



Australian Government

Department of Health

Australian clinical guidelines for acute exposures to chemical agents of health concern

A guide for the Emergency Department staff

Second edition, September 2015



**Australian Clinical Guidelines for Acute Exposures to Chemical Agents of Health
Concern: A Guide for the Emergency Department Staff**

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Australian health protection principal committee

The Australian Health Protection Principal Committee (AHPPC) is a standing committee of the Australian Health Ministers' Advisory Council. Chaired by the Chief Medical Officer of the Department of Health, the Committee includes representation by the Chief Health Officers of all States and Territories, the Department of Defence, Emergency Management Australia (EMA), the Chairs of its key standing committees: Communicable Disease Network Australia (CDNA); Public Health Laboratory Network (PHLN); Environmental Health Standing Committee (enHealth); National Health Emergency Management Standing Committee (NHEMS); Antimicrobial Resistance Standing Committee (AMRSC) and the Blood Borne Virus and Sexually Transmitted Infections Standing Committee (BBSTISC) and key subject matter experts.

To obtain details regarding AHPPC publications, contact email ahppc.secretariat@health.gov.au.

At the time of publication, the links to websites referred to in this document were correct. AHPPC acknowledge that, at times, organisations change internet addresses, or remove information from the internet.

Foreword

Improved telecommunications and transportation have led to increased mobility, accessibility and diversity around the world. Undoubtedly, all these have led to the growing threat of chemical, biological and radiological terrorism and the advent of new weapons. While biological and radiological agents pose serious threats, chemical agents are easier to fabricate and can produce the desired acute impact.

There are various versions of clinical guidelines on chemical warfare agents, mostly with a non-Australian focus. New agents are added every day and many industrial and commercial chemicals of interest are not covered. The Australian Clinical Guidelines for Acute Exposures to Chemical Agents of Health Concern: A Guide for the Emergency Department Staff (Chemical Guidelines) have been produced in collaboration with various Australian medical specialists, with the intention to provide health facilities around Australia with standardised management of chemical warfare, and toxic industrial chemical agent exposure, which may occur in a disaster. Where possible, a consensus has been achieved between practicing specialists. The Chemical Guidelines do not however necessarily represent the views of all the clinicians in Australia.

It is important to note that the Chemical Guidelines do not constitute a textbook and therefore deliberately provide little, if any, explanation or background to the chemicals and treatment outlined. They are designed to acquaint the reader rapidly with the chemical and the clinical picture it can produce, thereby providing practical advice regarding assessment and management. The recommendations contained in these guidelines do not indicate an exclusive course of action or serve as a standard of medical care. Variations, taking individual circumstances into account, may be appropriate.

The authors of these Chemical Guidelines have made considerable efforts to ensure the information upon which they are based is accurate and up to date. Users of these guidelines are strongly recommended to confirm that the information contained within them is correct by way of independent sources. The authors accept no responsibility for any inaccuracies, information perceived as misleading, or the success of any treatment regimen detailed in the guidelines.

The authors encourage all clinicians, hospital and health care managers to make themselves aware of these Chemical Guidelines and to become adequately prepared to provide a suitable response to a chemical event within their area.

Acknowledgement

Working group

A project of the scale of the Chemical Guidelines is an accomplishment of many people from many disciplines and skills. The Chemical Guidelines passed through many phases each of which required the cooperation and help of distinctive individuals and organisations.

We would like to extend our appreciation to the writing group of the Chemical, Biological, Radiological, Nuclear (CBRN) Technical Panel, a subcommittee of the National Health Emergency Management Standing Committee (NHEMS). The writing group members comprise of:

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- Dr David Simon, Scientific Services Branch, Public Health Services, SA Health, SA.
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Authority

The Australian Clinical Guidelines for Acute Exposures to Chemical Agents of Health Concern: A Guide for the Emergency Department Staff (Chemical Guidelines) was developed by the Chemical Biological Radiological Nuclear (CBRN) Technical Panel of the National Health Emergency Management Standing Committee (NHEMS). NHEMS is a standing committee of the Australian Health Protection Principal Committee (AHPPC).

The Chemical Guidelines were endorsed by the AHPPC on 1 September 2015.

The Chemical Guidelines have been developed under the auspices of the National Health Emergency Response Arrangements (NatHealth Arrangements 2009).

Professor Chris Baggoley
Chair AHPPC
Australian Government Department of Health

Abbreviations

Acronym	Descriptions
ABC	Airway, breathing, circulation
ABGs	Arterial Blood Gases
ADT	Adult Diphtheria Tetanus Vaccination
AFP	Australian Federal Police
AL	Aluminium
AHPPC	Australian Health Protection Principal Committee
ARDS	Adult Respiratory Distress Syndrome
AXR	Abdominal X-ray
BAL	British Anti-lewisite (Dimercaprol)
Ca	Calcium
CBR	Chemical, Biological and Radiological
CK	Creatinine Kinase
Chemical Guidelines	Australian Chemical Guidelines for Acute Exposures to Chemical Agents of Health Concern: <i>A Guide for the Emergency Department Staff</i>
CMP	Calcium, Magnesium and Phosphate
CNS	Central Nervous System
COHb	Carboxyhaemoglobin
COAG	Coagulation
CT	Computed Tomography
CW	Chemical Weapon
CPAP	Continuous Positive Airway Pressure
CXR	Chest X-ray
DIC	Disseminated Intravascular Coagulopathy
DMPS 3	Dimercapto-1-propanesulfonic acid
DMSA	Dimercaptosuccinic acid
ECG	Electrocardiograph
ED	Emergency Department
EG	Ethylene glycol
EMA	Emergency Management Australia
EtOH	Ethanol
EUC	Electrolytes, Urea and Creatinine

Acronym	Descriptions
FBC	Full Blood Count
G6PD	Glucose-6-phosphate dehydrogenase
GIT	Gastrointestinal Tract
GP	General Practitioner
HAZMAT	Hazardous Materials
HF	Hydrofluoric Acid
Hg	Mercury
HPA	Health Protection Agency, United Kingdom
K	Potassium
LFTs	Liver Function Tests
LOC	Loss of Consciousness
mcg	Microgram
MetHb	Methaemoglobin
Mg	Magnesium
MRI	Magnetic Resonance Imaging
Na	Sodium
NADPH	Nicotinamide adenine dinucleotide phosphate (reduced form)
OPIDN	Organophosphate-induced delayed neuropathy
OSHA	Occupational Safety and Health Administration, U.S. Department of Labour
PAPR	Positive Air-Purifying Respirator
PEEP	Positive End-Expiratory Pressure
PEFR	Peak Expiratory Flow Rate
PFIB	Perfluoroisobutene
PFTs	Pulmonary Functions Tests
PPE	Personal Protective Equipment
ppm	Concentration expressed as parts per million
QT	Interval between the Q wave & T wave in an ECG
QTc	Corrected QT interval, derived using the formula $QTc = QT/\sqrt{RR}$
RBCs	Red Blood Cells
RN	Registered Nurse
ST	ST-segment elevation
T wave	The period of ventricular repolarisation on an ECG
Zn	Zinc

Chapter 1 – How to use this document

Background

There are various versions of clinical guidelines available on chemical warfare agents, mostly with a non-Australian focus. With new agents adding to a growing list on a daily basis, industrial and commercial chemicals of interest are frequently not covered. These Chemical Guidelines have been produced in collaboration with various Australian medical specialists, with the intention to provide health facilities around Australia with standardised management guidance for chemical warfare, toxic industrial and commercial chemical agent exposure, which may occur in a disaster.

Scope of the Chemical Guidelines

Rather than be seen as a standalone framework, it is envisaged that the following Chemical Guidelines may be used at health facilities, especially in Emergency Departments nationwide, to supplement more specific local Chemical, Biological or Radiological (CBR) disaster and response plans. Therefore, the user should be familiar with the local arrangements and responsibilities of the agencies involved and adapt to the context of their jurisdiction.

In addition, the Chemical Guidelines are also intended to provide an easy to read and concise generic plan for the management of the intentional use of chemical weapons and other industrial/commercial chemicals of concern. Where possible, a consensus has been achieved between practising specialists. The Chemical Guidelines do not however necessarily represent the views of all the clinicians in Australia. At the same time, these Chemical Guidelines do not in any way replace the Poisons Information advice currently available.

Chapter 2 – Hospital management of a chemical event

Scope of chemical events

- Acute chemical emergencies can occur as a result of an industrial disaster, occupational exposure, recreational mishap, natural catastrophe, chemical warfare, criminal acts and acts of terrorism.
- For the purpose of these guidelines, the emphasis has been put on acute chemical emergencies due to chemical warfare, criminal acts and acts of terrorism.
- Potential targets where a chemical agent may be used as a weapon or warfare agent include:
 - Governmental Facilities;
 - Medical Facilities;
 - Embassies and Diplomatic residencies;
 - Train Stations;
 - Airports;
 - Universities and Schools;
 - Shopping Centres;
 - Stadiums;
 - Cinemas; and
 - Other crowded places.

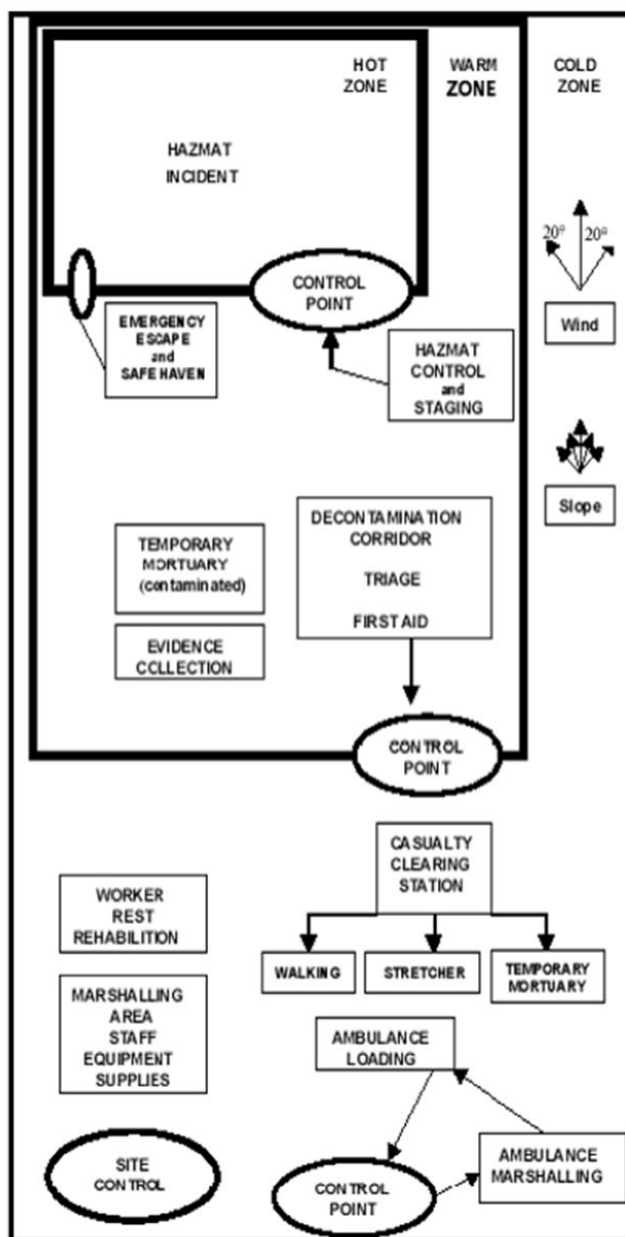
Event recognition

- General indicators of possible chemical agent use are (adapted from the website of the Australian Federal Police (AFP)):
 - **Mass casualties** – Health problems including nausea, disorientation, difficulty in breathing, convulsions and death.
 - **Patterns of casualties** – Casualties will likely be distributed downwind, or if indoors, by the ventilation system.
 - **Blisters/rashes** – Numerous individuals experiencing unexplained water-like blisters, wheals and /or rashes.
 - **Dead animals/fish** – Numerous animals dead in the same area.

- **Unexplained odours** – Smells ranging from fruity to flowery, sharp/pungent, or garlic/horseradish like bitter almonds. All smells will be completely out of character for the surroundings¹.
 - **Unusual liquid droplets** – A number of surfaces exhibit oily droplets/film. Water surfaces may also have an oily film on the surface.
 - **Dead/withered vegetation** – Trees, bushes, food crops and/or lawns that are dead, discoloured or withered, without drought conditions.
 - **Low-lying clouds** – Unusual low-lying cloud and fog-like conditions.
- Using the above indicators, these guidelines have assumed that a chemical incident has occurred. Hazardous materials teams (HAZMAT) will be involved at the scene to isolate the dangerous area and initiate triage and decontamination.
 - The guidelines also assume that the HAZMAT guidelines have been followed. If a disaster has been declared, the guidelines herein apply to the transport, triage, decontamination and medical management of patients after they have been evacuated, triaged and decontaminated within the regulated zones (e.g. 'hot', 'warm' and 'cold' zones) at the scene of the incident. See [figure 1](#) for HAZMAT management of the incident.
 - However, following a chemical incident with or without an associated fire or explosion, many patients will bypass the scene disaster management procedures and present themselves directly at Emergency Departments (ED). It is also highly likely that such patients may present before a healthcare facility is aware an incident has taken place. Please refer to [figure 2](#) for the patient management flowchart at the hospital and [figure 3](#) for a brief overview of the management of patients who present to ED without prior warning of a possible CBR event.
 - Although moving to fresh air and removing contaminated clothing achieves most of the decontamination required, for such ambulant patients, formal triage and decontamination is mandatory at the treating hospital immediately prior to entry into the hospital itself. This prevents the possible accumulation of a significant amount of chemical from large numbers of people with residual amounts grouping together (cumulative effect especially in an enclosed space).

¹ Note: Some chemical agents have no odour. The presence or absence of odour is not a reliable indicator of chemical release.

Figure 1: HAZMAT management at the incident scene (Reproduced from EMA – Health aspects of CBR hazards)



Activation of CBR disaster plan at hospitals

- This involves the same criteria as the hospital mass casualty disaster plans. Please refer to individual hospital and state disaster plans.
- Additional actions (according to the disaster plans of each hospital) could include:
 - **Alerting security personnel and liaising with Police, Ambulance and Fire Service.**
 - **Donning personal protective equipment.**

- **Preparatory briefing of staff** to familiarise them with the processes of triage, decontamination and treatment outlined in these guidelines.
- **Contacting medical physicist or radiation personnel** (according to hospital radiation safety protocol) so that arriving patients may be screened for the presence of radioactive contamination (if required). This step is recommended when assessing patients from a suspicious or unexplained blast.

Arrival of casualties

- **'Walking wounded'/Ambulatory patients**
 - Such patients may arrive sporadically or all at once. It is important to note that if the incident occurs geographically close to the hospital, ambulatory patients may arrive, without any prior incident site triage or decontamination, before ambulance patients.
 - It is vital that triage and decontamination of these patients occur at the designated decontamination facility of the hospital, which is usually near the ED but away from its entrance, so that free access to the ED for ambulances and emergency vehicles can be maintained.
- **'Worried well'**
 - As the news of a chemical incident is aired by media or gets around the public, 'worried well' public members may start to arrive sporadically or all at once. Each hospital needs to have contingency plans in place to deal with them. Work closely with Security staff members and Police.
- **Ambulance cases**
 - These patients should be met in the ambulance bay by a primary triage team (e.g. Triage RN and ED Doctor). See [figure 2](#).
- **Non-incident patients**
 - While the ED is treating disaster casualties, seriously ill non-disaster patients may arrive (e.g. cardiac arrest, acute myocardial infarction). These patients should be kept separate from potentially contaminated patients if possible. Separation is no longer required following completion of decontamination.

Deceased patients

- Normal Coronal and Disaster Victim Identification procedures should be followed.
- Patients deceased at the incident scene should not be taken to ED for certification of death.
- A holding area for deceased patients separate to normal mortuary facilities will need to be established.

- Patients who die in the pre-hospital setting should be taken directly to the dedicated receiving facility, as per normal Coronial arrangements.
- Patients who die in the hospital should not be transported to the hospital's normal mortuary. Instead, the bodies should be transported to a dedicated temporary facility.
- In large hospitals, such a temporary facility may be established on site. However, at smaller hospitals provisions may need to be made to transport the bodies of deceased patients to a dedicated facility off-site.
- The handling of the bodies of deceased patients will depend on the suspected agent(s) involved. Advice should be sought from the State Health Disaster Co-ordinator (or equivalent).

Decontamination

- It is possible that patients may have already undergone formal and complete decontamination at the scene of the incident.
 - If these decontaminated patients are then transported to a health care facility, they may be considered to have been decontaminated and do not require further decontamination at the health care facility.
- However, if there remains any doubt as to the adequacy of the initial decontamination measures, further decontamination is indicated prior to entry into the ED.
 - In addition, further specific decontamination efforts may be indicated based upon an individual assessment (e.g. further lavage of painful eyes after exposure to a chemical irritant); or on receiving advice that the chemical is a persistent agent.
- It is vital that a system is developed to enable the contemporaneous documentation of triage information, labelling and management of personal effects, decontamination procedures and medical interventions for each patient.

Hospital management of contaminated ambulatory patients

- Ambulatory patients, or the 'walking wounded', who have not been decontaminated at the scene of the incident by HazMat personnel, may exhibit only mild or no effects of the agent. They must be decontaminated prior to entry into the ED.
- This usually involves showering of multiple patients at a facility outside the ED. It is recommended that the sexes are segregated and the area is screened from passers-by.
- The showering facility should have a walk-through arrangement to facilitate rapid patient throughput.
- The following steps are required:
 - Patients need to be identified and registered using the disaster patient record packs.

- Some jurisdictions recommend wetting fully dressed patients to minimise off-gassing.
- Patients fully undress, or at least undress to their underwear.
- Patient's clothes and valuables placed in a sealed plastic bag with an identification label and then placed in secure storage.

Note: The hospitals may need to consider a requirement for 2 bags per person as well as the safe storage of the personal items i.e. one for clothes and other things; another one for items that are indispensable such as car keys, mobile phones, home keys etc.

- Consider whether clothing and personal effects need to be treated as evidence, and maintain chain of custody.
- Patients shower with soap and water.
- Following decontamination, patients proceed provided with gowns/other clothing to secondary triage.

Hospital management of contaminated non-ambulatory patients

- The decontamination process should occur away from the entrance of the ED in the designated decontamination facility of the hospital and occur simultaneously to initial resuscitative measures.
- The following steps are required:
 - Patients need to be identified and registered using the disaster patient record packs.
 - Resuscitation and decontamination started concurrently by attending medical and nursing staff with appropriate PPE.
 - Patients undressed completely and washed using soap and water sponging or spraying. The wash-down technique might include the use of a soap dispenser and a hand-held shower or spray unit to wash the patient over a 1 to 2 minute period. Particular attention should be paid to skin folds and dependent areas.
 - Patient's clothes and valuables placed in a sealed plastic bag with an identification label and then placed in secure storage.

Note: The hospitals may need to consider a requirement for 2 bags per person as well as the safe storage of the personal items i.e. one for clothes and other things; another one for items that are indispensable such as car keys, mobile phones, home keys etc.

- Consider whether clothing and personal effects need to be treated as evidence, and maintain chain of custody.

- Following initial medical stabilisation and decontamination, patients are reclothed (gowns or similar) and proceed to secondary triage.

Figure 2: Patient management flowchart for the hospital

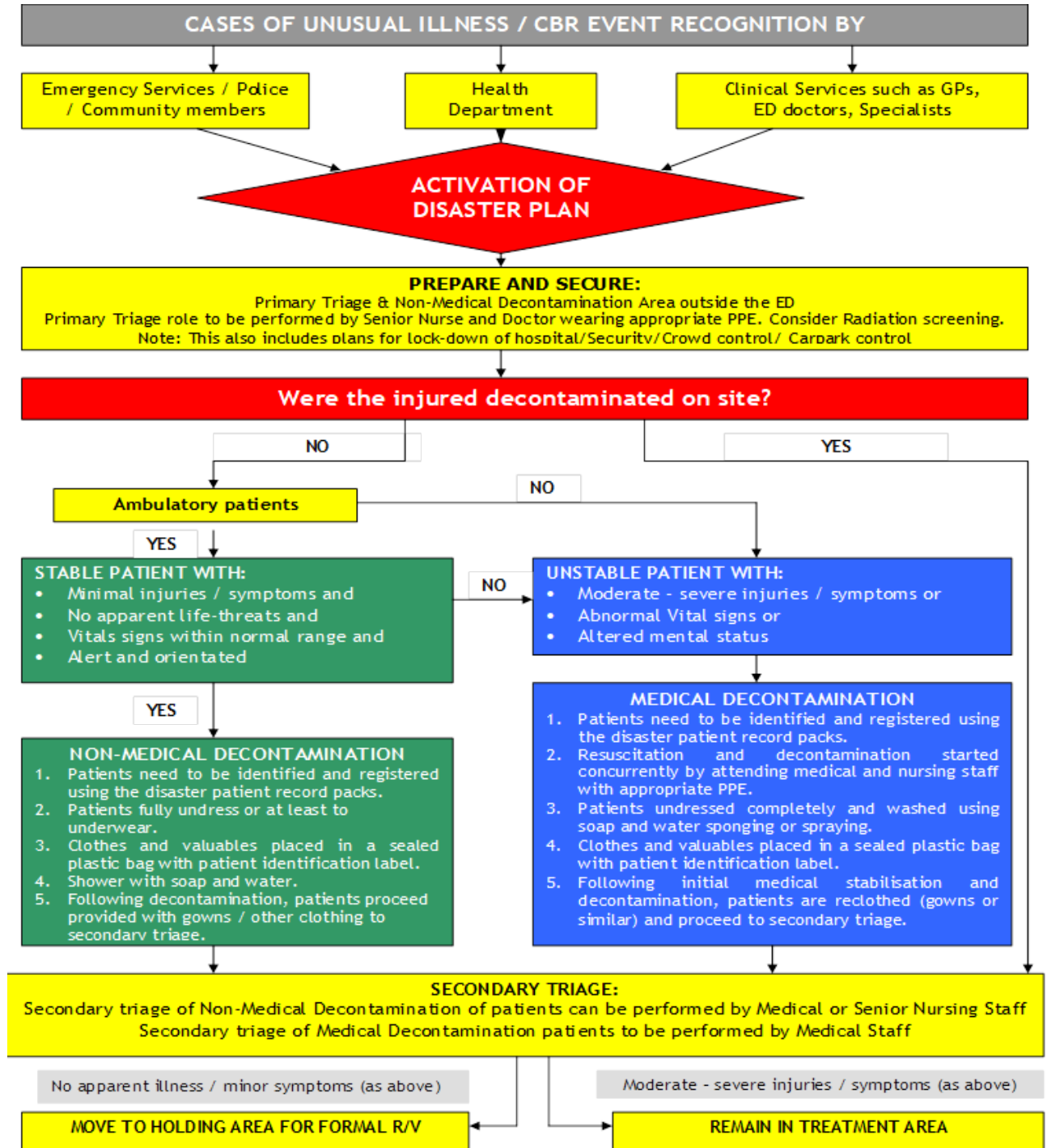
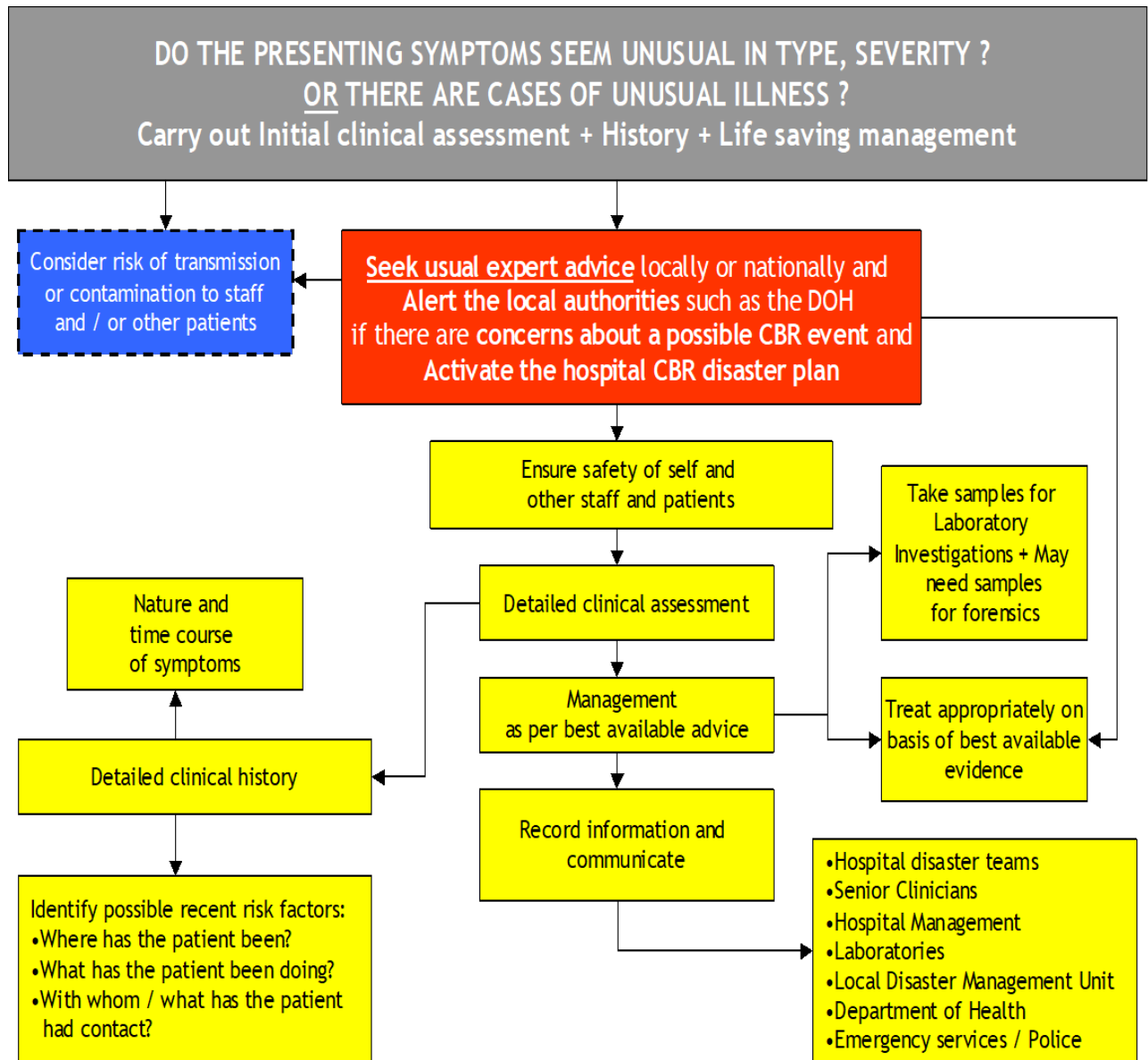


Figure 3: Brief overview of the management of patients who present to ED without prior notification or information of a possible CBR event



(Modified from the flowchart 3 in Guidance for Hospital Clinicians, issued by Health Protection Agency, United Kingdom (HPA) Version 3, March 2004)

Chapter 2.1 – Emergency Department management of an unidentified chemical agent

This section provides a basic patient management plan in a tabular format for an unidentified chemical agent. Please refer to [figure 4](#) for a flowchart on the same.

