxime. This should be discussed with the National Poisons Information Service (0870 600 6266).

2.3.4 Diazepam

- Intravenous diazepam (adult 5-10 mg; child 1-5 mg) has a central protective effect and is useful in controlling apprehension, agitation and convulsions; the dose should be repeated as required.

Patients who are slow to respond should be discussed with the NPIS (0870 600 6266).

The armed forces may each be issued with three autoinjectors (ComboPens) used for self-treatment or 'buddy aid'. Each injector contains atropine 2mg, Pralidoxime 500mg and a diazepam pro-drug (avizafone). Used autoinjectors may accompany patients to hospital and give an indication of pre-hospital treatment.

2.4 Admission criteria

2.4.1 All casualties must be triaged by a Triage Officer.

2.4.2 Mild symptoms: such as headache, nausea; small pupils, visual difficulties and painful eyes; running nose, eyes and excess salivation; mild muscle weakness and agitation

- Observe for 2 hours.
- Some individuals may suffer painful eyes. Atropine eye drops may be considered for relief of pain from ciliary spasm,. However, vision will remain impaired due to dilated pupils and these patients should not drive. Atropine eye drops are **not** indicated for patients who are suffering constricted pupils alone.
- If symptoms improve or the patient has not deteriorated within 2 hours then casualties should be discharged with information on criteria to seek further medical advice.

2.4.3 Moderate symptoms: such as dizziness, disorientation and confusion; sneezing, coughing and wheezing; marked drooling and excess phlegm production; vomiting and diarrhoea; marked weakness, difficulty in breathing

Should be kept in a 'holding facility' (eg a ward, a Local Authority designated temporary mortuary or other area where they may be observed).

- Medical staff must observe carefully for deterioration in medical condition and be prepared to move patients to the severe symptom group if necessary.
- Administer antidotes as appropriate.
- If symptoms improve or patient has not deteriorated within 24 hours then casualties should be discharged with information on criteria to seek further medical advice.

2.4.4 Severe symptoms: such as respiratory difficulty, convulsions and ventricular arrhythmias

- These casualties will be admitted to ITU or equivalent wards
- Administer antidotes.
- Supplemental oxygen should be administered. Excess secretions may require removal by suction.
- Monitor ECG and adequacy of respiration; ventilate if necessary
- Monitor red blood cell cholinesterase daily until symptoms improve; it is vital to treat the symptoms and not be lead by the cholinesterase concentration

3 LABORATORY PROCEDURES

3.1 Erythrocyte cholinesterase

Measurement of red cell cholinesterase is useful in confirming severe exposures and may also confirm that the patient is responding to oxime therapy. Depression of red cell cholinesterase more than 50% is associated with more severe effects, and more than 90% inhibition is associated with severe poisoning. However, the correlation between inhibition and clinical signs is not exact, particularly for less severe exposures.

An initial sample (4ml EDTA tube) should be taken. A further sample taken four hours after oxime therapy has commenced may show whether reactivation is occurring. Subsequent daily samples will confirm that significant reinhibition of enzyme is not occurring. However, **therapeutic decisions should be made on clinical grounds, not solely on the cholinesterase inhibition**.

Tubes should be clearly labelled, including the patient's name, date and time the sample was taken. They should be kept cool but **not** frozen. They should be sent within 4 – 8 hours to a designated laboratory that has been warned about sample receipt.

3.2 Plasma cholinesterase

Plasma cholinesterase correlates less well with clinical effects than erythrocyte cholinesterase. However, it may confirm exposure to nerve agents and the assay is more widely available. An initial sample (lithium heparin or EDTA) should be taken and sent to a designated laboratory.

A list of laboratories capable of undertaking assays in included in this document and is also available on TOXBASE.

3.3 Other

Other clinical tests will be dictated by the patient's condition.

Electrolytes should be checked as disturbances may contribute to the development of arrhythmias.

Hyperglycaemia may occur.

Blood gases are important to monitor respiratory function and acid – base balance.

4 Public Health Procedures

4.1 Surveillance and Detection of deliberate release

A deliberate release should be considered in the event of any cases where there is no clear history of occupational or other exposure to organophosphate pesticides or sheep dip. The likelihood of a deliberate release increases with the number of cases that are linked in time and place.