If a patient presents with exposure to an unidentified chemical agent and a specialist Hazmat Scientist or Chemist is not available then ED staff should make all attempts to determine the identity of the chemical agent through container shapes, labels, shipping papers and analytical tests or get a detailed history from the Emergency Services. Please note that [chapter 2.3](#) lists the common clinical toxidromes and chemical agent classifications that can also be used to help identify unknown chemical agents.

Note: The overall basic patient management is similar for all chemical poisonings. However, the efficacy of certain treatments will depend on the identity of chemical agent, as then a specific medication or antidote may be administered. Always seek expert advice from the Poisons Information Centre (13 11 26) when in doubt.

Safety

- Ensure safety of self and other staff and patients if there are cases of unusual illnesses or a patient presents with unusual symptoms.

Standard precautions

- Standard precautions in health care settings consist of the following work practices:
 - Aseptic technique for all invasive procedures, including appropriate use of skin disinfectants;
 - Personal hygiene practices, particularly hand washing and drying before and after all significant patient contacts;
 - The use of 70% alcohol-based chlorhexidine (0.5%) hand rub solutions as an adjunct to hand washing;
 - Use of personal protective equipment, which may include gloves, impermeable gowns, plastic aprons, masks/face shields and eye protection;
 - Appropriate handling and disposal of sharps and other clinical waste;
 - Appropriate reprocessing of reusable equipment and instruments, including appropriate use of disinfectants; and
 - Environmental controls, including design and maintenance of premises, cleaning and spill management including appropriate use of disinfectants.

ABC

- Evaluate and support airway, breathing and circulation.
 - Administer supplemental oxygen.
 - Early use of positive end-expiratory pressure (PEEP) or mechanical intubation may be required in cases of respiratory compromise.
 - Fibre-optic visualisation of the upper and lower airways may help to determine the extent of injury.
 - Be prepared to establish a surgical airway if the patient's condition precludes intubation.
 - Establish intravenous access in seriously ill patients.
 - Cardiac monitoring is essential in seriously ill patients.
 - Standard treatment protocols apply to patients who are hypotensive, comatose, or have seizures or cardiac arrhythmias.

Treatment based on route of exposure

- **Inhalational exposure**
 - Administer supplemental oxygen.
 - Treat bronchospasm with aerosolised bronchodilators.
 - Standard symptomatic and supportive treatment protocols apply.
- **Ingestion exposure**
 - Do not induce vomiting.
 - Gastrointestinal decontamination is not routine and should never undermine resuscitation and supportive care.
 - Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert clinical toxicology advice.
 - Consider CT chest and abdomen, or endoscopy to evaluate the extent of gastrointestinal tract (GIT) injury following ingestion of corrosive agents. Obtain an upright CXR if GIT perforation is suspected.
 - Standard symptomatic and supportive treatment protocols apply.
- **Dermal exposure**
 - Treat chemical burns as thermal burns.
 - Brush off as much chemical as possible.
 - Ensure copious lavage of the affected area takes place.
 - Administer standard topical therapy.

- Ensure adequate analgesia is prescribed.
- Extensive burns may require review by a Burns Specialist, General Surgeon and/or Plastic Surgeon.
- Update Adult Diphtheria Tetanus Vaccination (ADT) status as appropriate.
- Standard symptomatic and supportive treatment protocols apply.

Special circumstances: Hydrofluoric Acid (HF):

- All dermal exposures require use of calcium gels to neutralise the HF. If HF is not neutralised, ongoing tissue necrosis can occur despite minimal external signs. In more serious cases involving hands, Bier's blocks with intravenous calcium have been used.
- Expert advice is warranted in all cases of exposures. Contact the Poisons Information Centre (13 11 26).

- **Ocular exposure**

- Ensure that adequate eye irrigation has been completed. Irrigate with sterile 0.9% saline for at least 15 mins.
- Test the visual acuity and examine the eyes for corneal damage or burns.
- Relieve pain with analgesia.
- Pad the eye.
- Get an ophthalmology consult for patients who have severe corneal injuries or persistent ocular symptoms.

Laboratory/Radiographic/Ancillary testing

- In this setting, screening tests applied to asymptomatic patients rarely alter management and are not routinely indicated.
- Special investigations to refine diagnosis and management in symptomatic patients should be utilised as clinically indicated. Some of the special investigations may include:
 - Full Blood Count (FBC);
 - Electrolytes, Urea and Creatinine (EUC);
 - Glucose/Blood sugar level;
 - Arterial Blood Gases (ABGs);
 - Calcium, Magnesium and Phosphate (CMP);
 - Liver Functions Tests (LFTS) including Coagulation tests;
 - Pulse oximetry;
 - Electrocardiograph (ECG) and /or Cardiac monitoring;
 - Chest X-ray (CXR) /Abdominal X-ray (AXR);

- Pulmonary functions tests (PFTs); and
- 24-hour urine samples.

Hospital admission

- The requirement for hospitalisation is a clinical decision based on normal parameters.

Delayed effects

- When the chemical agent has not been identified, the patient with suspected exposure should be observed for an extended period (at least 6 to 8 hours) or admitted to the hospital as per clinical indications.

Patient discharge

- **Asymptomatic and minimally symptomatic patients**
 - These patients should undergo a secondary triage process and targeted medical evaluation as soon as possible.
 - This procedure must include counselling regarding symptoms to watch out for relating to the exposure and appropriate follow-up, should delayed manifestations occur.

Note:

- It is likely that psychiatric morbidity will occur following any mass casualty incident.
- Contingency plans are required to address potential acute and delayed psychiatric morbidity.

Follow up

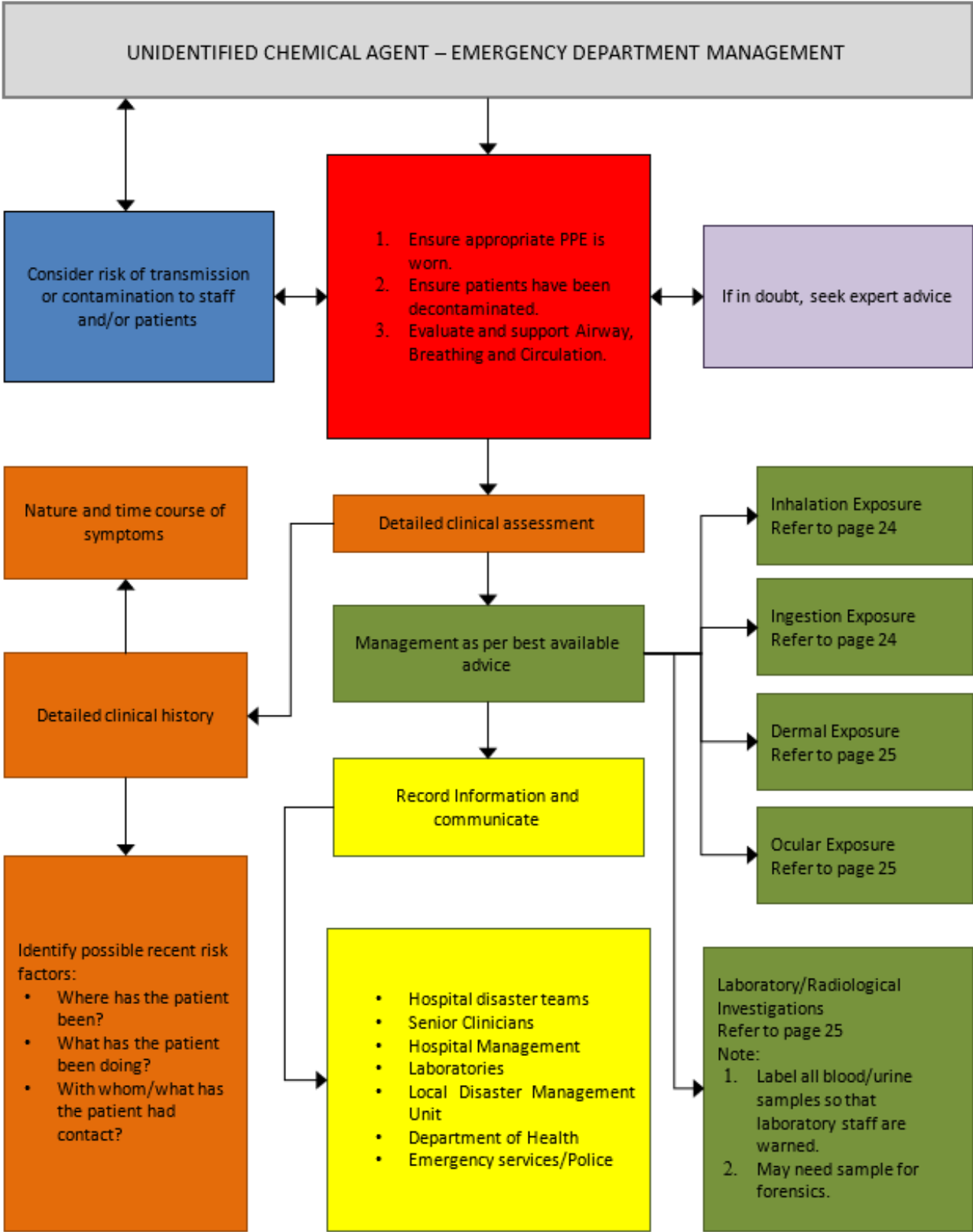
- **Patients**
 - Provide the patient with follow-up instructions to return to ED or see their General Practitioner (GP) to reevaluate initial findings in 48 to 72 hours.
 - Patients who have corneal injuries should be re-examined within 24 hours in ED or by their GP if they are discharged without being admitted to a hospital.
 - Patients with significant dermal burns should be followed up in Burns/Plastics clinic on discharge (will depend on individual hospitals).

Note:

- As previously, contingency plans are required to address potential acute and delayed psychiatric morbidity.

- Staff
 - Some of the staff may have come in contact with the contaminated patients. Ensure they are provided with follow-up instructions to report any health concerns and seek medical help.
 - It is likely that psychiatric morbidity will occur following any mass casualty incident so ensure that the staff are properly briefed and supported.

Figure 4: Emergency Department management of unidentified chemical agent used during a CBR incident



Chapter 2.2 – Personal protective equipment

Personal protective equipment (PPE) is a general term. It includes both the protective clothing used as a standard precaution within healthcare settings and equipment specifically designed to provide chemical protection.

The US Occupational Safety and Health Administration; OSHA Best practices for hospital-based first receivers of victims from mass casualty incidents involving the release of hazardous substances (January 2005) provides the following recommendations:

- 'Hospitals providing emergency response services must be prepared to carry out their missions without jeopardizing the safety and health of their own and other employees. Of special concern are the situations where contaminated patients arrive at the hospital for triage (sorting) or definitive treatment following a major incident.'
- 'Personnel who will be involved in decontamination must be equipped with PPE that is appropriate for the hazardous substances expected to be encountered.'
- 'The hospital's emergency response plan should address:
 - the designation of a decontamination team, including emergency department physicians, nurses, aides and support personnel; and
 - hospital staff use of PPE based on hazards present or likely to be present, routes of exposure, degree of contact, and each individual's specific tasks.
- Government experts, researchers and hospitals alike offer broad support for the use of PAPRs and chemically protective clothing (including gloves, boots and suits with the openings taped closed) for first receivers performing decontamination activities.'

Risk of exposure

In determining the risk of secondary chemical exposure from contaminated casualties it is important to understand the following principles:

- Gas and vapours affecting casualties largely dissipate from the casualty's skin and clothing within five minutes of removal of the casualty to fresh air.
- Removal of clothing reduces contamination by 75 to 90%. This assumes that the skin is covered by typical office type clothing.
- Pre-hospital decontamination eliminates secondary contamination risk
- Secondary exposure of health care personnel depends on:
 - the toxicity of the contaminant on hair, skin and clothes of casualties;
 - the concentration of the contaminant;

- whether splash contamination of casualties occurred, and
- the duration of healthcare worker exposure to victims.

As reported in the OSHA document previously cited, simulations demonstrate exposure levels of unprotected health care personnel during decontamination of mannequins wearing clothes saturated with moderately toxic chemicals were below industrial short-term exposure limits. However, the identification of the specific chemical contaminant is frequently unknown, and in a mass casualty incident health care personnel may have a longer duration of exposure to contaminated casualties.

Around 80% of people affected by a spill of hazardous materials will bypass on-scene decontamination and present directly to hospitals. Whilst the majority may not have significant exposure, health care personnel will have few if any indicators to discriminate between contaminated and uncontaminated individuals in the absence of significant symptoms.

The decision to use personal protective equipment

There is often a delay to the identification of the chemical involved in a particular incident. The decision to use PPE in particular circumstances is often made with minimal supporting information. A precautionary approach is recommended with the use of chemically-protective PPE considered the preferred option.

In hot climates the potential heat stress imposed by PPE is an additional factor to consider. Reliance on support from other agencies is subject to delay as initial resources are directed to the incident scene. In the pre-planning phase, guidance can be sought from local fire service hazardous material specialists regarding cooling strategies for personnel in PPE.

Equipment specifications

Options for PPE include:

- Aerosol and contact precautions with an impervious apron – surgical scrubs, P2 respirator, goggles or face-shield, gown, nitrile gloves and impervious apron. This ensemble has no chemically protective capability and offers minimal protection from water ingress when assisting people during decontamination. This ensemble is not required to manage people who have already undergone decontamination. However, this PPE is appropriate to manage people with ingestion of chemical substances who are not believed to be externally contaminated.
- Chemically-resistant splash suit (splash suit) – a suit made of chemically resistant fabric whose chemical permeation times are known and appropriate to the chemical hazard. The suit should have sealed or fused seams, a concealed zip fastening, and elasticated or fitted

wristbands and ankles. It should have an integrated hood, unless used with a Powered Air-Purifying Respirator with its own loose-fitting hood. It should be worn with chemically resistant gloves and boots. Chemically resistant tape may be used to ensure water-resistant closures, particularly at the wrists.

- Air-Purifying Respirator (APR) – a close fitting full-facemask used with an appropriate filter that offers broad spectrum chemical protection. Achieving an effective seal is crucial to achieve proper protection. An effective seal is not possible with glasses or facial hair or exceptionally small or large faces. As an APR filters air by negative pressure, the work of breathing is increased which may aggravate respiratory conditions and claustrophobia. Training is focussed on correct fitting (donning). The equipment is low maintenance.
- Powered Air-Purifying Respirator (PAPR) – a close fitting full-facemask used with a blower unit with an appropriate filter attached. The filtered air is supplied to the facemask under positive pressure by the blower unit, therefore it is not as important to achieve a perfect seal with the facemask. However, the PAPR should not be used with glasses or facial hair. If the rate and depth of breathing exceeds the blower flow rate, negative pressure will develop inside the facemask and air may leak around the seal. This is less likely if the blower unit flow rate is greater than 115 L/min. Positive pressure also reduces the work of breathing and provides limited cooling enabling the respirator to be better tolerated. The blower unit imposes additional cost, training and maintenance burdens. There is also increased noise from the blower unit affecting the ability to communicate effectively. This can be resolved with compact earpieces and microphones with further cost and training implications.
- Powered Air-Purifying Respirator and loose-fitting hood – a hood made from chemically resistant fabric with attached blower unit and filter. The hood may have a loose neck closure. In order to ensure that positive air pressure is maintained within the hood, the blower unit flow rate should exceed 170 L/min. A hood can provide effective protection irrespective of glasses, facial hair and face shape. Work of breathing, cooling and noise is similar to PAPRs with fitted face-pieces. With the hood, fitting is not a consideration for training, however staff still need to understand the limitations of this respirator and how to operate the blower unit. Cost and maintenance are significant.

Limitations of personal protective equipment

The limitations of PPE may be summarised as:

For the individual:

- Reduced sensory input:
 - narrowed field of vision from the visor of Air-Purifying Respirators and Powered Air-Purifying Respirators;
 - Air-Purifying Respirators;

- reduced sense of touch owing to glove thickness; and
- reduced hearing from the noise when moving in suits with chemically-resistant fabric and from blower units
- Increased difficulty with communication. This is two-fold – muffled speech and hearing difficulty. This combined with the reduced sensory input reduces the capacity to maintain situational awareness.
- Increased risk of tripping owing to unfamiliar footwear and surface conditions, in addition to water underfoot.
- Psychological distress:
 - Due to claustrophobia;
 - Due to lack of confidence in the protective performance of the PPE;
 - Appearing more confronting to patients; and
 - Because of stress of operating in a less familiar way.
- Potential heat related illness due to the relatively impervious fabric and complete coverage of most ensembles preventing evaporation of perspiration. This may manifest as slurred speech, staggering gait, or altered behaviour.
- Difficulty in fitting, and exclusion of those with glasses or facial hair from some configurations of PPE.
- Risk of latex allergy with some materials in respirator options.
- Work performance impacts as a summation of the impacts of previous points. This limits the types of care that can be delivered and reduces the time spent working in PPE.
- Adequacy of donning and doffing procedures.

A safety supervisor can oversee the management of operations in PPE and monitor personnel duration spent in PPE. A 'buddy' system, with staff working in pairs at all times when in PPE, will assist in monitoring staff more closely for early signs of heat illness. Training increases familiarity with the PPE and enables some of these limitations to be addressed. Training is mandatory to ensure safe and effective operations in PPE.

Organisational

- Initial capital cost of the equipment and recurrent expenditure as the shelf-life of components is reached.
- Maintenance and periodic inspection of equipment.
- Storage of the equipment:

- to preserve its effective performance integrity, PPE should be stored in a temperature controlled environment, away from moisture, and in such a way as to minimise damage; and
- sufficient equipment for management of mass casualties requires a significant amount of space.
- Training burden:
 - Having sufficient personnel trained to provide 24/7 coverage;
 - Maintaining competency with a requirement to retrain at least annually; and
 - Standardising training for consistency and interoperability.
- Developing and promoting standards for safe operation in PPE.
- Exercising functions in PPE.

Table 1: Pros and cons of various components of personal protective equipment (PPE)

PPE configuration	Pros	Cons
<p>Aerosol and contact precautions with scrubs, gown and impermeable apron</p>	<p>This is optimal PPE for dealing with radiological and most biological contamination. Health professionals are generally familiar with this equipment. Widely available in hospitals. Lower cost of replacement. Supply chain generally reliable. Minimal maintenance of equipment required. Quiet.</p>	<p>No chemical protective properties P2 respirators require fit testing annually and fit checking on each occasion of use under AS/NZS 1715:2009. Over-familiarity with this equipment may be associated with poor attention to donning and doffing. Incomplete coverage during decontamination procedures. Modest heat burden. Fogging of face shield or goggles likely.</p>
<p>APR</p>	<p>This provides respiratory protection for managing people presenting to hospitals with chemical, biological and radiological contamination when correctly fitted. The face-piece may be suitable for repeated use when appropriately maintained and cleaned. Disassembly and cleaning of face-piece similar to the same task for a bag-valve mask. Minimal maintenance required. Training improves confidence in the equipment, associated procedures, acclimatisation and tolerance to increased work of breathing. APR is silent, however hearing is compromised by the rustle of the fabric of the splash suit.</p>	<p>Health professionals generally unfamiliar with this equipment. Expensive. Low numbers of suppliers. Generally imported. Important to ensure correct filter type is selected. Requires fit testing. Design of face-piece may not fit all facial types unless multiple sizes available (Generally impractical due to cost). Cannot be worn with glasses or facial hair. Increased work of breathing may not be tolerated by staff with respiratory conditions and may aggravate feelings of claustrophobia. Training burden greater than P2 mask. Moderate heat burden. Fogging of visor possible. Used filters should be disposed as hazardous waste.</p>

PPE configuration	Pros	Cons
PAPR with fitted facemask	<p>This provides respiratory protection for managing people presenting to hospitals with chemical, biological and radiological contamination when correctly fitted.</p> <p>Positive pressure airflow may compensate for less than perfect fitting of the face-piece.</p> <p>Protective factor increased compared to APR if flow rate is at least 115 L/min.</p> <p>Reduced work of breathing.</p> <p>Heat burden reduced compared to APR.</p>	<p>Low turnover of equipment may require significant expenditure at end of equipment life.</p> <p>Health professionals generally unfamiliar with this equipment.</p> <p>Considerably more expensive than APR to purchase.</p> <p>May have additional expenditure for disposable or single use consumables.</p> <p>Low numbers of suppliers. Generally imported.</p> <p>Important to ensure correct filter type is selected.</p> <p>Over-confidence in the perceived higher protective features of this equipment may lead to reduced attention to correct donning and doffing.</p> <p>Requires fit testing.</p> <p>Design of face-piece may not fit all facial types unless multiple sizes available (Generally impractical due to cost).</p> <p>Cannot be worn with glasses or facial hair.</p> <p>Training burden greater than APR (fitting of facemask and operation of blower unit).</p> <p>Fogging of visor reduced compared to APR, but not eliminated.</p> <p>External connections, such as hoses, cords, and filters may become dislodged in congested emergency environments.</p> <p>Blower unit noise.</p> <p>Maintenance burden high. Battery recharging requires particular attention.</p> <p>Used filters should be disposed as hazardous waste.</p>

PPE configuration	Pros	Cons
PAPR with loose-fitting hood	<p>This provides respiratory protection for managing people presenting to hospitals with chemical, biological and radiological contamination.</p> <p>No fit testing required.</p> <p>Protective factor increased compared to APR if flow rate is at least 170 L/min.</p> <p>Will fit all facial types.</p> <p>Suitable to be worn with glasses or facial hair.</p> <p>Reduced work of breathing.</p> <p>Heat burden reduced compared to APR</p>	<p>Low turnover of equipment may require significant expenditure at end of equipment life.</p> <p>Health professionals generally unfamiliar with this equipment.</p> <p>Considerably more expensive than APR to purchase.</p> <p>May have additional expenditure for disposable or single use consumables. The hood may be disposable.</p> <p>Low numbers of suppliers. Generally imported.</p> <p>Important to ensure correct filter type is selected.</p> <p>Over-confidence in the perceived higher protective features of this equipment may lead to reduced attention to correct donning and doffing.</p> <p>Training burden similar to APR (operation of blower unit only).</p> <p>Fogging of visor minimised compared to APR.</p> <p>External connections, such as hoses, cords, and filters may become dislodged in congested emergency environments.</p> <p>Blower unit noise.</p> <p>Visor material may distort impairing vision. Storage of hoods with flexible visors requires extra care.</p> <p>Maintenance burden high. Battery recharging requires particular.</p> <p>Used filters should be disposed as hazardous waste.</p> <p>Low turnover of equipment may require significant expenditure at end of equipment life.</p>
Splash suit	<p>Ease of use.</p> <p>Comparatively low cost.</p>	<p>Health professionals generally unfamiliar with this equipment.</p>

PPE configuration	Pros	Cons
		<p>Fabric selection must be appropriate to the risk.</p> <p>Suit must be correctly donned with trouser leg over the boot to prevent water ingress during decontamination.</p> <p>Care needed to ensure adequate closure of zips and taping of wrists and ankles.</p> <p>High heat burden. In particular this may be problematic in hot weather/climate.</p> <p>Single use.</p> <p>Used splash suits should be disposed as hazardous waste.</p>

Table 2: Comparison of issues related to the selection of personal protective equipment (PPE) ensembles

PPE	Protects against	Protective factor	Cost	Consumables	Supply issues	Training burden	Maintenance burden	Fit testing	Heat burden@	Fogging	Noise issues – communication impediment	Protection against water	Risk of dislodgement of	Degree of difficulty donning and doffing
Scrubs and P2	R, most B	Nil for chemical	\$	Disposable	Widely available	+	+	Yes	Moderate	Possible	Negligible	Minimal	Unlikely	Low
APR and splash suit	CBR*	High#	\$\$	Filter and suit only	Constrained	++	++	Yes	High	Possible	Rustling of the suit	Good	Unlikely	High
PAPR and splash suit	CBR*	Highest	\$\$\$	Filter, suit, hoses, possibly batteries	Constrained	+++	+++	Yes	Moderate	Reduced likelihood	Significant	Good	Possible	Higher
PAPR with loose-fitting hood and splash suit	CBR*	Highest	\$\$\$	Filter, suit, hoses, possibly hood and batteries	Constrained	++	+++	No	Moderate	Reduced likelihood	Significant	Good	Possible	Low for donning, higher for doffing

* With an appropriate filter

Sufficient for managing people presenting to hospitals with chemical, biological and radiological contamination

@Most of the heat burden comes from inability to evaporate perspiration under the scrubs or splash suit

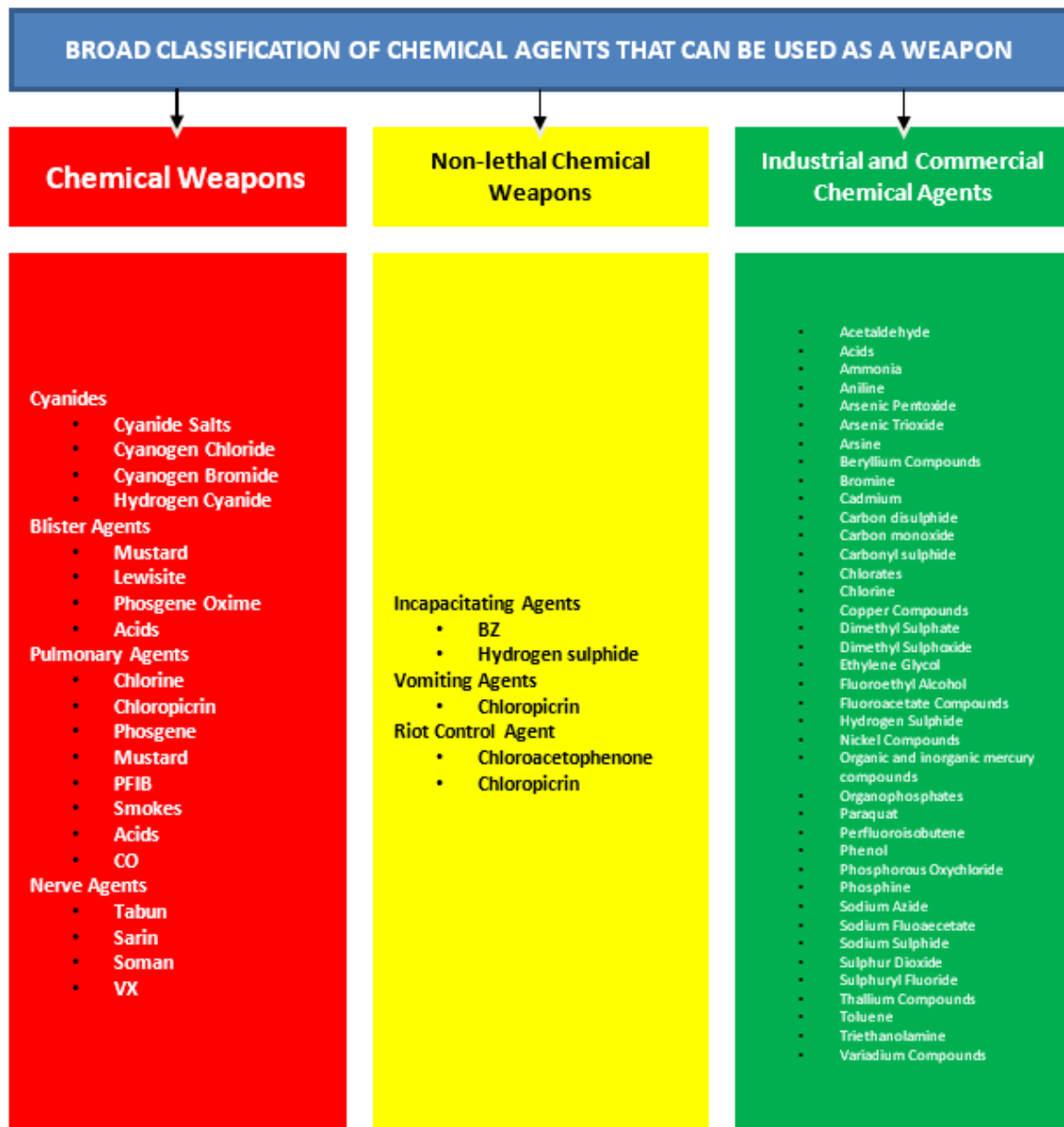
Chapter 2.3 – Aids to chemical agent recognition

The objective of this section is to provide useful clinical information that allows for easy recognition of a chemical agent based on signs and symptoms.

Classification of chemical agents

Figure 5 below lists the broadly classified potential chemical warfare agents used in these Chemical Guidelines.

Figure 5: Broad classifications of chemical agents that can be used as weapons



Clinical toxidromes

Certain chemicals are known to cause a cluster of signs and symptoms, which may sometimes be fairly typical for that chemical or class of chemicals. These clusters are also known as toxidromes. Some of the commonly known toxidromes are described in the table below:

Table 3: Common clinical toxidromes

Toxidromes	Clinical presentation	Potential chemical agents
Skin irritants	Pain, tingle, sting, erythema, dermatitis, burns	Mustard, Phosgene Oxime, Lewisite Acids, Nickel Chloride, Triethanolamine.
Eye irritants	Pain, blurred vision, lacrimation	Organophosphates, Carbamates, Aniline, Acids
Mucus-membrane irritation	Dyspnoea, irritation of skin, eyes, nose, throat, airways	Bromine, Ammonia, Acetaldehyde, Beryllium Compounds, Copper compounds, Acids, Phenol, Sodium Sulphide (hydrated)
Anticholinergic syndrome	'Hot as a hare, dry as a bone, mad as a hatter' Dryness of mouth Flushed, hot, dry skin Dilated pupils Tachycardia Hallucinations, restlessness	3-quinuclidinyl Benzilate (BZ)
Asphyxiation	Nausea, dizziness, weakness, light headedness, air hunger, choking, seizure, coma, death	Cyanides, Arsine, Hydrogen Sulphide, Sodium Azide, Sodium Chlorate, Carbon monoxide
Cholinergic syndrome	DUMBELLS Defecation Urination Miosis Bronchoconstriction Bradycardia Emesis Lacrimation Salivation	Organophosphates, Carbamates, Nerve Agents (VX, Sarin, Tabun, Soman)
Respiratory tract irritation	Dyspnoea, cough, chest pain, haemoptysis	Phosgene, Chlorine, Hydrogen

Clinical presentation of well-known chemical agents

Table 4: Clinical presentation of well-known chemical agents

Agents	Onset of sign and symptoms	Signs	Symptoms
Nerve agents <ul style="list-style-type: none"> • Tabun • Sarin • Soman • VX 	Seconds to hours	<ul style="list-style-type: none"> • Moderate exposure: <ul style="list-style-type: none"> ○ Pinpoint pupils (miosis) ○ Wheezing ○ Stridor ○ Hyper salivation ○ Increased secretions ○ Diarrhoea • High exposure: <ul style="list-style-type: none"> ○ Decreased concentration ○ Loss of consciousness ○ Seizures ○ Respiratory arrest 	<ul style="list-style-type: none"> • Moderate exposure: <ul style="list-style-type: none"> ○ Diffuse muscle cramping ○ Runny nose ○ Shortness of breath ○ Eye pain ○ Dimming of vision ○ Sweating ○ Muscle tremors • High exposure: <ul style="list-style-type: none"> ○ The above plus ○ Sudden loss of consciousness ○ Seizures ○ Flaccid paralysis (late sign)
Blood agents <ul style="list-style-type: none"> • Cyanide salts • Cyanogen chloride • Cyanogen bromide • Hydrogen cyanide • Carbon monoxide 	Seconds to minutes	<ul style="list-style-type: none"> • Moderate exposure: <ul style="list-style-type: none"> ○ Metabolic acidosis ○ Venous blood-O₂ level above normal ○ Hypotension ○ 'Pink' skin colour • High exposure: <ul style="list-style-type: none"> ○ All above signs plus ○ Coma ○ Convulsions ○ Cardio respiratory arrest 	<ul style="list-style-type: none"> • Moderate exposure: <ul style="list-style-type: none"> ○ Palpitations ○ Dizziness ○ Nausea and Vomiting ○ Headache ○ Eye irritation ○ Hyperventilation ○ Drowsiness • High exposure: <ul style="list-style-type: none"> ○ Immediate loss of consciousness ○ Convulsions ○ Death within 1 to 15 minutes
Blister agents <ul style="list-style-type: none"> • Mustard • Lewisite • Phosgene Oxime • Riot control agents • Acids 	Minutes to days	<ul style="list-style-type: none"> • Moderate to high exposure: <ul style="list-style-type: none"> ○ Skin erythema with blistering ○ Watery, swollen eyes ○ Pulmonary oedema ○ Metabolic shutdown ○ Leucopenia and Sepsis 	<ul style="list-style-type: none"> • Moderate to high exposure: <ul style="list-style-type: none"> ○ Burning, itching, or red skin ○ Mucosal irritation (tearing and burning, red eyes) ○ Shortness of breath ○ Nausea and vomiting

Agents	Onset of sign and symptoms	Signs	Symptoms
Pulmonary agents <ul style="list-style-type: none"> • Chlorine • Chloropicrin • Phosgene • Mustard • PFIB • Smokes • Acids 	1–24 hours	<ul style="list-style-type: none"> • Moderate to high exposure: <ul style="list-style-type: none"> ○ Pulmonary oedema ○ Pulmonary infiltrate 	<ul style="list-style-type: none"> • Moderate to high exposure: <ul style="list-style-type: none"> ○ Shortness of breath ○ Chest tightness ○ Laryngeal spasm ○ Mucosal and dermal irritation and redness
Organophosphates <ul style="list-style-type: none"> • Pesticides • Herbicides 	Minutes to days	<ul style="list-style-type: none"> • Muscarinic effects <ul style="list-style-type: none"> ○ Miosis ○ Bradycardia ○ Junctional rhythm ○ Peripheral vasodilation ○ Bronchoconstriction ○ Pulmonary oedema ○ Increased GI motility ○ Increased secretions • Nicotinic effects <ul style="list-style-type: none"> ○ Fasciculation ○ Weakness ○ Paralysis ○ Tachycardia ○ Hypertension • Central nervous system effects <ul style="list-style-type: none"> ○ Mixture of Nicotinic and Muscarinic 	<ul style="list-style-type: none"> • Muscarinic effects <ul style="list-style-type: none"> ○ Abdominal cramps ○ Chest tightness ○ Shortness of breath ○ Diarrhoea ○ Teary eyes ○ Nausea and Vomiting ○ Sweating ○ Urination • Nicotinic effects <ul style="list-style-type: none"> ○ Breathlessness ○ Fasciculation ○ Muscle fatigue/weakness/paralysis ○ Pallor ○ Tremor/twitching • Central nervous system effects <ul style="list-style-type: none"> ○ Anxiety ○ Confusion ○ Ataxia ○ Convulsions ○ Coma ○ Headaches ○ Slurred speech
Acids <ul style="list-style-type: none"> • Nitric acid • Sulphuric acid • Hydrofluoric acid • Hydrochloric acid 	Seconds to hours	<ul style="list-style-type: none"> • Moderate or high exposure: <ul style="list-style-type: none"> ○ Dysphagia ○ Acute abdomen ○ Dyspnoea/haemoptysis ○ Pulmonary oedema ○ Convulsions ○ Respiratory collapse 	<ul style="list-style-type: none"> • Moderate or high exposure: <ul style="list-style-type: none"> ○ Difficulty swallowing ○ Shortness of breath ○ Chest tightness ○ Wheezing ○ Abdominal pain ○ Haematemesis/haemoptysis

Chapter 3 – Individual chemical agents in detail

This chapter provides clinical information to institute appropriate management in a timely fashion for a range of individual chemical agents that can be used as potential weapons. The chemical agents are referenced alphabetically by their most commonly used names and broadly classified into 3 categories (Chemical Weapon, Industrial and Commercial Chemical Agent or Non-lethal Chemical Weapon). For ease of reference, an index for alternative names of agents and their broad classification has also been provided in [Appendix 1](#). For example, it would be evident through this index that Phenol is also known as Benzenol and/or Carboic acid. Expert advice should always be sought when in doubt.

To supplement the use of the Chemical Guidelines, a look-up table ([Appendix 2](#)) has been provided for individual hospitals to note any important phone numbers for easy reference e.g. Local Disaster Unit, Poisons Centre, state or territory Health, On-call Toxicologists, other hospitals etc.

Acetaldehyde

Alternative names	Acetic aldehyde Ethyl aldehyde Ethanal
Properties of the agent	<ul style="list-style-type: none">• It commonly exists as a colourless, irritating, flammable and highly reactive liquid, but it can also be a gas. It is water-soluble. It evaporates quickly at body temperature.• At dilute concentrations, it has a pleasant fruity odour and leafy green taste. At high concentrations, the odour becomes pungent and suffocating.• It is used industrially to produce acetic acid, dyes, foods and beverages.
Routes of exposure	<ul style="list-style-type: none">• Oral – rare• Inhalation – most common• Ocular• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• It is a skin and mucous membrane irritant, which causes a burning sensation of the nose, throat and eyes. It can also cause pulmonary oedema and narcosis. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Sympathomimetic symptoms including hyperventilation, hypertension and tachycardia may be noted at low levels of exposure. Higher levels produce bradycardia and hypotension.• It is a pulmonary irritant and may cause bronchitis and pulmonary oedema and signs and symptoms of chemical pneumonitis when inhaled. Very high concentrations may result in narcosis and respiratory depression. <p>Ingestion</p> <ul style="list-style-type: none">• Liquid acetaldehyde is an emetic. Higher levels may produce the same effects as those of inhalation. <p>Dermal</p> <ul style="list-style-type: none">• Prolonged contact causes erythema and burns. Repeated exposures may cause contact dermatitis. <p>Ocular</p> <ul style="list-style-type: none">• Splash contacts produce painful but superficial corneal injury.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Guided by clinical symptoms.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Monitor for signs of CNS depression.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed until symptoms resolve.

Inhalation

- Give humidified oxygen for hypoxia.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Dermal

- Flush affected areas thoroughly.
- Manage chemical burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with evidence of corneal burn.

Ammonia

Alternative names	Ammonia anhydrous Spirit of Hartshorn
Properties of the agent	<ul style="list-style-type: none">• Ammonia is a colourless, irritant, pungent-smelling gas at room temperature and pressure.• It is strongly water soluble and very alkaline.• It emits several oxides of nitrogen (that have varying toxicity) if heated above 300° C.• It is used as a refrigerant and in the production of fertiliser and explosives.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation – most common• Ocular• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Ammonia produces significant alkaline burns to all exposed surfaces. Symptoms may be delayed for up to 24 hours. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Beware potential airway burns resulting in airway compromise.• Mucosal burns around the nose and mouth may occur.• Chemical pneumonitis with wheeze, difficulty breathing, chest pain and hypoxaemia can occur. <p>Ingestion</p> <ul style="list-style-type: none">• Beware potential airway burns resulting in airway compromise.• Full thickness oral or oesophageal burns may occur.• Nausea and vomiting may occur. <p>Dermal</p> <ul style="list-style-type: none">• Alkaline burns with liquefactive necrosis may occur.• Contact with compressed liquid may cause frostbite. <p>Ocular</p> <ul style="list-style-type: none">• Lacrimation, corneal or conjunctival irritation and ulceration are common; total corneal epithelial loss may occur with temporary or permanent blindness.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Note: A normal CXR and ABG within the first few hours do not rule out the eventual development of chemical pneumonitis.

- Obtain an upright CXR if GIT perforation is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
131126

Asymptomatic patients:

- Asymptomatic patients with a significant history of exposure, consider observing them in hospital or at home. They are to re-present if they develop any symptoms and advise that there is a potential for delayed symptoms of chemical pneumonitis.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 hours.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Give humidified oxygen for hypoxia and bronchodilators for wheeze.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Ingestion

- Do not induce vomiting.
- Make nil by mouth, or permit clear fluids if symptoms mild.
- Administer intravenous fluids as necessary.
- Arrange a gastroenterology review for all suspected oesophageal burns.

Dermal

- Flush affected areas thoroughly.
- Manage corrosive burns as for thermal burns.
- Handle frostbitten areas with caution. Place frostbitten skin in tepid water and allow circulation to re-establish spontaneously.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with evidence of corneal burn.

Aniline

Alternative names

Blue or aniline oil
Cyanol
Phenylamine
Aminobenzene
Aminophen
Benzenamine

Properties of the agent

- It is a clear, oily liquid, which turns brown with exposure to air and light.
- It has a distinctive, amine-like, or musty, fishy odour and an acrid taste.
- It is heavier than water and its vapours are heavier than air; the fumes may be poisonous if inhaled.
- It is combustible and volatile with steam.
- Aniline is used in the manufacture of rubber products, dyes, pigments and pesticides.

Routes of exposure

- Oral
- Inhalation – most common
- Eye
- Dermal

Human toxicity

General:

- Aniline is a respiratory, skin and eye irritant. It is rapidly absorbed by all routes. A metabolite of aniline, phenylhydroxyamine, induces methaemoglobinaemia. Peak metHb levels may occur up to 20 hours after exposure to aniline.
- Symptoms of methaemoglobinemia include signs of hypoxaemia including cyanosis, headache, dizziness, weakness, lethargy, loss of coordination, dyspnoea, coma, seizures and death.
- Haemolytic anaemia may occur. Persons with G6PD deficiency or alcoholism are at particular risk of aniline–induced haemolysis. Heart, liver and kidney effects may be secondary to haemolysis.

Symptoms are based on route of exposure:

Inhalation / Ingestion

- Tachypnoea and tachycardia may be noted.
- Hypoxia, pulmonary infiltrates, respiratory failure and pulmonary hypertension have been attributed to exposure to rapeseed oil denatured in part with aniline as part of the "toxic oil syndrome".
- Severe headache, CNS disturbances and tremor may be noted. CNS depression may be a result of aniline-induced methaemoglobinemia.
- Nausea, vomiting, liver damage and jaundice may occur.
- Haematuria and haemoglobinuria may be seen with haemolysis.

Dermal

- Dermal absorption is rapid. Cyanosis, moderate skin irritation or sensitisation and allergic contact dermatitis may be noted.

Ocular

- Corneal damage, brown discolouration of conjunctiva and cornea, and mild to severe irritation of the eyes may be noted.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, FBC.
- Monitor methaemoglobin levels, haemoglobin, haematocrit and plasma free haemoglobin in patients with methaemoglobinemia. Methaemoglobinemia interferes with pulse oximetry reading, resulting in falsely high values.
- Urinalysis positive for blood with few or no red blood cells (RBC's) is an early indication of haemolysis.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
Note: A normal CXR and ABG within the first few hours do not rule out the eventual development of chemical pneumonitis.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- Asymptomatic patients should be observed for a minimum of 6 hours for delayed onset of methaemoglobinaemia.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.

Inhalation

- Give humidified oxygen for hypoxia.
- Treatment of respiratory failure may include invasive or non-invasive ventilation.
- If evidence of methaemoglobinaemia, 100% O₂.
- Note that pulse oximetry is not reliable, as methaemoglobin and methylene blue interfere with the readings.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with corneal burns.

Antidote for methaemoglobinaemia

- **Methylene Blue** should be administered if the methaemoglobinemia exceeds 20%. 1-2mL/kg methylene blue intravenously over 5 minutes. This can be repeated after 60 minutes.
- Measure methaemoglobin levels hourly until a consistent fall is documented.
- If methylene blue fails to control methaemoglobinaemia, consider exchange transfusion or hyperbaric oxygen therapy.

Arsenic pentoxide

Alternative names

Arsenic (V) oxide
Arsenic acid
Arsenic anhydride

Properties of the agent

- It is an odourless, white crystalline amorphous solid inorganic arsenic compound. It dissolves in water to form Arsenic acid.
- Less soluble and much less toxic than arsenic trioxide, it may be converted to arsenic trioxide in vivo.
- Little is known about the effects of arsenic pentoxide aside from those of arsenic and other arsenical compounds in general.
- It is used to preserve timber.

Routes of exposure

- Oral – most common. Oral toxicity is dependent on the solubility of the arsenical.
- Inhalation – possible.
- Eye – not applicable.
- Dermal – rapidly absorbed and may cause systemic symptoms.

Human toxicity

General:

- Inorganic arsenic inhibits cellular enzymes.
- Presentation depends on the route and type of exposure. In particular, is this an acute, subacute or chronic exposure?

Acute arsenic ingestion has a dose dependent risk assessment >1 mg/kg potentially lethal.

- Generally produces symptoms within 30 to 60 minutes, but onset may be delayed for several hours if ingested with food.
- Hypersalivation, garlicky breath odour.
- GIT: profuse bloody or watery ("rice-water-like") diarrhoea.
- Encephalopathy, seizures
- Cardiovascular collapse, acute cardiomyopathy, prolonged QT
- Renal and hepatic failure.
- Bone marrow depression within 24-72 hours for survivors of acute intoxication.
- Ascending peripheral neuropathy, predominantly motor.

Subacute exposure:

- GIT distress
- Leucopenia
- Haematuria
- Deranged LFTs

Chronic exposure:

- Multi system
- Constitutional symptoms
- Cutaneous lesions: Hyperkeratosis of palms and soles,
- hyperpigmentation, nail changes
- painful peripheral neuropathy
- Malignancies of skin and bladder

Inhalation

- Respiratory tract irritant causing cough, sore throat and dyspnoea.
- Life-threatening pulmonary oedema, acute respiratory failure and adult respiratory distress syndrome (ARDS) have been reported in selected cases.

Dermal

- Irritant or corrosive to skin.
- Sensitisation dermatitis may also occur.