Acute severe organophosphate poisoning is rare in the UK.

In the event of a covert release, particularly one producing only minor casualties, epidemiological studies may help determine the time and location of release.

4.2 Case definition

Careful registration of cases is important.

4.2.1 Possible case

- Patient reporting possible exposure with mild or moderate symptoms, probably not admitted for continuing medical care.

4.2.2 Probable case

- Patient reporting exposure and is symptomatic, likely to have required hospital care

4.2.3 Confirmed case

- Symptomatic patient with exposure requiring hospital care with depressed red cell acetyl cholinesterase or plasma pseudocholinesterase. (Not all will have required intensive care or admission so long as laboratory data is available on the patient)

4.3 Public Health action

4.3.1 Removal from exposure

Minimisation of harm by removal from exposure is probably the most important public health measure. Evacuation from highly contaminated areas is essential and is likely to be undertaken by the emergency services (or by self-evacuation). Once away from contaminated site consider ways of minimising individual exertions or exercise as this may increase speed of absorption until personal decontamination is undertaken.

4.3.2 Decontamination

Decontamination is essential to minimise the harm to the patient and to reduce the risk of secondary contamination and prevent secondary exposures and casualties.

4.3.3 Security

In order to function effectively, medical facilities and staff must not become contaminated. Site security must be considered to prevent access by patients prior to decontamination.

4.3.4 Epidemiological studies

The value of obtaining epidemiological data from patients attending is immense. A draft questionnaire has been provided to hospital trusts (Hospital Chemical Incident Response) and further advice may be issued. Health Authorities may wish to collaborate with acute trusts in collating these data.

4.4 Environmental Hazard summary

- Nerve agents are moderately soluble in water and highly soluble in lipids[1]
- Nerve agents are slowly hydrolysed in the environment to less toxic and non-toxic substances
- The physical properties of the 'G' nerve agents (Tabun GA, Sarin GB, and Soman GD) allow evaporation and they are 'non-persistent'. VX has a much lower vapour pressure, evaporating only slowly and is therefore persistent.
- Vapours are denser than air and may accumulate in low-lying areas.
- Nerve agents are rapidly detoxified by strong alkalis and chlorinated compounds

  Drinking Water Standards: no data available
  Soil Guidelines: no data available
  Air Quality Standards: no data available

5 National specialists

5.1 Laboratory diagnosis

Advice on obtaining assays is available through the NPIS and on TOXBASE.

Red Cell Acetyl Cholinesterase

Few laboratories in the UK provide a service to measure red cell cholinesterase. These include:

***Glasgow*, Department of Clinical Biochemistry, Royal Infirmary**

Head of Department: Dr. F.J. Dryburgh

Contact name: Duty Biochemist

Postal Address for specimens:
Department of Clinical Biochemistry
Glasgow Royal Infirmary University NHS Trust
84 Castle Street
Glasgow G4 0SF

Tel No (voice): 0141 211 4638 - Duty Biochemist
Tel No (fax): 0141 553 1703
Email :f.dryburgh@clinmed.gla.ac.uk

Accreditation status (1/1/96): Full Accreditation (CPA 0158)

***Sheffield*, Health & Safety Laboratory, Health & Safety Laboratory**

Head of Department: Dr. K. Wilson

Contact name: Mr H.J. Mason

Postal Address for specimens:
Biomedical Sciences Group
Health & Safety Laboratory
Broad Lane
Sheffield S3 7HQ

Tel No (voice): 0114 289 2716
Tel No (fax): 0114 289 2768
Email :howard.mason@hsl.gov.uk

Accreditation status (1/1/96): See "Any Other Information"

***London*, Medical Toxicology Unit (Trace Elements Laboratory),**

Head of Department: Dr B Widdop

Contact name: Mr I M House

Postal Address for specimens:
Medical Toxicology Unit
Avonley Road
London
SE14 5ER

Tel No (voice): 020 7771 5372
Tel No (fax): 020 7771 5373
Email :element@gstt.sthames.nhs.uk