Ocular

- Conjunctivitis, photophobia, and lacrimation may be noted.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, coagulation profile, FBC.
- ECG and continuous cardiac monitoring. Monitor QTc.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Quantitative 24-hour urine collections are the most reliable laboratory measure of arsenic poisoning; however, this test is not useful in acute management and is not readily available in all Australian hospitals. Concentration > 200 mcg/L (0.2 mg/L) indicates potentially harmful exposure.
- Arsenic is radio-opaque and an abdominal film should be obtained
- whenever arsenic ingestion is suspected.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- Patients who remain asymptomatic after exposure require no follow-up.
- Patients with relatively minor or transient symptoms following an ingestion exposure should have a 24-hour urinary collection for arsenic measurement.

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Cardiac monitoring is important.
- Maintain fluid and electrolyte balance.

Inhalation

- Give humidified oxygen for dyspnoea.
- Treat non-cardiogenic pulmonary oedema with PEEP or CPAP.
- Treatment of respiratory failure may include invasive or non-invasive ventilation

Ingestion

- Do not induce vomiting.
- Resuscitation and supportive care of hypovolaemia and shock secondary to gastrointestinal fluid losses.
- Activated charcoal NOT indicated.
- Whole bowel irrigation may be considered in selected patients after discussion with a Clinical Toxicologist if arsenic trioxide seen on AXR.
- Chelation therapy may be indicated in selected cases, where the urinary arsenic level is >200 µg/l with significant gastrointestinal or cardiac symptoms. DMPS is the treatment of choice although not readily available in Australasia. Alternative agents include Dimercaprol (BAL), D-penicillamine and DMSA (Succimer). Do not chelate asymptomatic patients without the guidance of a 24-hour urinary arsenic level.

Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Dermal

- Flush affected areas thoroughly.
- Manage corrosive burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Arsenic trioxide

Alternative names	Arsenic oxide White arsenic Diarsenic oxide Arsenious anhydride Arsenous oxide
Properties of the agent	<ul style="list-style-type: none">• It is the most toxic form of arsenic compound.• It is a white or transparent solid in the form of glassy, shapeless lumps or a crystalline powder that resembles sugar.• It has no odour or taste.• When arsenic trioxide is burnt, it releases toxic fumes and arsine gas, which is highly toxic.• It is used as a reagent in glass manufacture and rarely as a sheep dip.
Routes of exposure	<ul style="list-style-type: none">• Oral – bioavailability 60 – 90%.• Inhalation – bioavailability 60 – 90%.• Eye.• Dermal absorption via mucous membranes or abraded skin.

Human toxicity

General:

- Inorganic arsenic inhibits cellular enzymes.
- Presentation depends on the route and type of exposure. In particular, is this an acute, subacute or chronic exposure?

Acute arsenic ingestion has a dose dependent risk assessment >1 mg/kg potentially lethal.

- Generally produces symptoms within 30 to 60 minutes, but onset may be delayed for several hours if ingested with food.
- Hypersalivation, garlicky breath odour.
- GIT: profuse bloody or watery ("rice-water-like") diarrhoea.
- Encephalopathy, seizures
- Cardiovascular collapse, acute cardiomyopathy, prolonged QT
- Renal and hepatic failure.
- Bone marrow depression within 24-72 hours for survivors of acute intoxication.
- Ascending peripheral neuropathy, predominantly motor.

Subacute exposure:

- GIT distress
- Leucopenia
- Haematuria
- Deranged LFTs

Chronic exposure:

- Multi system
- Constitutional symptoms
- Cutaneous lesions: hyperkeratosis of palms and soles, hyperpigmentation, nail changes
- Painful peripheral neuropathy
- Malignancies of skin and bladder

Inhalation

- Respiratory tract irritant causing cough, sore throat and dyspnoea.
- Life-threatening pulmonary oedema, acute respiratory failure and adult respiratory distress syndrome (ARDS) have been reported in selected cases.

Dermal

- Irritant or corrosive to skin.
- Sensitisation dermatitis may also occur.

Ocular

- Conjunctivitis, photophobia, and lacrimation may be noted.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, coagulation profile, FBC.
- Monitor ECG and QTc.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Quantitative 24-hour urine collections are the most reliable laboratory measure of arsenic poisoning; however, this test is not useful in acute management and is not readily available in all Australian hospitals. Concentration > 200 mcg/L (0.2 mg/L) indicates potentially harmful exposure.
- Arsenic is radio-opaque and an abdominal film should be obtained whenever arsenic ingestion is suspected.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
131126

Asymptomatic patients:

- Patients who remain asymptomatic after exposure require no follow-up.
- Patients with relatively minor or transient symptoms following an ingestion exposure should have a 24-hour urinary collection arsenic measurement.

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Cardiac monitoring is important.
- Maintain fluid and electrolyte balance.

Inhalation

- Give humidified oxygen for dyspnoea.
- Treat non-cardiogenic pulmonary oedema with PEEP or CPAP.
- Treatment of respiratory failure may include invasive or non-invasive ventilation

Ingestion

- Do not induce vomiting.
- Resuscitation and supportive care of hypovolaemia and shock secondary to gastrointestinal fluid losses.
- Activated charcoal is NOT indicated.
- Chelation therapy may be indicated in selected cases, where the urinary arsenic level is $>200 \mu\text{g/l}$ with significant gastrointestinal or cardiac symptoms. DMPS is the treatment of choice, although not readily available in Australasia. Alternative agents include Dimercaprol (BAL), D-penicillamine and DMSA (Succimer). Do not chelate asymptomatic patients without the guidance of a 24-hour urinary arsenic level.
- Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Dermal

- Flush affected areas thoroughly.
- Manage corrosive burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Arsine

Alternative names	Arsenic trihydride Arsenous hydride Arseniuretted hydrogen Hydrogen arsenide
Properties of the agent	<ul style="list-style-type: none">• Arsine is a highly poisonous gas, which binds with oxidised haemoglobin to cause sudden and profound haemolysis with secondary renal failure and tissue hypoxia.• It is a colourless gas with garlic like odour. The threshold for odour detection is much greater than the threshold for toxicity.• It is used in fertiliser manufacture and metal processing.
Routes of exposure	<ul style="list-style-type: none">• Inhalation – most common.
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Arsine may be fatal if inhaled in sufficient quantities, but death may be delayed due to haemolysis and secondary renal failure. There are 3 phases to arsine poisoning: <p>Pre-Haemolysis Phase</p> <ul style="list-style-type: none">○ Serious exposure may produce symptoms in 30 to 60 minutes, but may be delayed for up to 36 hours. Initially, the patient may look and feel relatively well. Garlicky odour of breath may be noted. <p>Haemolysis Phase</p> <ul style="list-style-type: none">○ Early symptoms of haemolysis include generalised weakness, headache, shivering, thirst and abdominal pain. The weakness proceeds to muscle cramps and occasionally hypotension. Anorexia, nausea and vomiting may also occur.○ Haemoglobinuria may occur which can be confirmed by dipstick testing of urine.○ Cardiac dilatation and pulmonary oedema may be seen prior to the onset of renal failure. <p>Post-Haemolysis Phase</p> <ul style="list-style-type: none">○ Haemoglobinuria followed by renal function impairment and occasionally renal shutdown occurs. The haemolysis, if severe, can result in jaundice and bronzing of the skin.○ Persistent haemolysis may result in hyperkalaemia and dysrhythmias.○ Systemic symptoms may be seen, including arsenic encephalopathy.○ Sensori-motor peripheral neuropathy similar to that observed with inorganic arsenic may be delayed 1 to 6 months.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC. Closely monitor potassium, plasma haemoglobin, urinary haemoglobin, and renal function.
- Monitor ECG.
- Arsenic measurements are not relevant for arsine exposure.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- See section on patient discharge and follow-up on page 25.

Symptomatic patients:

- General advice
- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Cardiac monitoring should occur for at least 72 hours.
- Consider haematology consult.
- If major haemolysis has occurred, exchange transfusion may be performed to remove the plasma haemoglobin, in conjunction with haemodialysis to preserve renal function in selected cases following expert advice.
- Chelation therapy is not indicated, as the major toxicity is haemolysis and not arsenic poisoning.

Beryllium fluoride

Alternative names	Beryllium difluoride
Properties of the agent	<ul style="list-style-type: none">• It is a colourless to white or grey powder or crystals with no odour.• It is highly soluble in water.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation• Eye• Dermal
Human toxicity	<p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Irritation of the nose, airways and lungs, causing cough, chest tightness, shortness of breath, nasal discharge, epistaxis, nasopharyngitis and gingivitis may occur.• May develop acute lung injury, including pulmonary oedema. Symptoms may be delayed for up to 3 days. <p>Ingestion</p> <ul style="list-style-type: none">• Fever, tachycardia may be noted.• Metallic taste, nausea, vomiting, diarrhoea and abdominal pain may be noted. <p>Dermal</p> <ul style="list-style-type: none">• Local skin irritation and dermatitis can occur. Wounds from contaminated objects may form deep ulcerations, which are slow to heal and may form granulomata. <p>Ocular</p> <ul style="list-style-type: none">• Eye irritation with redness, discomfort, itching, swelling of lids and photophobia may be noted.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Specific assays for beryllium in lung and granuloma tissue are available in some Australian hospitals. Peripheral lymphocyte or broncho-alveolar lavage fluid cell transformation tests are useful in diagnosis and monitoring but do not help with acute management.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

- General advice
- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Ensure adequate analgesia is prescribed.

Inhalation

- Give humidified oxygen for hypoxia and bronchodilators for wheeze.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Ingestion

- Chelating therapy and haemodialysis are not recommended.

Dermal

- Debride contaminated wounds.

Beryllium oxide

Alternative names	Bertholite
Properties of the agent	<ul style="list-style-type: none">• It is a colourless to white powder or crystals.• It is soluble in water.
Routes of exposure	<ul style="list-style-type: none">• Oral – more common• Inhalation• Eye• Dermal
Human toxicity	<p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Acute pneumonitis with chest pain, bronchospasm, dyspnoea, cough and haemoptysis may be noted. <p>Ingestion</p> <ul style="list-style-type: none">• Fever, tachycardia may be noted.• Metallic taste, nausea, vomiting, diarrhoea and abdominal pain may be noted. <p>Dermal</p> <ul style="list-style-type: none">• Local skin irritation and dermatitis can occur. Wounds from contaminated objects may form deep ulcerations, which are slow to heal and may form granulomata. <p>Ocular</p> <ul style="list-style-type: none">• Eye irritation with redness, discomfort, itching, swelling of lids and photophobia may be noted.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Specific assays for beryllium in lung and granuloma tissue are available in some Australian hospitals. Peripheral lymphocyte or broncho-alveolar lavage fluid cell transformation tests are useful in diagnosis and monitoring but do not help with acute management.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Ensure adequate analgesia is prescribed.

Inhalation

- Give humidified oxygen for hypoxia and bronchodilators for wheeze.
- Treatment of acute pneumonitis may include invasive or non-invasive ventilation.

Ingestion

- Chelating therapy and haemodialysis are not recommended.

Dermal

- Debride contaminated wounds.

Beryllium sulphate

Alternative names	Nil known
Properties of the agent	<ul style="list-style-type: none">• It exists as colourless crystals, which are soluble in water.• It emits sulphur oxides when heated over 550-600°C.• It is also present in emissions from combustion of fossil fuels as found in coal-fired power stations.• It is used in the nuclear and electronic industries.
Routes of exposure	<ul style="list-style-type: none">• Oral – more common• Inhalation• Eye• Dermal
Human toxicity	<p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Acute pneumonitis with chest pain, bronchospasm, dyspnoea, cough and haemoptysis may be noted. <p>Ingestion</p> <ul style="list-style-type: none">• Fever, tachycardia may be noted.• Metallic taste, nausea, vomiting, diarrhoea and abdominal pain may be noted. <p>Dermal</p> <ul style="list-style-type: none">• Local skin irritation and dermatitis can occur. Wounds from contaminated objects may form deep ulcerations, which are slow to heal and may form granulomata. <p>Ocular</p> <ul style="list-style-type: none">• Eye irritation with redness, discomfort, itching, swelling of lids and photophobia may be noted.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Specific assays for beryllium in lung and granuloma tissue are available in some Australian hospitals. Peripheral lymphocyte or broncho-alveolar lavage fluid cell transformation tests are useful in diagnosis and monitoring but do not help with acute management.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Ensure adequate analgesia is prescribed.

Inhalation

- Give humidified oxygen for hypoxia and bronchodilators for wheeze.
- Treatment of acute pneumonitis may include invasive or non-invasive ventilation.

Ingestion

- Chelating therapy and haemodialysis are not recommended.

Dermal

- Debride contaminated wounds.

Bromine

Alternative names	Nil known
Properties of the agent	<ul style="list-style-type: none">• It is a dark, reddish-brown, volatile, fuming liquid with suffocating and irritating fumes.• It has a strong, disagreeable odour resembling chlorine.• Bromine is used in photography, medicines, fumigants and dyes.
Routes of exposure	<ul style="list-style-type: none">• Oral – rare• Inhalation – most common• Eye• Dermal
Human toxicity	<p>Symptoms are based on route of exposure:</p> <p>Note – exposure to any quantity can be dangerous</p> <p>Inhalation</p> <ul style="list-style-type: none">• Severe irritation of the respiratory tract resulting in cough, delayed pulmonary oedema, bronchospasm, chemical pneumonitis, ARDS, glottal spasm and glottal oedema may occur.• Note: Bromine is reported to be a more potent respiratory irritant than chlorine.• Diarrhoea, nausea, vomiting and abdominal pain have been reported following inhalation exposure. <p>Ingestion</p> <ul style="list-style-type: none">• Hypotension and shock may occur after ingestion with corrosive injury and haemorrhage from the GIT.• Mucosal burns, oesophagitis and gastroenteritis have been reported.• Haemorrhagic nephritis, with oliguria or anuria, may develop within 1 to 2 days after oral ingestion of liquid bromine, as sequelae to shock or haemolysis. <p>Dermal</p> <ul style="list-style-type: none">• Dermal burns may be noted. <p>Ocular</p> <ul style="list-style-type: none">• Lacrimation, corneal or conjunctival irritation and ulceration are common.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
131126

Asymptomatic patients:

- See section on patient discharge and follow-up on page 25.

Symptomatic patients:

General advice

- Do not induce vomiting.
- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Give humidified oxygen for hypoxia and bronchodilators for wheeze.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Ingestion

- Do not induce vomiting.
- Make nil by mouth, or permit clear fluids if symptoms mild.
- Administer intravenous fluids as necessary.
- Arrange a gastroenterology review for all suspected oesophageal burns.

Dermal

- Flush affected areas thoroughly.
- Manage chemical burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with corneal burns.

Cadmium

Alternative names	Colloidal cadmium
Properties of the agent	<ul style="list-style-type: none">• It is an odourless, soft, ductile, silver-white, somewhat bluish metal, which becomes brittle at 80oC and tarnishes in moist air.• It is also water soluble and colourless in water.• Cadmium compounds are used as pigments and paints, pesticides, catalysts, stabilisers and in photography.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation• Eye• Dermal – significant dermal absorption seldom occurs
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Cadmium is a severe lung and gastrointestinal irritant that can be fatal by inhalation and ingestion. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• The symptoms of acute poisoning after inhalation exposure may be delayed for 12 to 36 hours. These include chest pain, cough, bloody sputum, difficulty breathing, sore throat, 'metal fume fever' (shivering, sweating, body pains, headache), dizziness, irritability, weakness, nausea, vomiting, diarrhoea, tracheobronchitis, pneumonitis and pulmonary oedema. <p>Ingestion</p> <ul style="list-style-type: none">• After acute ingestion, symptoms usually appear in 15 to 30 minutes. These can include abdominal pain, burning sensation, nausea, vomiting, salivation, muscle cramps, vertigo, shock, unconsciousness and convulsions.• Protracted exposure can lead to renal damage, anaemia and liver injury.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Obtain an ECG.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
Note: A normal CXR and ABG within the first few hours do not rule out the eventual development of chemical pneumonitis.
- Blood cadmium levels are a reflection of acute cadmium exposure. Levels above 5 mcg/dl suggest excessive exposure. Urine cadmium levels appear to be a better measurement of chronic exposure. However, none of these tests are available rapidly and do not help in acute management.
- Urinary metallothionein may be a better indicator of body burden, but is not widely available in Australia.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
131126

Asymptomatic patients:

- Asymptomatic patients with a suspected history of significant exposure should be observed for at least 12 hours as it has delayed effects.
- For minor exposures in asymptomatic patients, see the section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures / ingestions should be admitted and observed for at least 24 to 48 hours.
- Use benzodiazepines to control seizures
- Ensure adequate analgesia is prescribed.

Inhalation

- Give humidified oxygen for hypoxia.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Ingestion

- Do not induce vomiting, although this may occur naturally.
- Appropriate fluids limit cadmium toxicity in kidneys and liver, but require close monitoring.
- Activated charcoal has no proven benefit in cadmium poisoning but may be considered in selected cases following expert clinical toxicology advice.
- Although not demonstrably efficacious, chelation therapy with calcium disodium Edetate may be of benefit immediately following acute exposure. Dimercaprol is not recommended because of injury from mobilised cadmium.

Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Carbon disulphide

Alternative names	Carbon disulfide Carbon bisulfide Carbon sulfide Dithiocarbonic anhydride Alcohol of Sulfur
Properties of the agent	<ul style="list-style-type: none">• Highly flammable, colourless to faintly yellow liquid with a boiling point of 46 degrees C and a vapour that is heavier than air at room temperature.• In the pure form, it has a sweetish aromatic odour detectable at 0.1 –• 0.2 ppm by most people. The odour threshold is 100 times lower than the OSHA permissible exposure limit of 20 ppm.• Liquid carbon disulphide degrades some plastics and rubber.• Many metals ignite in unheated carbon disulphide.
Routes of exposure	<ul style="list-style-type: none">• Oral exposure is unlikely, but may occur as carbon disulphide is a liquid at room temperature.• Inhalation is the major route of exposure.• Carbon disulphide is corrosive, causing severe eye and skin burns.• Eye exposure to 10-20 ppm causes irritation of mucous membranes.• Dermal absorption may be a significant route of exposure.

Human toxicity

Symptoms are based on route of exposure:

General:

- Carbon disulfide is a known severe health hazard that mainly affects the central nervous system.

Inhalation:

- Significant acute exposure produces central nervous system excitement followed by depression, with stupor, restlessness and unconsciousness.
 - Mild exposure results in dizziness and headache;
 - Moderate exposure: nervousness, fatigue and weight loss;
 - Severe exposure: agitation, delirium, seizures, coma, tremor, spasticity, dysrhythmias, cardiovascular collapse, dyspnoea and respiratory failure.
- Other symptoms include general pains, insomnia, lethargy, nausea, vomiting, indigestion, anorexia, abdominal pain, and irritation of mucous membranes.
- Extreme intoxication may result in a Parkinsonian-like syndrome, with speech disturbances, muscle spasticity, tremor, memory loss, mental depression and psychosis.
- Repeated exposure produces nervousness, irritability, indigestion, bizarre dreams, insomnia, fatigue, anorexia and headache.

Ingestion:

- Ingestion of as little as 15 mL can be fatal. Symptoms include dyspnoea, hypothermia, pallor, cyanosis, mydriasis, spasmodic tremors, convulsions, collapse, coma, Cheyne-Stokes respiration, respiratory paralysis and death. In humans, the lowest reported lethal oral dose of carbon disulfide is 14 mg/kg.

Dermal:

- Dermal exposure results in burning pain and erythema, with vesicle formation, blistering, exfoliation, and possibly second- and third-degree burns.

Ocular:

- Irritation, lacrimation.

Chronic exposure:

- Chronic exposure can produce permanent central and peripheral nervous system damage. Other consequences include atherosclerotic tendencies, ECG abnormalities, gastrointestinal disturbances, fatty degeneration of the liver (with jaundice), renal damage, fatigue, memory loss, insomnia, melancholia, mania, hallucinations, increased suicide rate, sexual dysfunction, cranial nerve damage, hearing loss, visual disturbances, altered pupillary reaction to light, retinal microaneurysms, optical, otic and peripheral neuropathies, loss of reflexes, tremors, and blood dyscrasias.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required

For significant exposures:

- Monitor EUC and LFTs.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Perform continuous ECG monitoring. Monitor blood pressure.
- Carbon disulphide blood concentrations and measurement of urinary metabolites can be used to evaluate exposure to carbon disulphide. However, this test is not useful in acute management and is not readily available in Australian hospitals.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- As carbon disulphide is a potent irritant, asymptomatic patients only need a short period of observation.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Decontamination may be required to relieve skin and eye irritation, or following exposure to liquid carbon disulphide.
- Administer a benzodiazepine IV for seizures.
- Observe for 6 to 8 hours. If respiratory symptoms resolve in this time, further complications are unlikely.
- Discharged patients should be reviewed at 24 hours if they had initial skin or eye symptoms.
- Inhalation

Give humidified oxygen for hypoxia.

- Maintain a patent airway and support respiration as required with invasive or non-invasive ventilation.

Ingestion

- Administer activated charcoal as a slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old.
- Consider gastric lavage after ingestion of a potentially life-threatening amount of poison if it can be performed soon after ingestion (generally within 1 hour).

Dermal

- Flush affected areas thoroughly.
- Manage burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange urgent ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Carbon monoxide

Alternative names	Carbonic oxide Carbon oxide CO Exhaust gas Flue gas
Properties of the agent	<ul style="list-style-type: none">It is an extremely poisonous, odourless, tasteless and colourless gas, which is produced when there is incomplete combustion of carbon containing fuels (e.g. coal, petroleum, peat, natural gas, etc.).
Routes of exposure	<ul style="list-style-type: none">Inhalation – most common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">Carbon monoxide combines with haemoglobin to form carboxyhaemoglobin, which is then unable to transport oxygen, resulting in tissue hypoxia; and cellular poisoning by combining with other haem compounds such as myoglobin and cytochrome oxidase.Clinical symptoms of mild poisoning are non-specific and may mimic those of a non-specific viral illness, with vomiting, headache, malaise, weakness, fatigue and shortness of breath.The main manifestations of carbon monoxide poisoning develop in the organ systems most dependent on oxygen use: the CNS and myocardium. The symptoms are listed below:<ul style="list-style-type: none">Mild toxicity – throbbing temporal or frontal headache, fatigue, dyspnoea on exertion, light-headedness and dizziness.Moderate toxicity – severe headache, weakness, dizziness, nausea, vomiting, tachycardia, tachypnoea, flushing, perspiration, decreased vigilance, diminished manual dexterity, impaired sensorimotor task performance, prolonged reaction time, difficulty thinking, impaired judgement, blurred or darkened vision, ataxia, loss of muscular control, tinnitus and drowsiness.Severe toxicity – may produce syncope, seizures, confusion, disorientation, involuntary evacuations, ventricular dysrhythmias, cardiorespiratory depression, respiratory failure, coma and death.Delayed effects, attributable to hypoxia, usually result in neuropsychiatric effects.Contact with liquid carbon monoxide or its container can cause local frostbite.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Determine carboxyhaemoglobin (COHb) level when the patient is first seen and repeat every 2 to 4 hours until patient is asymptomatic or level is within the normal range.
Note: COHb levels correlate poorly with signs and symptoms of toxicity. Interpretation may be confounded by delays in obtaining blood samples and therapeutic interventions (oxygen administration). The so called classic 'cherry-red skin' of carbon monoxide poisoning is rare.
- Monitor ECG, EUC, CK, ABGs if symptomatic or if the COHb level is greater than 20%. Pulse oximetry is not a reliable estimate of oxyhaemoglobin saturation.
- Obtain CXR.
- CT or Magnetic Resonance Imaging (MRI) scan should be considered if neurologic symptoms persist.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours.
- High flow normobaric oxygen, via a non-rebreathing reservoir face- mask or endotracheal tube, should be instituted as soon as possible in a patient suspected to have CO poisoning.

Inhalation

- Symptomatic and supportive treatment protocols apply.
- Treatment of respiratory failure may include invasive or non-invasive ventilation
- Intravenous fluids should be limited to 2/3 to 3/4 of normal maintenance. Osmotic diuretics (e.g. mannitol), or other methods to reduce intracranial pressure may be used but are unlikely to affect outcome.
- Consider hyperbaric oxygen therapy for severely poisoned patients (coma, seizures, other neurologic abnormalities and myocardial ischemia) and in pregnant patients. Institute hyperbaric therapy as quickly as possible, ideally within 6 to 8 hours.

Dermal

- Handle frostbitten areas with caution. Place frostbitten skin in tepid water and allow circulation to re-establish spontaneously.

Carbonyl sulphide

Alternative names	<p>COS Carbonyl sulfide Carbon monoxide monosulfide Carbon oxide sulfide Carbon oxysulfide Oxycarbon sulfide</p>
Properties of the agent	<ul style="list-style-type: none">• Pure carbonyl sulphide is a colourless odourless gas that is heavier than air.• Commercial carbonyl sulphide does however have a typical sulphur odour.• Corrosive to common metals when moisture is present.• Reacts vigorously with oxidants.• Flammable.• Potential use as a grain fumigant in the future in Australia.• Used in industrial production of thiocarbamate herbicides and for the preparation of aliphatic polyureas in other countries.• It also inhibits cytochrome oxidase to impair cellular respiration via metabolism to hydrogen sulphide and its degradation products (see Hydrogen Sulphide sheet).
Routes of exposure	<ul style="list-style-type: none">• Inhalation is the major route of exposure.• Eye exposure causes irritation to mucous membranes and cornea.• Dermal exposure may cause irritation but direct absorption unlikely.• Ingestion is unlikely, as carbonyl sulphide is a gas at room temperature.

Human toxicity

General

- Harmful by inhalation at levels from around 100 ppm upwards.
- High concentrations may be fatal. As with hydrogen sulphide, exposure to the gas induces olfactory fatigue so that one may underestimate the level at which the gas is present – hence carbonyl sulphide has poor warning properties.
- It may cause serious effects or lethality at concentrations causing no signs or symptoms.

Direct

- Irritant to pulmonary mucosa, eyes, mucous membranes; and neurotoxic (including brain stem and central respiratory paralysis).

Indirect

- Cellular inhibition of cytochrome via metabolism to hydrogen sulphide.

Symptoms are based on route of exposure:

Inhalation:

- Tachypnoea, palpitations, tachycardia, arrhythmia, sweating, weakness, & muscle cramps [see hydrogen sulphide].
- Sudden collapse & unconsciousness (with or without a warning cry) to high levels.
- Death from prompt respiratory paralysis, usually with a terminal asphyxial convulsion.
- GI effects include profuse salivation, nausea, vomiting and diarrhoea.
- Central nervous effects include giddiness, headache, vertigo, amnesia, confusion, and unconsciousness.
- After sublethal exposures recovery is slow; patient may have cardiac dilatation, slow pulse, peripheral neuropathy, albuminuria, amnesia or psychological disturbance. Recovery is eventually complete in most nonfatal cases.

Ingestion:

- Unlikely.

Dermal:

- Skin: direct contact (as a solution) may produce erythema & pain.
- Exposure to compressed gas may result in frostbite injury.

Ocular:

- Eyes: painful conjunctivitis, photophobia, lacrimation, and corneal opacity.
- Exposure to compressed gas may result in frostbite injury.

Laboratory/radiographic testing

For minimal or mild exposures:

- Obtain baseline PEFR, pulse oximetry and ECG.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Perform continuous ECG monitoring. Monitor blood pressure.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Severely affected patients should have long-term follow-up lung function tests.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- Asymptomatic patients and those who experience only minor sensations of burning of the nose, throat, eyes and respiratory tract can be discharged after observation. In most cases, these patients will be free of symptoms in an hour or less.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- All symptomatic patients should be admitted and observed for at least 24 hours.
- Cardiac monitoring for 24 hours is recommended.

Inhalation

- Administer humidified oxygen.
- Relieve bronchospasm with bronchodilators.
- Treatment of respiratory failure may include invasive or non-invasive ventilation.
- CXR and PFTs should be repeated 2-3 months post discharge.

Dermal

- Flush affected areas thoroughly.
- Manage corrosive burns as for thermal burns.
- Handle frostbitten areas with caution. Place frostbitten skin in tepid water and allow circulation to re-establish spontaneously.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Antidote

- Consult a clinical toxicologist regarding the possible use of Sodium nitrite.

Chlorine

Alternative names	Nil known
Properties of the agent	<ul style="list-style-type: none">• It is a yellow-green, heavier-than-air gas with a sharp acrid smell at room temperature and pressure.• Chlorine gas reacts with moist tissue to form hydrochloric acid and is a potent irritant to the eyes, skin and upper respiratory tract.
Routes of exposure	<ul style="list-style-type: none">• Inhalation – most common• Eye
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Chlorine causes acid burns to exposed surfaces.• Chlorine is heavier than air and may cause asphyxiation in poorly ventilated, enclosed and low-lying areas. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Tachycardia and tachypnoea are common. Severe exposure may cause cardiovascular collapse and respiratory arrest.• Airway irritation, coughing, choking, laryngeal oedema, bronchospasm and hypoxia may occur in moderate concentrations. In high concentrations, syncope and almost immediate death may occur.• Acute lung injury is common after severe exposure, resulting in non- cardiogenic pulmonary oedema, chemical pneumonitis and haemoptysis after a latent period of up to 36 hours. <p>Ingestion</p> <ul style="list-style-type: none">• Ingestion is unlikely to occur because chlorine is a gas at room temperature. Solutions that are able to generate chlorine (e.g., sodium hypochlorite solutions) may cause corrosive injury if ingested. <p>Dermal</p> <ul style="list-style-type: none">• Direct contact with liquid chlorine or concentrated vapour causes severe chemical burns, leading to cell death and ulceration.• Frostbite can occur following exposure to compressed liquid chlorine. <p>Ocular</p> <ul style="list-style-type: none">• Severe corneal and conjunctival irritation, lacrimation, ulceration and scarring can occur.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Monitor ECG.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
Note: A normal CXR and ABG within the first few hours do not rule out the eventual development of potentially life-threatening pneumonitis and/or non-cardiogenic pulmonary oedema.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- Asymptomatic patients and those who experience only minor sensations of burning of the nose, throat, eyes and respiratory tract can be discharged. In most cases, these patients will be free of symptoms in an hour or less.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours.
- Consider admitting all patients with pre-existing respiratory disease.
- All patients with moderate or severe clinical effects should have post-discharge lung function tests.

Inhalation

- Administer humidified oxygen.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Dermal

- Flush affected areas thoroughly.
- Manage corrosive burns as for thermal burns.
- Handle frostbitten areas with caution. Place frostbitten skin in tepid water and allow circulation to re-establish spontaneously.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Chloroacetophenone

Alternative names	CN Mace Tear Gas 2-Chloroacetophenone Alpha-chloroacetophenone Mono-chloroacetone Phenyl-chloromethyl phenone
Properties of the agent	<ul style="list-style-type: none">• It is a clear to yellow-brown solid with an apple-blossom odour, dispersed as an aerosol.• It is commonly used as a riot control agent.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation – most common• Eye• Dermal – common
Human toxicity	<p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• High concentrations may cause upper respiratory tract irritation with nasal discomfort, rhinorrhoea and sneezing, skin vesicle formation and visual impairment.• Delayed onset non-cardiogenic pulmonary oedema is seen with prolonged exposure to high concentrations of chloroacetophenone in confined spaces. <p>Ingestion</p> <ul style="list-style-type: none">• Ingestion of contaminated food and water causes nausea, vomiting and diarrhoea. <p>Dermal</p> <ul style="list-style-type: none">• Chloroacetophenone is a skin sensitiser and splash contact may cause papulovesicular dermatitis within 72 hours of exposure.• Erythema and superficial skin burns occur with prolonged exposure. <p>Ocular</p> <ul style="list-style-type: none">• Ocular signs and symptoms predominate and peak within a few minutes. These can include lacrimation, a burning sensation, eyelid swelling, sharp pain, blepharospasm, photophobia and temporary blindness. Symptoms usually resolve within 15 to 30 minutes. Some individuals may experience ocular symptoms for up to 24 hours.• Splash contact may cause burns and corneal opacity.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- Symptoms usually settle within 15 to 30 minutes following removal from exposure. See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with persistent respiratory symptoms should be admitted and observed for at least 24 to 48 hours.

Inhalation

- Humidified oxygen and bronchodilators may provide symptomatic relief.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Dermal

- Flush affected areas thoroughly.
- Manage burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Chloropicrin

Alternative names	Trichloronitromethane Nitrochloroform Nitrotrichloromethane PS G 25 'War gas' or 'Vomiting gas' Acquinite Dolochlor
Properties of the agent	<ul style="list-style-type: none">• It is a slightly oily, colourless liquid with pungent odour.• It is mostly non-flammable but may react violently with various substances with a risk of fire and explosion. It gives off hydrochloric acid and nitrous vapours on decomposition.• In Australia, it is used as a grain and soil fumigant.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation – most common• Eye• Dermal – common

Human toxicity

Symptoms are based on route of exposure:

General

- Headache, nausea and vomiting are common following exposure.
- Severe exposure may result in coma, hepatic and cardiac necrosis, and renal impairment.
- Anaemia and cardiac arrhythmias have been reported.

Inhalation

- Irritation of mucous membranes may cause a burning sensation in the mouth, rhinorrhoea, sneezing, bronchospasm, cough, upper airway oedema and increased bronchial secretions.
- Inhalation or aspiration may result in dyspnoea and non-cardiogenic pulmonary oedema, which is the most frequent cause of early death. Late deaths may result from secondary infections, bronchopneumonia and / or bronchiolitis obliterans.
- Irregular respirations and periods of apnoea may occur. Chloropicrin is a sensitiser and may induce recurrent asthma.

Ingestion

- Orthostatic hypotension may be noted.
- Nausea, vomiting, epigastric pain and gastric burns may occur.

Dermal

- Skin irritation, with burning sensation and erythema, may be noted.
- Blistering and dermatitis may occur. Full thickness burns can occur with prolonged exposure.

Ocular

- Lacrimation, blepharospasm and pain are common.
- Severe exposure can result in corneal perforation.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, FBC, cardiac enzymes.
- Monitor vital signs and replace fluids.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs. In symptomatic patients, CXR findings may lag behind clinical findings by several hours.
- The odour is a distinctive warning property of this liquid compound so history from the patient may help.
- Plasma chloropicrin levels are not clinically useful and not readily available in Australian hospitals.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information 131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours.

Inhalation

- Give humidified oxygen for hypoxia and bronchodilators for wheeze.
- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Consider PEEP or CPAP for non-cardiogenic pulmonary oedema.

Ingestion

- Do not induce vomiting.
- Administer 120 to 240 mL of water if the patient is alert, conscious and cooperative. Administer intravenous fluids to unconscious patients or patients who are not alert and cooperative.

Dermal

- Flush affected areas thoroughly.
- Manage burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Copper sulphate

Alternative names	Blue stone Copper basic sulphate Copper monosulphate Triangle
Properties of the agent	<ul style="list-style-type: none">• Copper is an essential trace element.• It is used as an inorganic fungicide, algacide, herbicide and molluscide.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Copper sulphate, a soluble copper salt, is a strong irritant of the skin and mucous membranes. <p>Symptoms are based on route of exposure:</p> <p>Ingestion</p> <ul style="list-style-type: none">• The prompt emetic effect of ingested copper sulphate may limit its oral toxicity. Vomitus is characteristically greenish-blue. Haemorrhagic gastroenteritis associated with mucosal erosions, a metallic taste, burning epigastric sensation and diarrhoea may occur.• Hepatomegaly, liver tenderness and jaundice may occur on the second or third day post-ingestion.• In cases of massive ingestion, gastrointestinal irritation, haemolytic anaemia, methaemoglobinaemia (rare), kidney and liver failure, shock and in some cases, death may result, <p>Dermal</p> <ul style="list-style-type: none">• Skin exposure may result in severe irritation. <p>Ocular</p> <ul style="list-style-type: none">• Copper sulphate is corrosive to the cornea.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC, MetHb.• Consider obtaining whole blood copper levels in symptomatic patients, but these are rarely useful during acute management.• If respiratory tract irritation or respiratory depression is evident, monitor ABGs, CXR and PFTs.• Note that pulse oximetry is not reliable in patients with methaemoglobinaemia, as methaemoglobin and methylene blue interfere with the readings.• Consider gastroenterology review.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Ensure adequate analgesia is prescribed.

Ingestion

- Do not induce vomiting, although this may occur spontaneously.
- Make the patient nil by mouth.
- Administer intravenous fluids as necessary.
- Arrange a gastroenterology review for all suspected gastrointestinal burns.

Dermal

- Copious flushing may relieve skin irritation.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Antidote

- Chelating agents, such as **D-penicillamine** and **dimercaprol**, may be indicated in selected cases following discussion with a clinical toxicologist.

Antidote for methaemoglobinaemia

- **Methylene blue** is administered for symptomatic methaemoglobinaemia.
- Methylene Blue should be administered if the methaemoglobinemia exceeds 20%. 1-2mL/kg methylene blue intravenously over 5 minutes. This can be repeated after 60 minutes.
- Measure methaemoglobin levels hourly until a consistent fall is documented.
- If methylene blue fails to control methaemoglobinaemia, consider exchange transfusion or hyperbaric oxygen therapy.

Note: For antidote indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Copper oxychloride

Alternative names	Basic Copper chloride Copper chloride oxide, hydrate (9ci) Colloidox Blue Copper Copper chloride oxide
Properties of the agent	<ul style="list-style-type: none">• Copper is an essential trace element.• Copper oxychloride is used as an effective fungicide in Australia.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Copper oxychloride is a strong irritant of the skin and mucous membranes. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Metal fume fever, wheezing and rales have been reported in workers exposed to fine copper dust or heated copper oxychloride. <p>Ingestion</p> <ul style="list-style-type: none">• Nausea, vomiting, diarrhoea, a metallic taste and burning epigastric sensation may be noted.• Gastrointestinal bleeding may occur.• Hepatomegaly, liver tenderness, increased levels of transaminases and jaundice may occur on the second or third day after ingestion of copper salts.• Anuria, haemolytic anaemia and renal failure have been described. <p>Dermal</p> <ul style="list-style-type: none">• Severe irritation, itching, erythema, dermatitis and eczema may occur.• Dermal exposure can also result in systemic toxicity. <p>Ocular</p> <ul style="list-style-type: none">• Irritation, conjunctivitis, palpebral oedema, ulceration and corneal burns may be noted.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• Consider obtaining whole blood copper levels in symptomatic patients, but these are rarely useful during acute management.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Ensure adequate analgesia is prescribed.

Inhalation

- Administer humidified oxygen.
- Relieve bronchospasm with bronchodilators.

Ingestion

- Do not induce vomiting. Emesis is rapid and spontaneous in most patients.
- Make the patient nil by mouth.
- Consider gastroenterology review.

Antidote

- Chelating agents, such as penicillamine, may be indicated in selected cases following discussion with a clinical toxicologist.

Cyanide

Alternative names	Cyanide salts (K, Mercury (Hg), Na, Ca, Zn) Cyanogen bromide Cyanogen iodide Cyanogen chloride Hydrogen Cyanide Cyanogens
Properties of the agent	<ul style="list-style-type: none">• Cyanide is widely used in metal processing, chemical manufacture, gold mining and in pesticides or rodenticides.• Heating of cyanide salts, or a reaction with acid, may result in release of hydrogen cyanide gas.• Cyanide is a by-product of combustion of synthetic materials.
Routes of exposure	<ul style="list-style-type: none">• Oral – Ingestion can be rapidly fatal, depending upon concentration, presence of food in stomach and type of cyanide salt.• Inhalation (as Hydrogen Cyanide).• Eye – there is a theoretical risk of systemic toxicity by this route.• Dermal.

Human toxicity

General:

- Cyanide has a very rapid onset of toxicity. It interferes with cellular respiration and aerobic metabolism by inhibiting cytochrome oxidase.
- It is readily absorbed through the lungs, with symptoms within seconds to minutes, depending on the concentration and duration. Skin or ocular absorption may contribute to systemic poisoning.
- Onset of symptoms may be immediate or delayed for 30 to 60 minutes after gastrointestinal exposure.
- Death is usually from cardiorespiratory failure and may be within few minutes of exposure (especially after inhalation).

Symptoms are based on route of exposure:

General

- Mild effects include: headache, anxiety, nausea and vomiting.
- Moderate effects include: headache, dizziness, vomiting, confusion, ataxia, hyperventilation, dyspnoea, hypotension, bradycardia and short-lived unconsciousness. Transient loss of vision and hearing has been reported with sub-lethal exposure.
- Severe effects include: coma, convulsions, deteriorating cardiorespiratory function.
- Associated lactic acidosis.
- Long-term effects include: Parkinsonian changes, memory impairment, and extrapyramidal effects.

Inhalation

- Dyspnoea, tachypnoea, involuntary gasping, cough and chest tightness may develop early.
- Sudden loss of consciousness, convulsions and apnoea may develop within few minutes.
- Cyanosis is rarely reported.

Ingestion

- Cyanide salts may react with gastric acid to produce hydrogen cyanide gas. This may be exhaled by the patient or evolve from vomitus.

Dermal/ocular

- May cause skin and eye irritation and dilated pupils.

Laboratory/radiographic testing

For minimal or mild exposures:

- No testing is required for asymptomatic patients.

For significant exposures:

- Monitor EUC, Serum Lactate.
- Obtain arterial / venous blood gas as metabolic acidosis, often severe, combined with reduced arterial-venous oxygen saturation difference (<10 mm Hg) suggests diagnosis in a suspected patient.
- Serum cyanide levels are not commonly available and are not helpful in acute management. Collect blood in fluoride heparinised tube to confirm diagnosis later.
- If respiratory depression is evident monitor ABGs, and CXR.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).
- Any patient who is fully conscious and breathing normally more than 5 minutes after presumed inhalational exposure to cyanide agents has ceased, will recover spontaneously and requires 100% oxygen and reassurance.
- Asymptomatic patients with suspected ingestion of cyanide should be observed for at least 6 hours, in case of delayed onset of effects.

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed until they have normal vital signs, acid-base status and oxygenation.
- Speed is critical. Evaluate and support airway, breathing and circulation. Administer 100% oxygen therapy via a non-rebreathing facemask. Early intubation in a severe exposure is recommended.
- Patients deteriorating or with more severe symptoms (e.g. cardiovascular instability or reduced level of consciousness), should receive antidote treatment.
- Hypotension and seizure are common following severe intoxications and should be managed with standard protocols.

Antidotes

- **Hydroxocobalamin** is the antidote of choice 5 to 10 g IV by slow infusion
- **Sodium thiosulphate** in addition in severe cases 12.5 g over 10 – 20 minutes.

Note: For indications, contraindications, dosing regimens and clinical end-points, seek expert clinical toxicology advice.

If these are not available, sodium nitrite and dicobalt edetate are further possible antidotes: please discuss with a clinical toxicologist.

Dimethyl sulphate

Alternative names	DMS Methyl sulphate
Properties of the agent	<ul style="list-style-type: none">• Dimethyl sulphate is a colourless, oily liquid with a faint, onion-like odour.• It is an organic sulphur compound, an alkyl ester of sulphuric acid.• It is used in the manufacture of pharmaceuticals, dyes, perfumes and pesticides.• It is considered carcinogenic.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• On contact with mucosa, DMS hydrolyses slowly to sulphuric acid, methanol, and methyl hydrogen sulphate. The methanol is neurotoxic. The sulphuric acid and methyl hydrogen sulphate are irritant, corrosive and anaesthetic to the mucosa.• There is a latent period of between 20 minutes and 12 hours. Signs of toxicity are generally delayed for 3 to 4 hours due to the anaesthetic properties.• CNS effects include early signs of headaches and nervousness. Seizures, paralysis, delirium and coma are delayed signs. Irreversible loss of vision has been reported.• Fever, tachycardia, and myocardial damage with ST segment changes, and T wave inversions occur.• Delayed renal, hepatic and cardiac failure may occur.• Death from severe poisoning is due to respiratory failure. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Severe irritation of the respiratory tract, with oedema of the major airways, dyspnoea and non-cardiogenic pulmonary oedema may occur. These symptoms may be delayed for up to 10 hours. Productive cough may persist for months. <p>Ingestion</p> <ul style="list-style-type: none">• Mucosal desquamation and necrosis of the nose and throat with laryngeal oedema may occur. Nausea, vomiting, haematemesis and dysphagia have been reported. <p>Dermal</p> <ul style="list-style-type: none">• It has a strong corrosive and vesicant action. Onset of dermal effects is typically delayed for several hours. <p>Ocular</p> <ul style="list-style-type: none">• Conjunctival and corneal irritations corneal oedema, lacrimation, blurred vision, eye pain and photophobia have been reported.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC and Urinalysis (for albumin and haematuria).
- Monitor ECG.
- Plasma osmolality and methanol levels should be monitored if neurotoxic symptoms are present.
- Monitor vital signs and fluid balance.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain an upright CXR if GIT perforation is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- Asymptomatic patients with a history of exposure to DMS must be observed for at least 24 hours because of delayed onset of potentially catastrophic symptoms.

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 hours.
- Dimethyl sulphate is metabolised to a number of metabolites including methanol, the role of methanol in toxicity is unknown.
- All patients with moderate or severe clinical effects should have post-discharge periodic lung function tests, ECG, and ophthalmological review.

Inhalation

- Keep patients at absolute bed rest whilst under observation.
- Administer humidified oxygen.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Make the patient nil by mouth.
- Administer intravenous fluids.
- Consider gastroenterology review and endoscopy in patients suspected of having corrosive burns of the GIT.

Dermal

- Flush affected areas thoroughly.
- Manage corrosive burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Dimethyl sulphoxide

Alternative names	DMSO Methyl sulphoxide
Properties of the agent	<ul style="list-style-type: none">• It is a clear (water-white), very hygroscopic liquid, with slightly sulphurous odour.• It also has a slightly bitter taste with a sweet after-taste.• It is excreted through the lungs and skin; and is responsible for the characteristic odour (garlic-like) from patients.• It has shown very few toxic symptoms in humans.• DMSO carries other chemicals through the skin and toxic effects may be due to these agents.
Routes of exposure	<ul style="list-style-type: none">• Oral – common• Inhalation – most common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• It is a skin, eye and respiratory tract irritant. It may cause skin irritation and reddening if spilled on clothing and allowed to remain.• It readily penetrates the skin and may carry other dissolved chemicals into the body. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• The unusual garlic-like breath is a common finding. Dyspnoea and increased symptoms of asthma may be noted. <p>Ingestion</p> <ul style="list-style-type: none">• Anorexia, nausea, vomiting, and diarrhoea or constipation may occur.• Elevated liver enzymes may be noted. Two doubtful cases of liver toxicity have been reported. <p>Dermal</p> <ul style="list-style-type: none">• It releases histamine from mast cells and rarely can cause an anaphylactic reaction.• Stinging or burning of the skin, rashes and vesicles may occur. Systemic contact dermatitis has been reported from intravesical administration. <p>Ocular</p> <ul style="list-style-type: none">• Transient photophobia and colour vision disturbances have been reported.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 hours.
- Vomiting should not be induced.
- Early use (within 1 hour of ingestion) of activated charcoal can be considered following expert clinical toxicology advice.

Inhalation

- Give humidified oxygen for hypoxia and bronchodilators for wheeze.

Ethylene glycol

Alternative names	Ethylene glycol 1,2-Ethanediol Glycol alcohol EG Antifreeze
Properties of the agent	<ul style="list-style-type: none">• Ethylene glycol, commonly sold as automobile radiator antifreeze, is a colourless, clear, sweet- or bittersweet-tasting, viscous liquid.• It is considerably hygroscopic and is able to absorb twice its weight of water at 100% relative humidity.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation – common• Eye• Dermal- negligible
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Most exposures occur from the ingestion of antifreeze. It is rapidly absorbed from the GIT. Toxicity can be divided into three stages: <p>Stage 1: Neurological (0.5 to 12 hours post-ingestion)</p> <ul style="list-style-type: none">• A transient inebriation and euphoria, similar to ethanol intoxication, occurs within the first several hours. Nausea and vomiting result from direct gastric irritation. As antifreeze is metabolised, metabolic acidosis and CNS depression may develop. Symptoms due to toxic metabolites occur 4 to 12 hours post-ingestion. Serious intoxications may progress to coma associated with hypotonia, hyporeflexia and less commonly, seizures and meningismus. Occasionally papilloedema may be present. CNS depression in paediatric patients who ingest antifreeze may be multifactorial and involve alcohol-induced hypoglycaemia. <p>Stage 2: Cardiopulmonary (12 to 24 hours post-ingestion)</p> <ul style="list-style-type: none">• Tachycardia and hypertension may be noted. Severe metabolic acidosis with compensatory hyperventilation can develop with multiple organ failure in significant poisonings. Hypoxia, congestive heart failure and ARDS have been reported. Most deaths are reported during stage 2. <p>Stage 3: Renal (24 to 72 hours post-ingestion)</p> <ul style="list-style-type: none">• Oliguria, acute tubular necrosis, renal failure and occasionally bone marrow suppression occur during stage 3. Renal failure may appear early in severe poisonings and progress to anuria. Calcium oxalate crystals may be detected in the urine of some patients but the absence of calcium oxalate crystals does not rule out the diagnosis. Haematuria and proteinuria are common. In surviving cases, renal function usually returns to normal, but in some cases permanent renal damage may occur. Serious hepatic injury is uncommon.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.
- All patients with suspected ingestion should have blood and urine tests.