Accreditation status (1/1/96): Provisional Accreditation

Cardiff, Therapeutics and Toxicology Centre, Cardiff and Vale NHS Trust

Head of Department, Dr AD Hutchings

24 hour service

Postal Address for specimens:

Toxicology labs, UWCM Therapeutics and Toxicology Centre
Academic Centre
Llandough Hospital
Penlan Road
Penarth
CF64 2XX

Tel: 029 20 711711 (switch) Tel: 029 20 711711
Fax: 029 20 350142
Email deborah.williams@lhct-tr.wales.nhs.uk
Web http://www.uwcm.ac.uk/uwcm/llandough/ttc

Plasma cholinesterase

Less helpful but still useful information can be obtained by measuring cholinesterase. The list of laboratories able to do this are listed below:

Cholinesterase	Serum	London, Clinical Biochemistry, Lewisham Hospital
Cholinesterase	Whole Blood	Nottingham, Chemical Pathology, Queen's Medical Centre
Cholinesterase	Serum	Nottingham, Clinical Chemistry, City Hospital
Cholinesterase	Plasma – Lithium Heparin	Sheffield, Chemical Pathology & Neonatal Screening, Children's Hospital
Cholinesterase	Serum	Sheffield, Health & Safety Laboratory
Cholinesterase	Whole Blood	BIRMINGHAM, CLINICAL CHEMISTRY,
Cholinesterase Activity	Serum	Bristol, Lewis Laboratory, Southmead Hospital
Cholinesterase Phenotyping	Serum	Nottingham, Clinical Chemistry, City Hospital
Cholinesterase Genotype, DNA	Whole Blood - EDTA	Bristol, Lewis Laboratory, Southmead Hospital
Cholinesterase Genotyping: chromosome 3q26.1-q26.2, mutations in exon 4	Whole Blood - EDTA	Karlsruhe,Laboratory Prof. Seelig
Cholinesterase Phenotype	Serum	Bristol, Lewis Laboratory, Southmead Hospital
Cholinesterase Phenotype	Serum or Lithium Heparin Plasma	Glasgow, Clinical Biochemistry, Royal Infirmary
Cholinesterase Phenotype	Serum	Southampton, Chemical Pathology, General Hospital
Cholinesterase Phenotype	Serum	Sunderland, Biochemistry Dept, District General Hospital
Cholinesterase Studies	Plasma	Brighton, Pathology, Royal Sussex County Hospital
Pseudocholinesterase	Serum	Penarth, Welsh National Poisons Unit, Llandough Hospital
Pseudocholinesterase	Plasma – Lithium Heparin	Sheffield, Chemical Pathology & Neonatal Screening, Children's Hospital

5.2 Treatment

Agency	24 hour number	Area served
National Poisons Information Service	0870 600 6266	UK
Chemical Incident Provider Units		
Chemical Incident Response Service, London	0207 635 9191	Eastern, London, South East, South West, North West, Trent Regions
Chemical Hazard Management and Research Centre, Birmingham	0207 394 5112	West Midlands Region
Chemical Incident Service, Newcastle	0191 222 7195 (office) 0191 230 3761 (out of hours)	Northern and Yorkshire Region
Chemical incident Management Support Unit, Cardiff	029 2071 5278	Wales and Northern Ireland
Scottish Centre for Infection and Environmental Health	0141 300 1100 (office hours – ask for on call consultant) 0141 211 3600 (out of hours)	Scotland
Other		
National Focus for Chemical Incidents	08701 545654	UK
Regional Health Emergency Planning Advisers		UK
Emergency Planning Co-ordination Unit, Department of Health, England	020 7210 5771	UK

6 References

1 Hall AH & Rumack BH (Eds). TOMES System ? Micromedex, Englewood, Colorado. CD ROM. Vol 41 (Expires 31 July 1999)

2 Suzuki T, Morita H, Ono K, Maekawa K, Nagai R, Yazaki Y, 1995. Sarin poisoning in Tokyo subway (letter). Lancet; 345(1): 980

3 Okumura T, Takasu N, Ishimatsu S, Miyanoki S, Mitsuhashi A, Kumada K, Tanaka K, Hinohara S, 1995. Report of 640 victims of the Tokyo Subway Sarin attack. Ann Emerg Med; 28(2): 129-135