For significant exposures:

- Monitor EUC, Glucose, LFTs, CMP, FBC, serum osmolality, CK.
- Monitor vital signs and replace fluids.
- Monitor ABGs. Calculate the osmolar gap and anion gap. High osmolar gap is suggestive of EG poisoning but can also be raised in methanol poisoning and diabetic ketoacidosis.
Note: Within the first few hours post-ingestion, the absence of an increased anion gap metabolic acidosis does not rule out EG poisoning.
- Obtain an ECG and look for QTc prolongation (suggestive of hypocalcaemia).
- Urine sample can be sent for urinalysis to look for calcium oxalate crystals but their absence does not preclude EG poisoning.
- EG levels may not be available in all hospitals and may not be helpful in acute management. Toxicity cannot be ruled out based on a non-toxic EG level.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- All patients with suspected significant EG ingestion with mild or no symptoms should have appropriate blood and urine tests. If the tests are normal, they can be discharged.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- All patients should be evaluated and treated without delay.
- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours as it has delayed effects.
- Definitive care for any significantly poisoned patient is treatment with antidote (an alcohol dehydrogenase inhibitor, usually ethanol or fomepizole) AND haemodialysis.
- Blood sugar should be monitored frequently as hypoglycaemia may occur, particularly in children.

Antidote

- **Intravenous ethanol** is a recognised antidote for EG poisoning. Ethanol (ETOH) inhibits the metabolism of EG to its toxic metabolites. Ethanol can be administered either orally or intravenously.
Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Ethyl mercury chloride

Alternative names	EMC Chloroethylmercury Ethylmercuric chloride Granosan
Properties of the agent	<ul style="list-style-type: none">• Ethyl mercury chloride is an organic mercury compound, which is found as silver crystals or a white solid.• It is an extremely heavy, silver coloured liquid.• Organic mercury compounds are often used as antiseptics/antibacterial, fungicides, herbicides, seed disinfectants and as preservatives in pharmaceuticals.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation – common• Eye• Dermal – common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Toxicity following acute exposures may be delayed for weeks to months, with predominant GIT and CNS effects. Of the three forms of mercury (elemental, inorganic and organic), organic mercury is considered to be the most neurotoxic (cerebellar damage).• Organic mercury can be absorbed through the skin to produce systemic effects. Irritation or burns can result from exposure to some compounds. Sensitisation has been reported.• When mercury poisoning is suspected in critically ill patients, chelation therapy should be started regardless of the form of mercury causing toxicity.• Early symptoms are a metallic taste in the mouth, numbness and tingling of digits and face, tremor, headache, fatigue, emotional lability and cognitive dysfunction.• Severe symptoms include incoordination, hearing loss, constriction of visual fields, spasticity of muscles and deterioration of mental capacity. Death is not uncommon and significant neurological damage is common in survivors.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
 - Monitor vital signs and replace fluids.
 - If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
 - Obtain whole blood mercury levels and 24-hour urine collection for mercury (Normal range for whole blood mercury levels rarely exceed 1.5 mcg/dL and normal urine excretion level rarely exceeds 15 mcg/L in unexposed individuals).
- Note:** Levels do not help with acute management, but can be used to confirm diagnosis later on.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert advice.

Antidote

- Chelation therapy (**D-Penicillamine** and/or **DMSA/Succimer**) is indicated in selected symptomatic cases.
 - Haemodialysis should be considered early in severe cases, with diminishing urine output following chelation.
- Note:** For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Fluoroethyl alcohol

Alternative names	2-Fluoroethanol Fluoroethanol 2-Fluoro-ethanol Ethylene fluorohydrin
Properties of the agent	<ul style="list-style-type: none">• It is a liquid fluoro alcohol compound, which is miscible in water. Little specific data is available about the toxicity of Fluoroethyl alcohol; its toxicity is expected to be similar to that of fluoroacetate, as it is oxidised to fluoroacetate by tissue alcohol dehydrogenase.• In Australia it is widely used as a pesticide.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation – common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• It is rapidly absorbed and may cause systemic toxicity after any route of exposure.• Clinical effects usually develop within 30 minutes to 2.5 hours of exposure, but may be delayed for as long as 20 hours. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Respiratory depression and pulmonary oedema have been reported. <p>Ingestion</p> <ul style="list-style-type: none">• Prolonged QTc intervals, ventricular tachycardia or fibrillation and asystole may occur.• Hyperactive behaviour, auditory hallucinations, cerebellar dysfunction, loss of speech, paraesthesia, seizures, coma, carpopedal spasm and neurologic impairment may be noted.• Nausea, vomiting, excessive salivation and diarrhoea may be noted. Mild hepatic dysfunction has been reported.• Metabolic acidosis, hyperglycaemia, hypocalcaemia, hyperuricaemia and carpopedal spasm may be noted.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• Obtain an ECG and institute continuous cardiac monitoring. Measure QTc.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Fluoroacetate levels are not clinically useful.• Samples for analysis should include suspected baits, vomitus, stomach contents, liver and kidney.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 hours as it has delayed effects.
- Cardiac monitoring is required for at least 24 hours.
- Correct electrolyte disturbances.
- Control seizures with benzodiazepines.
- Treat hypotension with a fluid bolus. Consider the use of inotropes.

Inhalation

- Administer humidified oxygen for hypoxia.
- Monitor for respiratory depression. Intubate and ventilate as indicated.

Ingestion

- Do not induce vomiting.
- (It's an alcohol – AC won't work).

Hydrochloric acid

Alternative names

Muriatic acid
Bleaching agent
Hydrogen chloride

Properties of the agent

- It is a colourless to light yellow aqueous solution that fumes in air.
- It exists as hydrogen chloride in its gaseous form, which is a colourless to light yellow with an irritating, pungent odour.
- It is used as a chemical intermediate.

Routes of exposure

- Oral
- Inhalation – most common
- Eye
- Dermal – common

Human toxicity

General:

- It is a severe corrosive irritant of the skin, eye and mucous membranes.
- Death after exposure to hydrochloric acid may result from circulatory shock, asphyxia (with laryngeal and glottal oedema), or stomach perforation (with peritonitis, gastric haemorrhage and infection).

Symptoms are based on route of exposure:

Inhalation

- Inhalation of hydrochloric acid fumes produces nose, throat and laryngeal burning and irritation, pain and inflammation, coughing, sneezing, choking, hoarseness, dyspnoea, bronchitis, chest pain, laryngeal spasms and upper respiratory tract oedema, as well as headache and palpitations.
- The onset of respiratory symptoms may be delayed for several hours.

Ingestion

- Ingestion may result in burns, gastrointestinal bleeding, gastritis, perforations, dilatation, necrosis, stenosis and fistula formation.
- Nephritis may also develop after hydrochloric acid ingestion.
- Metabolic acidosis, massive fluid and electrolyte shifts may occur with extensive dermal or gastrointestinal burns.
- Hyperkalaemia, hyperphosphataemia, hypocalcaemia and hyperchloraemia may be noted.
- Haemolysis may occur following significant ingestion. Disseminated intravascular coagulation has been reported.

Dermal

- Chemical burns to the skin are often associated with concurrent thermal burns and trauma. Complications seen with thermal burns including cellulitis, sepsis, contractures and osteomyelitis may occur.
- Systemic toxicity from absorbed acid can occur.

Ocular

- Eye exposure may result in pain, swelling, corneal erosions and blindness.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Monitor for signs of CNS depression.
- Obtain an ECG.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain an upright CXR if GIT perforation is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- Asymptomatic patients and those who experience only minor sensations of burning of the nose, throat, eyes and respiratory tract can be discharged.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours.
- Correct fluid and electrolyte abnormalities.
- Ensure adequate analgesia is prescribed.

Inhalation

- Keep patients at absolute bed-rest whilst under observation.
- Administer humidified oxygen.
- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Make the patient nil by mouth.
- Administer intravenous fluids.
- Consider gastroenterology review.
- Consider endoscopy in patients suspected of having corrosive burns of the GIT.

Dermal

- Irrigate with copious amounts of water.
- Manage corrosive burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Hydrofluoric acid

Alternative names

Anhydrous hydrofluoric acid
Fluohydric acid
Fluoric acid
Hydrofluoride
HF

Properties of the agent

- It is a colourless to green foaming liquid with an irritating odour.
- It is the most acidic substance known.
- It is used to clean metal and etch glass. It is used in petroleum refineries.

Routes of exposure

- Oral
- Inhalation
- Eye
- Dermal

Human toxicity

General:

- It is a serious systemic poison. Its severe and sometimes delayed health effects are due to deep tissue penetration by the fluoride ion. The surface area of the burn is not predictive of its effects.
- Hydrogen fluoride is irritating to the skin, eyes and mucous membranes and inhalation may cause respiratory irritation or haemorrhage. Systemic effects can occur from all routes of exposure and may include nausea, vomiting, gastric pain, or cardiac arrhythmia. Symptoms may be delayed for several days, especially in the case of exposure to dilute solutions of hydrogen fluoride (less than 20%).
- Systemic fluoride toxicity may also result in severe hypocalcaemia, hypomagnesaemia, hyperkalaemia, metabolic acidosis, cardiac dysrhythmias and death.
- Death may result from both local injury and systemic complications of hypocalcaemia and hyperkalaemia.
Note: Rapid decontamination is critical. Use calcium containing gels, solutions or medications to neutralise the fluoride ion.

Symptoms are based on route of exposure:

Inhalation

- It produces rapid onset of eye, nose and throat irritation, cough, hoarseness, choking, dyspnoea, fever and cyanosis.
- Laryngeal oedema, chemical pneumonitis, tracheobronchitis and non- cardiogenic pulmonary oedema occur with severe exposure.

Ingestion

- Ingestion results in oro-pharyngeal burns, epigastric pain, nausea, vomiting, haematemesis and late strictures.

Dermal

- The fluoride ion readily penetrates tissues deeply to cause liquefactive necrosis of soft tissue.
- There is initial erythema, which develops central blanching, followed by blister formation and ulceration.
- Severe exposures may result in tendonitis, and decalcification and corrosion of bone.
- Extreme pain may be prolonged for days.

Ocular

- Conjunctivitis, conjunctival ischaemia, corneal opacities and corneal necrosis may occur, associated with eyelid oedema.
- Perforation of the globe may occur.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC. Calcium and magnesium should be monitored hourly.
- Monitor ECG.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain an upright CXR if GIT perforation is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.
- Consider obtaining serum and urinary fluoride levels (the fatal plasma level of fluorine is 3 mg/L; urinary fluorine output of less than 5 mg/L is used as an index of safe working level for long-term exposure). Note: These levels are not very useful in acute management.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- All patients with exposure to hydrofluoric acid or hydrogen fluoride gas should be observed for at least 6 to 8 hours.

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours as it has delayed effects.
- Commence treatment prior to the onset of symptoms where exposure to high concentrations occurred or large areas were affected.
- Apply calcium-containing gels or use intravenous calcium gluconate as appropriate.
- Replace depleted magnesium.
- Correct acidosis.
- Insulin, glucose and bicarbonate may be required to treat hyperkalaemia.
- Ensure adequate analgesia is prescribed.
- Expert clinical toxicology advice is warranted in all cases of HF exposures.

Inhalation


- Keep patients at absolute bed-rest whilst under observation.
- Administer humidified oxygen.
- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Administer nebulised calcium gluconate.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Make the patient nil by mouth.
- Use of intravenous calcium is paramount to treatment.
- Consider gastroenterology review and endoscopy in patients suspected of having corrosive burns of the GIT.

Dermal

- Irrigate with copious amounts of water.
- All dermal exposures require use of calcium to neutralise the HF. If HF is not neutralised, ongoing tissue necrosis can occur despite minimal external signs.
- Massage 2.5% calcium gluconate gel into the affected area for at least 30 minutes.
- If no gel is available, mix calcium gluconate (10ml of 10%) with KY jelly (10g), or use a calcium or magnesium solution for soaking.
- If analgesia is not achieved within 1 hour, local injections of calcium gluconate (5% solution, not to exceed 0.5ml/cm²) may be considered.
- In more serious cases involving extremities, intra-arterial calcium gluconate (10ml of 10% solution with 5% Dextrose to a total volume of 50ml, infused into the artery of the affected hand or foot, over 4 hours) is the preferred



treatment. This may be repeated up to 3 times for persistent pain.

- A Bier's block technique, with 10-20ml intravenous calcium gluconate, diluted to a total volume of 40ml, has also been used.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Hydrogen sulphide

Alternative names

Dihydrogen monosulphide
Dihydrogen sulphide
Hydrosulphuric acid
Sulphuretted hydrogen
Sulphur hydride

Properties of the agent

- It is a flammable, heavier-than-air, colourless gas with strong, offensive sulphur or 'rotten eggs' odour and a sweetish taste.
- It rapidly paralyses olfactory nerve endings in high concentrations and hence odour is not a dependable means of detecting this gas.
- It is commonly found in the presence of degrading protein waste and accumulates in confined spaces and low-lying areas. It is a leading cause of sudden death in the workplace.
- It is used in analytical chemistry and metallurgy.
- It reacts with moisture on mucous membranes to produce sodium sulphide, an irritant.
- It also inhibits cytochrome oxidase to impair cellular respiration.

Routes of exposure

- Inhalation – almost exclusively.
- Eye
- Dermal
- Ingestion – unlikely

Human toxicity

General:

- Patients may present acutely with dizziness, nausea, bradycardia, tachycardia, hyperventilation and respiratory depression even to the point of apnoea and hypotension or hypertension.

Symptoms are based on route of exposure:

Inhalation

- Short exposures to high concentrations may produce brief unconsciousness or “knockdown”.
- Prolonged exposures may cause rhinitis, pharyngitis, cough, bronchial or alveolar haemorrhage, respiratory depression, cyanosis, pulmonary oedema, bronchitis and dyspnoea may be noted.
- Headache, sweating, vertigo, anosmia, irritability, staggering gait, disorientation, somnolence, weakness, confusion and delirium may be noted following exposure to non-fatal levels.
- Asphyxial seizures, coma and death associated with rapid respiratory paralysis may be noted following exposure to high levels.
- Respiratory symptoms may take weeks or months to resolve.

Dermal

- Frostbite may occur following exposure to compressed gas.

Ocular

- Conjunctival injection, lacrimation, seeing coloured halos, ocular pain, corneal bullae, blurred vision, erythema and oedema of the eyelids, and blepharospasm may be noted.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Monitor EUC, FBC, lactate.
- Monitor ECG.
- Monitor vital signs and replace fluids.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- Asymptomatic patients and those who experience only minor sensations of burning of the nose, throat, eyes and respiratory tract can be discharged. In most cases, these patients will be free of symptoms in an hour or less.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- All symptomatic patients should be admitted and observed for at least 24 hours.
- Cardiac monitoring for 24 hours is recommended.
- Correct acidosis.
- Hyperbaric oxygen may be considered for patients with persistent coma where other treatments have proven ineffective.

Inhalation

- Administer humidified oxygen.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.
- CXR and PFTs should be repeated 2-3 months post discharge.

Dermal

- Flush affected areas thoroughly.
- Manage corrosive burns as for thermal burns.
- Handle frostbitten areas with caution. Place frostbitten skin in tepid water and allow circulation to re-establish spontaneously.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Antidote

- Consult a clinical toxicologist regarding the possible use of Sodium nitrite.

Lewisite

Alternative names	Blister agent L 2-Chlorovinyl-dichloroarsine
Properties of the agent	<ul style="list-style-type: none">• Lewisite is a dark oily liquid that gives off a colourless, heavier-than-air vapour with a "geranium-like" odour.• It causes immediate pain and irritation of exposed surfaces, followed by blisters and oedema.
Routes of exposure	<ul style="list-style-type: none">• Oral – common• Inhalation – common• Eye• Dermal – most common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Lewisite causes immediate pain, erythema and blisters in exposed surfaces; systemic absorption causes increased capillary permeability and haemodynamic collapse known as, 'Lewisite shock'.• Unlike mustard gas, Lewisite does not produce immunosuppression. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Airway irritation, burning nasal pain, aphonia, cough, pseudomembranous bronchitis, dyspnoea and pulmonary oedema may be seen.• ARDS and chemical pneumonitis may be seen in overwhelming exposure. <p>Ingestion</p> <ul style="list-style-type: none">• Nausea, salivation, vomiting, watery or bloody diarrhoea may be noted with any route of exposure. <p>Dermal</p> <ul style="list-style-type: none">• Severe erythema, blistering and chemical burns can develop rapidly. <p>Ocular</p> <ul style="list-style-type: none">• Lacrimation, pain, blepharospasm, eyelid oedema and corneal damage may be noted.• Blindness may result.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, FBC.• Monitor blood volume / central venous pressure.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Urinary arsenic excretion may help to confirm Lewisite exposure in doubtful cases but is not useful in acute management.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

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Asymptomatic patients:

- If there is no pain or irritation in patients arriving from a potential exposure, then patients can be discharged.
- There is no need to observe asymptomatic patients.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 48 hours, as they are likely to be in shock.
- Correct fluid and electrolyte abnormalities
- Ensure adequate analgesia is administered.

Inhalation

- Monitor airway patency.
- Administer humidified oxygen.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Dermal

- Flush affected areas thoroughly.
- No treatment is required for simple erythema.
- Manage skin injury as for thermal burns.
- Carbamazepine has been effective as analgesia for dermal injury.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Antidote

- Dimercaprol, also known as British Anti-Lewisite (BAL), is the specific antidote developed for lewisite. It is useful for systemic effects but has little effects on local lesions of eyes, skin or throat. Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Mercuric chloride

Alternative names	Nil known
Properties of the agent	<ul style="list-style-type: none">• Mercuric chloride is an inorganic mercury compound. It occurs as a white crystalline or powder solid.• It is used in disinfecting and preserving, gold refining, lithography, dry batteries, embalming, tanning, chemical synthesis and as a fungicide,• insecticide and analytical reagent.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation• Eye• Dermal – common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Mercuric chloride is one of the most toxic mercury salts. This review is based on the properties of inorganic mercury compounds in general, except where specific effects have been attributed to mercuric chloride.• When mercury poisoning is suspected in critically ill patients, chelation therapy should be started regardless of the form of mercury causing toxicity.• Fluid and electrolyte loss secondary to corrosion of tissues is often the cause of death in acute poisonings. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Aspiration or inhalation of inorganic mercury can lead to pneumonitis and acute tracheal, laryngeal and pulmonary oedema. <p>Ingestion</p> <ul style="list-style-type: none">• Sudden and profound circulatory collapse with tachycardia, weak and shallow pulse, hypotension and peripheral vasoconstriction can occur from ingestion of inorganic mercurials.• Haematemesis, nausea, bloody diarrhoea and oedema of the upper GIT have been reported.• Peripheral neuropathy and brain damage can occur even from acute exposures.• Renal failure may occur within 24 hours of an acute exposure. <p>Dermal</p> <ul style="list-style-type: none">• Severe skin irritation has been noted with mercuric chloride. <p>Ocular</p> <ul style="list-style-type: none">• Mercuric chloride is corrosive to the eyes and throat. Persistent visual disturbance has been reported from acute systemic poisoning.

Laboratory/radiographic testing

For minimal or mild exposures:

- No testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Monitor vital signs and replace fluids.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain whole blood mercury levels and 24-hour urine collection for mercury (Normal range for whole blood mercury levels rarely exceed 1.5 mcg/dL and normal urine excretion level rarely exceeds 15 mcg/L in unexposed individuals).

Note: Levels do not help with acute management, but can be used to confirm diagnosis later on.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Correct fluid and electrolyte abnormalities.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Give humidified oxygen for hypoxia and bronchodilators for wheeze.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert advice.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin injury.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Antidote

- Chelation therapy (D-Penicillamine and /or DMSA/Succimer) is indicated in selected symptomatic cases.

Note: For indications, contraindications, dosing regimens and clinical end points discuss with an expert clinical toxicologist.

Mercuric nitrate

Alternative names	Nil known
Properties of the agent	<ul style="list-style-type: none">• Mercuric nitrate is a colourless, odourless, inorganic mercury compound.• It is used in antiseptics, fungicides and paints.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation• Eye• Dermal – common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• This review is based on the properties of inorganic mercury compounds in general.• When mercury poisoning is suspected in critically ill patients, chelation therapy should be started regardless of the form of mercury causing toxicity.• Fluid and electrolyte loss secondary to corrosion of tissues is often the cause of death in acute poisonings. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Aspiration or inhalation of inorganic mercury can lead to pneumonitis and acute tracheal, laryngeal and pulmonary oedema. <p>Ingestion</p> <ul style="list-style-type: none">• Sudden and profound circulatory collapse with tachycardia, weak and shallow pulse, hypotension and peripheral vasoconstriction can occur from ingestion of inorganic mercurials.• Haematemesis, nausea, bloody diarrhoea and oedema of the upper GIT have been reported.• Peripheral neuropathy and brain damage can occur even from acute exposures.• Renal failure may occur within 24 hours of an acute exposure. <p>Dermal</p> <ul style="list-style-type: none">• Severe skin irritation has been noted with mercuric chloride. <p>Ocular</p> <ul style="list-style-type: none">• It is corrosive to the eyes and persistent visual disturbance has been reported from acute systemic poisoning.

Laboratory/radiographic testing

For minimal or mild exposures:

- Ni testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Monitor vital signs and replace fluids.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain whole blood mercury levels and 24-hour urine collection for mercury (Normal range for whole blood mercury levels rarely exceed 1.5 mcg/dL and normal urine excretion level rarely exceeds 15 mcg/L in unexposed individuals).
- Note: Levels do not help with acute management, but can be used to confirm diagnosis later on.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Correct fluid and electrolyte abnormalities.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Give humidified oxygen for hypoxia and bronchodilators for wheeze.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert advice.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin injury.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Antidote

- Chelation therapy (D-Penicillamine and /or DMSA/Succimer) is indicated in selected symptomatic cases.

Note: For indications, contraindications, dosing regimens and clinical end points discuss with an expert clinical toxicologist.

Mercuric oxide

Alternative names	Mercury oxide
Properties of the agent	<ul style="list-style-type: none">• Mercuric oxide is an inorganic mercury compound.• It commonly exists as a black or brownish-black powder, but may also be red, orange or yellow.• It is used as a fungicide, chemical intermediate and preservative in cosmetics. Mercuric oxide is also used in alkaline batteries and pigments.• Mercuric salts are corrosive and nephrotoxic.
Routes of exposure	<ul style="list-style-type: none">• Oral – common• Inhalation – rare but can occur• Eye• Dermal – common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• This review is based on the properties of inorganic mercury compounds in general.• When mercury poisoning is suspected in critically ill patients, chelation therapy should be started regardless of the form of mercury causing toxicity.• Fluid and electrolyte loss secondary to corrosion of tissues is often the cause of death in acute poisonings. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Severe and potentially lethal pulmonary oedema has been reported from inhalation of large amounts of elemental mercury. Mercuric salts could potentially act in the same manner. Inhalation of mercury vapour can also cause pneumonia. <p>Ingestion</p> <ul style="list-style-type: none">• Sudden and profound circulatory collapse with tachycardia, weak and shallow pulse, hypotension and peripheral vasoconstriction can occur from ingestion of inorganic mercurials.• Haematemesis, nausea, bloody diarrhoea and oedema of the upper GIT have been reported.• Adhesions have occurred after ingestion of disc batteries containing mercuric oxide.• Peripheral neuropathy and brain damage can occur even from acute exposures.• Renal failure may occur within 24 hours of an acute exposure. <p>Dermal</p> <ul style="list-style-type: none">• Severe skin irritation and dermatitis may be noted. <p>Ocular</p> <ul style="list-style-type: none">• Brown deposits of mercury in the lens and visual defects can occur.

Laboratory/radiographic testing

For minimal or mild exposures:

- Ni testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Monitor vital signs and replace fluids.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain whole blood mercury levels and 24-hour urine collection for mercury (Normal range for whole blood mercury levels rarely exceed 1.5 mcg/dL and normal urine excretion level rarely exceeds 15 mcg/L in unexposed individuals).

Note: Levels do not help with acute management, but can be used to confirm diagnosis later on.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Correct fluids and electrolytes.

Inhalation

- Give humidified oxygen for hypoxia.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert advice.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin injury.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Antidote

- Chelation therapy (**D-Penicillamine** and/or **DMSA/Succimer**) is indicated in selected symptomatic cases.

Note: For indications, contraindications, dosing regimens and clinical end points discuss with an expert clinical toxicologist.

Mercurous nitrate

Alternative names	Nil known
Properties of the agent	<ul style="list-style-type: none">Mercurous Nitrate is a colourless inorganic mercury compound with a slight odour.It is used in disinfecting and preserving, gold refining, lithography, dry batteries, embalming, tanning, chemical synthesis and as a fungicide, insecticide and analytical reagent.
Routes of exposure	<ul style="list-style-type: none">Oral – most commonInhalationEyeDermal – common
Human toxicity	<p>General: This review is based on the properties of inorganic mercury compounds in general.</p> <ul style="list-style-type: none">When mercury poisoning is suspected in critically ill patients, chelation therapy should be started regardless of the form of mercury causing toxicity.Fluid and electrolyte loss secondary to corrosion of tissues is often the cause of death in acute poisonings. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">Severe and potentially lethal pulmonary oedema has been reported from inhalation of large amounts of elemental mercury. Inhalation of mercury vapour can also cause pneumonia. <p>Ingestion</p> <ul style="list-style-type: none">Sudden and profound circulatory collapse with tachycardia, weak and shallow pulse, hypotension and peripheral vasoconstriction can occur from ingestion of inorganic mercurials.Haematemesis, nausea, bloody diarrhoea and oedema of the upper GIT have been reported.Peripheral neuropathy and brain damage can occur even from acute exposures.Renal failure may occur within 24 hours of an acute exposure. <p>Dermal</p> <ul style="list-style-type: none">Severe skin irritation and dermatitis may be noted. <p>Ocular</p> <ul style="list-style-type: none">Brown deposits of mercury in the lens and visual defects can occur.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
 - Monitor vital signs and replace fluids.
 - If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
 - Obtain whole blood mercury levels and 24-hour urine collection for mercury (Normal range for whole blood mercury levels rarely exceed 1.5 mcg/dL and normal urine excretion level rarely exceeds 15 mcg/L in unexposed individuals).
- Note:** Levels do not help with acute management, but can be used to confirm diagnosis later on.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.

Inhalation

- Give humidified oxygen for hypoxia.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert advice.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin injury.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Antidote

- Chelation therapy (**D-Penicillamine** and/or **DMSA/Succimer**) is indicated in selected symptomatic cases.
- Note:** For indications, contraindications, dosing regimens and clinical end points discuss with an expert clinical toxicologist.

Methyl fluoroacetate

Alternative names	Fluoroacetic acid, methyl ester Methylester kyseliny fluoroctove TL 551 Acetic acid, fluoro-, methyl ester
Properties of the agent	<ul style="list-style-type: none">• It is an odourless, tasteless, highly water-soluble and readily absorbed, white powder. It is usually mixed with a black dye. It remains stable for long periods of time due to its carbon-fluorine bond.• It is highly stable and toxic.• It is also widely used to make sodium fluoroacetate or fluoroacetate-1080.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation – common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• It is rapidly absorbed and may cause systemic toxicity after any route of exposure.• Clinical effects usually develop within 30 minutes to 2.5 hours of exposure, but may be delayed for as long as 20 hours. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Respiratory depression and pulmonary oedema have been reported. <p>Ingestion</p> <ul style="list-style-type: none">• Prolonged QTc intervals (hypocalcaemia), ventricular tachycardia or fibrillation and asystole may occur.• Hyperactive behaviour, auditory hallucinations, cerebellar dysfunction, loss of speech, paraesthesia, seizures, coma, carpopedal spasm and neurologic impairment may be noted.• Nausea, vomiting, excessive salivation and diarrhoea may be noted. Mild hepatic dysfunction has been reported.• Metabolic acidosis, hyperglycaemia, hypocalcaemia and hyperuricaemia may be noted
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• Obtain an ECG and institute continuous cardiac monitoring. Measure QTc.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Fluoroacetate levels are not clinically useful.• Samples for analysis should include suspected baits, vomitus, stomach contents, liver and kidney.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 hours as it has delayed effects.
- Cardiac monitoring is required for at least 24 hours.
- Treat seizures with benzodiazepines.
- Monitor and correct fluids and electrolytes, especially calcium concentration.

Inhalation

- Give humidified oxygen for hypoxia.
- Treatment of non-cardiogenic pulmonary oedema may include invasive or non-invasive ventilation.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert advice.

Methyl mercury

Alternative names	Organic mercury
Properties of the agent	<ul style="list-style-type: none">• Methyl mercury is an organic mercury compound, used as a fungicide in some countries. It is formulated as either an aqueous solution or a dust.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation – rare• Eye• Dermal – common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Toxicity following acute exposures may be delayed for weeks to months, with predominant GIT and CNS effects. Methyl mercury concentrates in the nervous system and red blood cells and is considered to be the most neurotoxic (cerebellar damage) of the mercury compounds.• When mercury poisoning is suspected in critically ill patients, chelation therapy should be started regardless of the form of mercury causing toxicity.• Early symptoms are a metallic taste in the mouth, numbness and tingling of digits and face, tremor, headache, fatigue, emotional lability and cognitive dysfunctioning.• Severe symptoms include incoordination, hearing loss, constriction of visual fields, spasticity of muscles and deterioration of mental capacity. Death is not uncommon and significant neurological damage is common in survivors. <p>Dermal:</p> <ul style="list-style-type: none">• Organic mercury can be absorbed through the skin to produce systemic effects. Irritation or burns can result from exposure to some compounds. Sensitisation has been reported.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• Monitor vital signs, replace fluids and correct electrolyte disturbances.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Obtain whole blood mercury levels and 24-hour urine collection for mercury (Normal range for whole blood mercury levels rarely exceed• 1.5 mcg/dL and normal urine excretion level rarely exceeds 15 mcg/L in unexposed individuals). <p>Note: Levels do not help with acute management, but can be used to confirm diagnosis later on.</p>

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert advice.

Dermal

- Flush exposed areas thoroughly.
- Manage corrosive burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Antidote

- Chelation therapy (**D-Penicillamine** and /or **DMSA/Succimer**) is indicated in selected symptomatic cases.

Note: For indications, contraindications, dosing regimens and clinical end points discuss with an expert clinical toxicologist.

Mustard

Alternative names

HD
H
Sulphur mustard
Nitrogen mustard
Yperite
Distilled mustard
Bis (2-chloroethyl) sulphide

Properties of the agent

- Mustard is a colourless to light brownish-yellow liquid.
- It has a faint garlic, mustard or onion smell.
- It readily vapourises in warm weather.
- It was introduced as a chemical warfare agent in 1917 during World War I.

Routes of exposure

- Oral – unlikely
- Inhalation – most common
- Eye
- Dermal

Human toxicity

General:

- Mustard is a vesicant and alkylating agent.
- It causes blisters and oedema of exposed surfaces; it also has systemic effects similar to that of ionising radiation.
- Mustard can cause cellular changes within minutes of contact but the onset of pain and other clinical effects may be delayed for 1 to 24 hours.
- Nausea, vomiting and epigastric pain can occur regardless of the route of entry.
- Mustard can lead to immunosuppression with leucopenia, thrombocytopenia and anaemia.

Symptoms are based on route of exposure:

Inhalation

- Sore throat, hoarseness, dyspnoea, paroxysmal cough, oedema, blistering and ulceration of the airways may occur. Lung parenchyma is usually spared, except where there is prolonged exposure to high concentrations.
- Acute symptoms may persist for up to 1 year.
- Chronic sequelae, including asthma, bronchitis, tracheal/bronchial stenosis and emphysema, may develop over the next ten to fifteen years following significant exposure.

Ingestion

- Ingestion of contaminated food or water may result in nausea, vomiting, abdominal pain, bloody diarrhoea and dehydration.

Dermal

- Erythema, stinging and itching usually develop within a few hours following dermal exposure. Blisters, bullae and burns form within 24 hours.
- Maximal vesiculation occurs by 72 hours.
- Complete healing of skin lesions occurs by 29 days.
- Purplish discolouration of the skin and depigmentation may persist.

Ocular

- Pain, photophobia, lacrimation, blepharospasm, corneal and conjunctival ulceration with temporary (common) or permanent (rare) blindness may develop.
- Keratitis may recur for up to 40 years.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Monitor EUC, FBC.
- Monitor vital signs and replace fluids.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Monitor immunological function.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

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Asymptomatic patients:

- Patients who are asymptomatic or with mild exposure may be discharged with advice to rest, to return if symptoms recur or develop.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 48 to 72 hours as it has delayed effects.
- Correct fluid and electrolyte imbalance.
- Ensure adequate analgesia is prescribed.
- Monitor immunological function until full recovery occurs even after discharge from the hospital. Neutropenia and thrombocytopenia have occurred up to 2 weeks post exposure.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Administer humidified oxygen for hypoxia and bronchodilators for bronchospasm.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.
- Long-term follow up of PFTs is required.

Dermal:

- Decontaminate affected persons. Manage skin injury as for thermal burns.
- Avoid excessive hydration, as fluid losses will be less than comparable thermal injury.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Nerve agents (dermal)

Alternative names	VX
Properties of the agent	<ul style="list-style-type: none">• VX is a liquid anti cholinesterase nerve agent.• Nerve agents with low volatility work primarily by being rapidly absorbed through skin.• VX is the typical example of this class and a drop or less on the skin is a potentially fatal dose.
Routes of exposure	<ul style="list-style-type: none">• Inhalation – common• Eye• Dermal – most common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Nerve agents cause CNS effects (confusion, coma, convulsions), muscarinic effects (salivation, lacrimation, urination, diaphoresis/sweating, gastrointestinal symptoms, emesis, miosis, bronchorrhoea, bronchospasm and bradycardia) and nicotinic effects (fasciculations, muscle cramps, flaccid paralysis, apnoea, tachycardia and hypertension). <p>Dermal:</p> <ul style="list-style-type: none">• Dermal exposure to liquid nerve agent results in a latent period, from 15 minutes to 18 hours, during which the exposed person remains asymptomatic. The latent period is followed by the precipitant onset of symptoms.• Mild dermal exposure causes localised sweating and fasciculation.• Moderate dermal exposure causes nausea, vomiting, diarrhoea and generalised weakness.• Severe dermal exposure causes loss of consciousness, seizures, generalised fasciculation, flaccid paralysis, apnoea, generalised increased secretions, and involuntary urination and defaecation.• Fasciculations are highly specific for anticholinesterase poisoning but are often not seen. Fasciculations localised to the exposure site may be transient and the small area affected easily overlooked. <p>Ocular:</p> <ul style="list-style-type: none">• Miosis is unusual in dermal exposures to nerve agent.• The usual causes of death are respiratory failure and refractory hypotension.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, FBC.
- Monitor ECG
- Monitor for signs of CNS depression.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Exposure can be confirmed by measurement of low plasma and red cell cholinesterase activity (organophosphates and carbamate insecticides may also reduce these); severity correlates to some extent with the extent of inhibition.
- **Note:** Levels do not help with acute management, but can be used to confirm diagnosis later on. The test is not readily available in all hospitals.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Decontaminate exposed persons.
- Patients with significant exposures should be admitted and observed for at least 72 hours in ICU as patients may deteriorate rapidly.
- Administer supplemental oxygen therapy, alleviate bronchospasm with atropine, and intubate and ventilate if required.
- Administer intravenous fluids.
- Administer antidote.
- Patients with hypotension not responding to intravenous fluids have a poor prognosis.

Antidote

- **Atropine** is the most important antidote. Large doses (5 to 20 mg) are often required, but this is usually less than in severe organophosphates pesticide poisoning. It is given as escalating doubling boluses to reverse the muscarinic signs (e.g. 2mg, 4mg, 8mg, 16 mg, etc., intravenous at 5-minute intervals. Then an infusion at 10 to 20% of the total bolus dose per hour should be commenced. Boluses and infusion of atropine should be sufficient to keep the heart rate greater than 80 beats a minute, systolic BP > 80 mmHg and the lungs clear.
- Atropine does not reverse the nicotinic effects or miosis.
- Patients with moderate or severe poisoning should receive **pralidoxime** with an initial dose of 2 g (30 mg/kg) intravenously over 30 minutes followed by an infusion of 500 mg/hour (8 mg/kg/h) after the initial dose. This should be continued for at least 24 hours and up to 7 days depending on the patient's state.
Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.
- Benzodiazepines such as diazepam are used to control seizures.

Nerve agents (volatile)

Alternative names	Soman (GD) Sarin (GB) Tabun (GA)
Properties of the agent	<ul style="list-style-type: none">• They are volatile anticholinesterase nerve agents.• Nerve agents with high volatility act primarily by evaporating and then being inhaled.• Sarin is a typical example of this class and inhalation of sarin may be rapidly fatal.
Routes of exposure	<ul style="list-style-type: none">• Oral – rare• Inhalation – most common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Nerve agents cause CNS effects (confusion, coma, convulsions), muscarinic effects (salivation, lacrimation, urination, diaphoresis/sweating, gastrointestinal symptoms, emesis, miosis, bronchorrhoea, bronchospasm and bradycardia) and nicotinic effects (fasciculations, muscle cramps, flaccid paralysis, apnoea, tachycardia and hypertension).• Survivors of the sarin release in Tokyo reported severe headache and myalgia.• The usual causes of death despite medical treatment are respiratory failure and refractory hypotension. <p>Inhalation:</p> <ul style="list-style-type: none">• Inhalational exposure to nerve agent vapour causes symptoms in seconds to minutes.• Mild vapour exposure causes miosis, rhinorrhoea, mild bronchoconstriction, and mild bronchorrhoea.• Moderate vapour exposure causes miosis, rhinorrhoea, bronchoconstriction, and increased secretions.• Severe vapour exposure causes loss of consciousness, seizures, generalised fasciculation, flaccid paralysis, apnoea, generalised increased secretions, and involuntary urination and defaecation.• Generalised fasciculations appear as ripples under the skin, and may persist after consciousness and voluntary movement have been regained. <p>Ocular:</p> <ul style="list-style-type: none">• Miosis is associated with conjunctival injection, blurred vision, dimmed vision, and eye pain, especially on focusing. Impaired papillary dilation may persist for up to 9 weeks.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, FBC.
- Monitor ECG.
- Monitor for signs of CNS depression.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Exposure can be confirmed by measurement of low plasma and red cell cholinesterase activity (organophosphates and carbamate insecticides may also reduce these); severity correlates to some extent with the extent of inhibition.

Note: Levels do not help with acute management, but can be used to confirm diagnosis later on. The test is not readily available in all hospitals.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Decontaminate exposed persons.
- Patients with significant exposures should be admitted and observed for at least 72 hours in ICU as patients may deteriorate rapidly.
- Administer supplemental oxygen therapy, alleviate bronchospasm with atropine, and intubate and ventilate if required.
- Administer intravenous fluids.
- Administer antidote.
- Patients with hypotension not responding to intravenous fluids have a poor prognosis.

Antidote

- **Atropine** is the most important antidote. Large doses (5 to 20 mg) are often required, but this is usually less than in severe organophosphates pesticide poisoning. It is given as escalating doubling boluses to reverse the muscarinic signs (e.g. 2mg, 4mg, 8mg, 16 mg, etc., intravenous at 5-minute intervals. Then an infusion at 10 to 20% of the total bolus dose per hour should be commenced. Boluses and infusion of atropine should be sufficient to keep the heart rate greater than 80 beats a minute, systolic BP > 80 mmHg and the lungs clear.
- Atropine does not reverse the nicotinic effects or miosis.
- Patients with moderate or severe poisoning should receive **pralidoxime** with an initial dose of 2 g (30 mg/kg) intravenously over 30 minutes followed by an infusion of 500 mg/hour (8 mg/kg/h) after the initial dose. This should be continued for at least 24 hours and up to 7 days depending on the patient's state.
Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.
- Benzodiazepines are used to control seizures.
- Atropine eye drops may be used to relieve eye pain.

Nickel chloride (hydrated)

Alternative names	Nickel (II) chloride Nickel (2+) chloride Nickel dichloride
Properties of the agent	<ul style="list-style-type: none">• Soluble nickel salt is an odourless, golden yellow or green powder, or brown solid in the form of scales.• It is commonly used in nickel plating, ceramics, the dye and printing industry and as an adsorbent of ammonia in gas masks.
Routes of exposure	<ul style="list-style-type: none">• Oral – unlikely• Inhalation – common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• There is limited human data available for Nickel Chloride Hydrated. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Sore throat, hoarseness, bronchospasm, cough, shortness of breath may develop. May also cause occupational asthma. Chronic exposure may cause pulmonary interstitial fibrosis. <p>Ingestion</p> <ul style="list-style-type: none">• Relatively non-toxic in small amounts. Acute exposure to large amounts may cause severe gastrointestinal irritation with nausea, vomiting, abdominal pain and diarrhoea. Symptoms may occur within 2 hours.• Headache, giddiness and myalgia have been reported.• Hyperbilirubinaemia has been reported. <p>Dermal</p> <ul style="list-style-type: none">• Acute dermatitis (most common reaction) and pruritus may be noted.• Usually there is limited systemic absorption from dermal exposure. Once acquired, nickel sensitivity usually persists. <p>Ocular</p> <ul style="list-style-type: none">• Eye irritation may be noted.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 hours.

Inhalation

- Administer humidified oxygen for hypoxia and bronchodilators for bronchospasm.

Ingestion

- Do not induce vomiting.
- Nickel is eliminated mainly in the urine, so ensure adequate intravascular volume and urine output is maintained.
- Use of activated charcoal is controversial.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin injury

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Nitric acid

Alternative names	Engraver's acid Red fuming nitric acid White fuming acid Aqua fortis Hydrogen nitrate Nitrous fumes
Properties of the agent	<ul style="list-style-type: none">• It is a colourless to yellow or brownish-red corrosive, non-flammable, fuming liquid with a characteristic-choking odour.• It is used in the manufacture of nylon, polyurethane, fertilisers and explosives.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalational – most common• Eye• Dermal – common

Human toxicity

General:

- It is a severe corrosive irritant of the skin, eyes and mucous membranes.
- On exposure to organic matter, nitric acid releases nitric oxide which oxidises haemoglobin to methaemoglobin

Symptoms are based on route of exposure:

Inhalation

- Inhalation of nitric acid fumes produces nose, throat and laryngeal burning and irritation, pain and inflammation, coughing, sneezing, choking, hoarseness, dyspnoea, bronchitis, chest pain, laryngeal spasms and upper respiratory tract oedema, chemical pneumonitis and non-cardiogenic pulmonary oedema, as well as headache and palpitations.
- The onset of respiratory symptoms may be delayed for 3–30 hours.

Ingestion

- Ingestion may result in burns, gastrointestinal bleeding, gastritis, perforations, dilatation, oedema, necrosis, and vomiting. Stenosis and fistula formation are late events.
- Metabolic acidosis, massive fluid and electrolyte shifts may occur with extensive dermal or gastrointestinal burns.
- Hyperkalaemia, Hyperphosphataemia, hypocalcaemia and hyperchloraemia may be noted.
- Haemolysis may occur following significant ingestion. Disseminated intravascular coagulation has been reported.

Dermal

- Immediate severe penetrating burns with deep ulceration are characteristic.

Ocular

- Eye exposure may result in pain, swelling, corneal erosions and blindness. It causes yellow opacification of the cornea.
- Perforation of the globe may occur.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Monitor for signs of CNS depression.
- Obtain an ECG and institute continuous cardiac monitoring. Monitor QTc.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain an upright CXR if GIT perforation is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
131126

Asymptomatic patients:

- Asymptomatic patients and those who experience only minor sensations of burning of the nose, throat, eyes and respiratory tract can be discharged.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours.
- Correct fluid and electrolyte imbalance.
- Ensure adequate analgesia is prescribed.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Administer humidified oxygen.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Make the patient nil by mouth.
- Administer intravenous fluids.
- Consider gastroenterology review.
- Consider endoscopy in patients suspected of having corrosive burns of the GIT.

Dermal

- Flush exposed areas thoroughly.
- Manage corrosive burns as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Organophosphates

Alternative names	Chlorpyrifos, Coumaphos, Diazinon, Dichlorvos, Dimethoate, Fenthion, Malathion, Parathion, Trichlorfon. Carbamates: Aldicarb, Car bendazim, Carbendazole, Carbazine, Propoxur.
Properties of the agent	<ul style="list-style-type: none">• Organophosphates and carbamates are used as pesticides.• They are classified as anticholinesterase agents and most life-threatening form of poisoning is via oral route.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation• Eye – rare• Dermal – common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Anticholinesterase poisonings usually present with muscarinic features (excessive secretions, respiratory distress, abdominal pain, diarrhoea, and small pupils) and CNS features (agitation, confusion, coma). The heart rate and blood pressure are frequently abnormal but may be high or low.• Nicotinic effects include fasciculations, tremor, weakness, respiratory muscle paralysis, tachycardia and hypertension. Muscle weakness and loss of deep tendon reflexes are also common but may be delayed in onset. Fasciculations are highly specific for anticholinesterase poisoning but are often not seen.• The usual causes of death despite medical treatment are respiratory failure and refractory hypotension.• Intermediate syndrome is delayed paralysis (2-4 days) and is associated with particular agents (e.g. fenthion, diazinon, malathion).• Organophosphate induced delayed neuropathy (OPIDN) is rare and occurs 1 to 5 weeks post exposure to particular agents (e.g. fenthion, chlorpyrifos, parathion). It is an ascending sensorimotor polyneuropathy.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• Monitor for signs of CNS depression.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Exposure can be confirmed by measurement of low plasma and red cell cholinesterase activity (nerve agents and carbamate insecticides may also reduce these); severity correlates to some extent with the extent of inhibition. <p>Note: Levels do not help with acute management, but can be used to confirm diagnosis later on. The test is not readily available in all hospitals.</p>

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours in ICU as patients may deteriorate rapidly.
- Administer supplemental oxygen therapy.
- Administer intravenous fluids.
- Administer antidote.
- Benzodiazepines are the preferred treatment for organophosphate related seizures.
- Patients with hypotension not responding to intravenous fluids have a poor prognosis.

Antidote

- **Atropine** is the most important antidote. Large doses (up to 1 gram) may be required. It is given as escalating doubling boluses to reverse the muscarinic signs (e.g. 2mg, 4mg, 8mg, 16 mg, etc., intravenous at 5- minute intervals. Then an infusion at 10 to 20% of the total bolus dose per hour should be commenced. Boluses and infusion of atropine should be sufficient to keep the heart rate greater than 80 beats a minute, systolic BP > 80 mmHg and the lungs clear.
- Patients with moderate or severe poisoning should receive **pralidoxime** with an initial dose of 2 g (30 mg/kg) intravenously over 30 minutes followed by an infusion of 500 mg/hour (8 mg/kg/h) after the initial dose. This should be continued for at least 24 hours and up to 7 days depending on the patient's state.

Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice. Pralidoxime is indicated in some organophosphate exposures but not in carbamate exposures.

Paraquat

Alternative names

Dimethyl viologen
Gramoxone S
Methyl viologen
Dimethyl bipyridylium dimethylsulphate
Dimethyl bipyridylium dichloride

Properties of the agent

- Paraquat is available as colourless crystals (dichloride salt) or a yellow solid (bismethyl sulphate) salt). It is soluble in water with a faint ammonia-like odour.
- It is a herbicide.
- Paraquat causes NADPH depletion and free radical and superoxide formation, which continues indefinitely in the presence of NADPH and oxygen. Superoxides disrupt cell structures and functions.

Routes of exposure

- Oral – most common.
- Inhalation – paraquat is non-volatile, however if the formulation contains a stenching agent this may cause nausea.
- Dermal – prolonged contact may result in systemic absorption.

Human toxicity

General:

- Paraquat is extremely toxic if ingested. One mouthful of 20% paraquat is potentially lethal. Violent, protracted vomiting suggests a significant intoxication.
- Blue vomitus or blue discolouration of the tongue is characteristic.
- Supplemental oxygen should be avoided in poisonings.
- Large doses result in death within hours from multi organ failure.
- Lower doses cause pulmonary damage with death occurring in days or weeks.

Symptoms:

General

- Systemic symptoms include headache, lethargy, myalgia, hepatic damage and jaundice, pancreatitis, metabolic acidosis, coma and convulsions.

Respiratory system

- Progressive pulmonary fibrosis associated with dyspnoea and pulmonary oedema may occur 3 to 14 days following exposure to paraquat.
- Pulmonary haemorrhage may occur with acute exposure.

Ingestion

- Ventricular arrhythmias, hypotension and cardio respiratory arrest may occur with large ingestions.
- Nausea, vomiting, diarrhoea and abdominal pain are common.
- Pancreatitis may develop in some cases of acute paraquat poisoning.
- Paraquat is also caustic to the oral, oesophageal and gastric mucosa and may cause perforation.
- Transient reversible liver injury may be noted 24 to 96 hours following exposure.
- Delayed glomerulonephritis, possibly of immunological origin, may be noted. Renal failure and functional renal insufficiency secondary to hypovolaemia may be noted.
- Methaemoglobinaemia has been observed after ingestion of a paraquat solution.

Dermal

- Paraquat is poorly absorbed through intact skin (absorbed only through abraded or injured skin).
- On intact skin it may result in erythema and ulceration developing over 1-3 days.

Ocular

- Protracted opacification of the cornea may result following eye exposure.

Laboratory/radiographic testing

For minimal or mild exposures:

- All cases of paraquat ingestion should be considered as potentially fatal and admitted.

For significant exposures:

- Serum paraquat levels may be performed and this level can be used to predict the likelihood of survival. Unfortunately this test is rarely performed in Australian hospitals and usually can take a week to receive the result.
- Perform dithionate test on acidified urine. A change in colour (to blue) indicates the presence of paraquat in the urine.
- Monitor EUC, LFTs, CMP, FBC. Serum amylase is indicated if pancreatitis is suspected.
- Monitor methaemoglobin levels if methaemoglobinaemia is suspected.
- Obtain an ECG and institute continuous cardiac monitoring.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- All cases of paraquat ingestion must be treated as potentially fatal and admitted.

Symptomatic patients:

General advice

- Consultation with a clinical toxicologist is recommended for all patients with exposure to paraquat.
- Patients with significant exposures should be admitted and observed closely for days. Death may occur within 24 to 36 hrs. If they survive this, patients may die days to weeks later of pulmonary fibrosis
- Minimise use of supplemental oxygen as much as possible, as it may increase the risk of pulmonary injury. Do not exceed paO₂ of 90%.

Ingestion

- Do not induce vomiting.
- Control vomiting, preferably with a serotonin antagonist.
- **Activated charcoal** may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert clinical toxicology advice. **Fuller's Earth** is an alternative to activated charcoal. If neither of these is available the patient should be given any available food.
- Administer intravenous fluids and correct electrolyte and acid-base disturbance.
- Consult a clinical toxicologist for use of **N-acetyl cysteine**, **steroids** and **aspirin**, **vitamin C** in significant exposures.
- Haemodialysis and haemoperfusion may be of benefit if performed early.

Dermal

- Flush affected areas thoroughly.
- Manage skin injury as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Perchloric acid

Alternative names	<p>Perchloric acid – hydronium perchlorate, dioxonium perchlorate</p> <p>Also perchlorates – ammonium perchlorate, magnesium perchlorate, potassium perchlorate, and sodium perchlorate</p>
Properties of the agent	<ul style="list-style-type: none">• Perchloric acid is clear liquid that has no odour.• The anhydrous form of this material is an explosion hazard. Perchloric acid is stable at concentrations below 73%.• Ammonium perchlorate, an oxidizer, is widely used in solid propellants, fireworks, flares and munitions components.• Perchlorate is used in diagnosis and treatment of Graves' disease.• Perchlorate is a competitive inhibitor of sodium/iodine transport, decreasing the active transport of iodine into the thyroid. It was used as an anti-thyroid drug in the treatment of hyperthyroidism in the 1950s and 1960s but was discontinued because of the occasional occurrence of aplastic anaemia. More recently, lower doses of perchlorate have been used successfully in the treatment of iodine-induced hyperthyroidism.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation• Eye• Dermal

Human toxicity

General

- Extremely hazardous in case of inhalation (lung corrosive). Very hazardous in case of skin contact (corrosive, irritant), of eye contact (corrosive), or ingestion.
- Perchloric acid wounds heal very slowly.
- Perchlorates – thyrotoxic.

Inhalation

- Inhalation may produce severe irritation of respiratory tract, characterised by coughing, choking, or shortness of breath and delayed lung oedema. Inhalation may be fatal as a result of spasm, inflammation, oedema of the larynx and bronchi, chemical pneumonitis, and pulmonary oedema.

Dermal

- Skin inflammation is characterised by itching, scaling, reddening, or occasionally blistering. Skin burns and deep penetrating ulcerations are seen with high concentrations.

Ocular

- Inflammation of the eye is characterised by redness, watering and itching. Loss of vision may occur at higher concentrations.

Ingestion

- Causes burns of the mouth, pharynx and gastrointestinal tract with pain, dysphagia, necrotic areas, epigastric pain, nausea and vomiting, corrosive ulceration, gastric bleeding, profound thirst, scanty urine, shock and circulatory collapse and metabolic derangement.

Potential chronic health effects

- Repeated or prolonged contact with spray mist may produce chronic eye irritation, severe skin irritation and respiratory tract irritation leading to frequent attacks of bronchial infection.
- Prolonged or repeated inhalation may cause nosebleeds, nasal congestion, erosion of the teeth, perforation of the nasal septum, chest pain and bronchitis.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC, Thyroid function.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain an ECG.
- Monitor for signs of CNS depression.
- Obtain an upright CXR if GIT perforation is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- Asymptomatic patients and those who experience only minor sensations of burning of the nose, throat, eyes and respiratory tract can be discharged.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours.
- Correct fluid and electrolyte imbalance.
- Ensure adequate analgesia is prescribed.

Inhalation

- Keep patients at absolute bed-rest whilst under observation.
- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Administer humidified oxygen for hypoxia and bronchodilators to relieve bronchospasm.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Make the patient nil by mouth.
- Administer intravenous fluids.
- Consider gastroenterology review.
- Consider endoscopy in patients suspected of having corrosive burns of the GIT.

Dermal

- Irrigate with copious amounts of water.
- Manage skin injury as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Perfluoro isobutene

Alternative names	PFIB Perfluoroisobutylene Isobutene, octafluoro Octafluoroisobutylene Octafluoro-sec-butene
Properties of the agent	<ul style="list-style-type: none">• Perfluoroisobutene (PFIB) is a colourless gas.• It is usually encountered as a result of burning polytetrafluoroethylene (Teflon) or perfluoroethylpropylene (Halon) in household or industrial fires.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation – most common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• PFIB causes respiratory irritation, pulmonary oedema and polymer fume fever, usually within 4 hours of exposure. Often, it can produce delayed effects. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Respiratory tract irritation, pneumonitis, non-cardiogenic pulmonary oedema and mild hypoxia may develop. Haemorrhagic inflammation of the lungs, with bloody sputum, may also occur.• Hyperpyrexia, mild sinus tachycardia and reversible mild hypertension may be noted. <p>Ingestion</p> <ul style="list-style-type: none">• Nausea and vomiting may occur. <p>Ocular</p> <ul style="list-style-type: none">• Chemical conjunctivitis has been described.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Based on history and clinical picture. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, CMP, FBC.• Obtain an ECG.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs. CXR findings of pulmonary oedema worsen up to 12 hours post exposure.• Urinary fluoride excretion can confirm polymer exposure in doubtful cases. (Urinary fluorine output of less than 5 mg/L is used as an index of safe working level for long-term exposure). <p>Note: These levels do not help with acute management and are not routinely available in all Australian hospitals.</p>

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- As the severity of the initial symptoms does not correlate with the severity of the exposure, all asymptomatic patients should be observed for at least 24 hours.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24-48 hours.

Inhalation

- Administer humidified oxygen.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ocular

- Irrigation may be used to relieve eye irritation.

Phenol

Alternative names	Benzenol Carbolic acid Hydroxybenzene Monohydroxybenzene Oxybenzene
Properties of the agent	<ul style="list-style-type: none">• Phenol is composed of colourless or white acicular (radiating, needle- like) crystals when perfectly pure. It is discoloured by exposure to light and air, which makes this compound turn pink or red. Discolouration is hastened by the presence of alkalinity or impurities.• It has a burning taste and its odour has been described as distinctive and aromatic.• It is used in the manufacture of resins, plastics and disinfectants.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation – limited• Eye• Dermal – phenol is rapidly absorbed, leading to systemic toxicity

Human toxicity

General:

- Concentrated phenol is extremely corrosive and may cause oral, oesophageal and gastric burns following ingestion.
- Symptoms often involve a transient CNS stimulation followed by CNS depression. Coma and seizures can occur in minutes.

Symptoms are based on route of exposure:

Inhalation

- Tachypnoea is commonly reported; pulmonary oedema and bronchospasm may also occur.
- Stridor has been reported from exposure to high concentrations of phenol.

Ingestion

- Oral and oesophageal burns may result from ingestion. Exposure may result in hepatic injury, which may result in death.
- Dysrhythmias, hypotension and tachycardia have been reported in patients following ingestion and other exposures.
- Methaemoglobinaemia may occur in large ingestions

Dermal

- Phenol is corrosive to the skin, but because of anaesthetic properties, it numbs rather than causing a burning pain on contact. Skin becomes red and swollen, then white and opaque. Deep burns may result that become gangrenous.
- Mild dermal contact with phenolic compounds may result in irritation, dermatitis and abnormal pigmentation.
- Diaphoresis may be noted with systemic toxicity.

Ocular

- Eye exposure may result in severe burns. Partial or complete loss of vision may be noted.

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC, Methaemoglobin.
- Obtain an ECG and institute continuous cardiac monitoring.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain an upright CXR if GIT perforation is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.
- Phenol urine concentrations should be monitored to determine if they are within normal range (0.5 to 81.5 mg/L) however they do not help with acute management and are not routinely available in all Australian hospitals.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 hours.
- Methylene blue is indicated for symptomatic methaemoglobinaemia.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Administer humidified oxygen. Inhaled beta agonists for bronchospasm.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Consider gastroenterology review and endoscopy in patients suspected of having corrosive burns of the GIT.

Dermal

- Irrigate with copious amounts of water.
- Manage skin injury as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Antidote for methaemoglobinaemia

- **Methylene Blue** should be administered if the methaemoglobinemia exceeds 20%. 1-2mL/kg methylene blue intravenously over 5 minutes. This can be repeated after 60 minutes.
- Measure methaemoglobin levels hourly until a consistent fall is documented.
- If methylene blue fails to control methaemoglobinaemia, consider exchange transfusion or hyperbaric oxygen therapy.

Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Phosgene

Alternative names	Carbonic dichloride Carbon oxychloride Carbonyl chloride Chloroformyl chloride
Properties of the agent	<ul style="list-style-type: none">• Phosgene is a colourless gas with the smell of "newly mown hay".• It is produced commercially by chlorinating carbon monoxide.• It reacts slowly with water to give off hydrochloric acid and carbon dioxide.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation – most common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Phosgene is a pulmonary oedemagen.• The severity of the initial symptoms is no indication of the severity of the exposure. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Irritation of the upper airways causes cough, a burning sensation of the throat, dyspnoea and pain in the chest may occur.• Severe non-cardiogenic pulmonary oedema can develop after a delay of 1 to 24 hours. The severity and timing of the onset of the pulmonary oedema may be exacerbated by physical activity following exposure. Hypotension, bradycardia and sinus arrhythmias may be associated.• Haemolysis and sludging may occur within the pulmonary capillaries.• Death may be caused by respiratory failure or right heart failure.• Arterial blood gases and lung function tests may remain abnormal for several months. Chronic bronchitis and emphysema may ensue.• Nausea and vomiting may occur. <p>Dermal</p> <ul style="list-style-type: none">• Severe dermal burns or frostbite may be noted following skin exposure to the liquefied material. <p>Ocular</p> <ul style="list-style-type: none">• Irritation, lacrimation, photophobia, blepharospasm, corneal opacities and perforation may occur.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, FBC.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- As the severity of the initial symptoms does not correlate with the severity of the exposure, all asymptomatic patients should be observed for at least 24 hours.

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24-48 hours.
- Correct acidosis.

Inhalation

- Administer humidified oxygen.
- Keep patients at absolute bed-rest whilst under observation.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.
- CXR and PFTs should be repeated 2-3 months post discharge.

Dermal

- Flush affected areas thoroughly.
- Manage corrosive burns as for thermal burns.
- Handle frostbitten areas with caution. Place frostbitten skin in tepid water and allow circulation to re-establish spontaneously.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Phosgene oxime

Alternative names	CX Cycloheximide Dichloroformoxime Blister agent 'Nettle rush' gas or nettle agent
Properties of the agent	<ul style="list-style-type: none">• Phosgene oxime is a colourless, crystalline solid or a yellowish-brown liquid with a strong odour.• The solid material will produce enough vapour to cause injury.• The mechanism of injury is unknown.• It penetrates garments and rubber very quickly and causes immediate pain with tissue damage. Extreme pain may last for several days. <p>Note: Persons whose clothing or skin is contaminated with liquid or solid phosgene oxime can cause secondary contamination by direct contact or through off-gassing vapour. Persons exposed only to phosgene oxime vapour pose no risk of secondary contamination.</p>
Routes of exposure	<ul style="list-style-type: none">• Oral – rare• Inhalation – most common• Eye – common• Dermal – common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• CX is an urticant or a nettle agent, which is capable of producing wheals, erythema and urticaria. It also causes immediate pain and corrosive burns to exposed surfaces. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Immediate pain and irritation of the upper airways, followed by dyspnoea and pulmonary oedema may be noted. <p>Dermal</p> <ul style="list-style-type: none">• Pain, 'nettle rash' and skin necrosis with brown pigmentation may be noted.• Within 5 to 20 seconds there is greyish blanching of the affected area. Marginal erythema and oedema develop over 5 to 30 minutes. Necrosis occurs over several hours. The lesion becomes pigmented over 24 hours. An eschar develops over 7 days.• The skin injury may extend to muscle.• Healing may be incomplete at 4 to 6 months.• Pulmonary oedema can occur through skin absorption. <p>Ocular</p> <ul style="list-style-type: none">• Pain, corneal ulceration, conjunctivitis, dimming of vision and blindness may be noted.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, FBC.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- Patients arriving from the scene of potential release within 30 to 60 minutes will have pain or irritation if they were exposed. These patients should be observed for at least 24 to 48 hours due to delayed effects.
- Asymptomatic patients should be discharged home. See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures and symptoms should be admitted and observed for at least 24 to 48 hours due to the risk of developing pulmonary oedema.

Inhalation

- Administer humidified oxygen.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Dermal

- Flush affected areas thoroughly.
- Manage skin injury as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Phosphine

Alternative names	Hydrogen phosphide Phosphorus hydride Phosphorus trihydride
Properties of the agent	<ul style="list-style-type: none">• Phosphine gas is produced from phosphide salts (e.g. Al, Mg, Zn and Ca).• It is colourless with the odour of garlic and rotten fish.• It is slightly heavier than air but is easily dispersed.• It causes severe oxidative stress and interferes with cellular metabolism.• It is used as a fumigant/rodenticide.
Routes of exposure	<ul style="list-style-type: none">• Oral – following ingestion, effects may be delayed up to 1 week• Inhalation – freely absorbed and excreted by the lungs• Eye• Dermal

Human toxicity

General:

- Acute phosphine gas or phosphide salt poisoning usually leads to symptoms within a few hours but the effects may get progressively worse over the first 24 hours.
- Exposure to the gas leads to irritant and respiratory effects, whereas ingestion of phosphide salts presents predominantly with organ failure and intractable hypotension.
- Following significant exposure methaemoglobinaemia, haemolysis and disseminated intravascular coagulopathy (DIC) are possible complications. Hepatic and renal toxicity is possible as is lactic acidosis. Severe electrolyte abnormalities (particularly of K, Ca and Mg) are also possible.
- Death occurs within 4 to 14 days.

Symptoms are based on route of exposure:

Inhalation

- Mild exposures to Phosphine can cause nasal and eye irritation, cough, headache, fatigue, nausea, vomiting and abdominal pain.
- More serious exposures typically cause severe dyspnoea, pulmonary oedema, cardiac arrhythmias and hypotension.

Ingestion

- Local gastrointestinal effects include anorexia, retrosternal pain, nausea, vomiting, diarrhoea and abdominal pain.
- Multiple organ failure with acute cardiac failure, shock, ARDS, acute renal and hepatic failure may lead rapidly to death.
- CNS effects (headache, paraesthesias, ataxia, tremor, weakness, diplopia, seizures, coma) usually only occur in severe poisoning and may be secondary to the multi-organ failure.

Dermal

- Phosphide salts can be absorbed readily through broken skin and cause systemic symptoms as listed above. Phosphine gas has limited toxicity by this route.
- Contact with liquefied phosphine gas may cause frostbite.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC, COAGS, serum lactate.
- Monitor methaemoglobin levels, haemoglobin, haematocrit and plasma free haemoglobin in patients with methaemoglobinemia. Methaemoglobinemia interferes with pulse oximetry reading, resulting in falsely high values.
- Urinalysis positive for blood with few or no red blood cells (RBC's) is an early indication of haemolysis.
- If respiratory tract irritation or respiratory depression is evident, monitor ABGs, CXR and PFTs.
- Monitor ECG and blood pressure.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- Asymptomatic patients with a history of exposure to phosphine must be observed for at 4 to 6 hours, and discharged with instructions to return if symptoms develop.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24–48 hours.
- Patient may deteriorate quickly.
- Correct fluid, electrolyte, acid-base imbalances and coagulation abnormalities.
- Benzodiazepines may be used to control seizures.
- Methylene blue is indicated in cases of symptomatic methaemoglobinaemia.

Inhalation

- Administer humidified oxygen.
- Keep patients at absolute bed rest whilst under observation.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert clinical toxicology advice.

Dermal

- Flush exposed areas thoroughly.
- Handle frostbitten areas with caution. Place frostbitten skin in tepid water and allow circulation to re-establish spontaneously.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Antidote for methaemoglobinaemia

- **Methylene Blue** should be administered if the methaemoglobinemia exceeds 20%. 1-2mL/kg methylene blue intravenously over 5 minutes. This can be repeated after 60 minutes.
- Measure methaemoglobin levels hourly until a consistent fall is documented.
- If methylene blue fails to control methaemoglobinaemia, consider exchange transfusion or hyperbaric oxygen therapy.

Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Phosphorus oxychloride

Alternative names	Phosphorus chloride Phosphorus chloride oxide Phosphorus oxide trichloride Phosphorus oxytrichloride f Phosphoryl chloride Phosphoryl trichloride
Properties of the agent	<ul style="list-style-type: none">• Phosphorus oxychloride is a clear, corrosive liquid with a pungent odour.• It is commonly use in the manufacturing of plasticisers, pesticides, non- flammable hydraulic fluids, flame-retardants for plastics, and is a precursor for Tabun (GA), a nerve agent.• It reacts violently with water to produce hydrogen chloride and phosphoric acid.
Routes of exposure	<ul style="list-style-type: none">• Oral – common• Inhalation – most common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Phosphorus compounds can cause pulmonary oedema and may cause chemical burns to exposed surfaces. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• A sense of suffocation, cough, bronchitis, retrosternal chest discomfort, pulmonary oedema dyspnoea and wheezing may be noted. <p>Ingestion</p> <ul style="list-style-type: none">• It may produce severe burns of mouth, throat and stomach.• Nephritis, albuminuria and microscopic haematuria may be noted. <p>Dermal</p> <ul style="list-style-type: none">• Skin burns may be noted. <p>Ocular</p> <ul style="list-style-type: none">• Irritation, conjunctivitis and corneal burns have been reported.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Obtain an upright CXR if GIT perforation is suspected.• Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24-48 hours.

Inhalation

- Monitor airway patency.
- Administer humidified oxygen.
- Administer inhaled beta agonists for bronchospasm.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Keep nil by mouth.
- Consider gastroenterology review and endoscopy in patients suspected of having corrosive burns of the GIT.

Dermal

- Irrigate with copious amounts of water.
- Manage skin injury as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

3-Quinuclidinyl benzilate

Alternative names	Agent 3 Agent Buzz Agent 15 QNB BZ Agent BZ EA-2277
Properties of the agent	<ul style="list-style-type: none">• 3-Quinuclidinyl benzilate, commonly known as BZ is a chemical warfare agent with anticholinergic properties, which affects both peripheral and central nervous system.• BZ is also classified as an odourless hallucinogenic agent, which would most likely be disseminated as an aerosol, with the primary route of absorption through the respiratory system.• Its pharmacologic activity is similar to other anticholinergics, but with a much longer duration of action of 48 to 72 hours, and up to 5 days in higher dose exposures.
Routes of exposure	<ul style="list-style-type: none">• Oral – 80 % bioavailability• Inhalation – most common (40–50% bioavailability)• Eye – not applicable• Dermal – 5 to 10% bioavailability via intact skin, with effects delayed 24 hours

Human toxicity

General:

- Symptoms can last 2-4 days.
- 'Dry as a bone, red as a beet, hot as a hare, mad as a hatter'.
- Exposure to BZ (most likely aerosol) causes an anticholinergic syndrome. Signs/symptoms are dependent on the dose and time post exposure. Prolonged effects may occur depending on the dose of BZ absorbed.
- CNS effects predominate and may include restlessness, apprehension, abnormal speech, confusion, agitation, tremor, ataxia, stupor and coma. Hallucinations are prominent. Motor coordination, perception, cognition and new memory formation may be altered.

Peripheral nervous system effects may include:

- Mydriasis may last 3 days, and result in photophobia, conjunctival injection and eye pain.
- Tachycardia, rarely exceeding 150 beats/minute. Moderate increases in blood pressure may be noted.
- Decreased intestinal motility, with decreased secretions from the stomach, pancreas and gallbladder. Nausea and vomiting may occur.
- Drying of oral mucous membranes. Breath may develop a foul odour.
- Red and flushed skin.
- Urinary retention and enlarged bladder may be palpable on examination.
- Increased temperature from inability to sweat and dissipate heat. Marked hyperthermia may be noted in hot environments.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Consider BZ if a number of people arrive after an exposure to an unknown substance and manifest an anticholinergic syndrome.
- There is no diagnostic test for BZ exposure.
- Monitor EUC, FBC. Ensure good urine output (as BZ is renally excreted).
- Monitor for signs of CNS depression.
- Obtain an ECG.
- Monitor for signs of hyperthermia.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24–48 hours or until asymptomatic.
- Decontaminate in well-ventilated area. Remove, double-bag and seal contaminated clothing. Patient should be washed with copious amounts of water under low pressure for at least 10–15 minutes.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert advice.

Antidote

- Physostigmine may be indicated for symptom management in severely affected persons. However, it will not shorten the duration of symptoms.

Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Sodium azide

Alternative names	Azide Azium
Properties of the agent	<ul style="list-style-type: none">• It is an odourless, white crystalline solid.• It rapidly hydrolyses in water to form hydrazoic acid and hydrogen azide gas – a colourless, volatile, highly explosive liquid with a characteristic odour (described as 'sickening').• It is used as a chemical reagent and in the manufacture of airbags (rapidly breaks down to nitrogen).
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation – common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• It inhibits cytochrome oxidase and interferes with cellular respiration and aerobic metabolism. Sodium azide causes clinical effects like carbon monoxide, hydrogen sulphide or cyanide.• Clinical effects may be nearly immediate or delayed in onset. Effects in some cases have required days or months to resolve.• It is potent hypotensive agent regardless of route of exposure.• Mild to moderate effects include headache, weakness, mild hypotension, syncope, nausea, vomiting, diarrhoea, abdominal pain, feeling of apprehension, and malaise.• Severe effects include severe hypotension, blurred vision, CNS depression, seizures, coma, hyperthermia or hypothermia, sweating, bradycardia or tachycardia, ECG changes, arrhythmias, lactic acidosis, and pulmonary oedema. <p>Symptoms are based on route of exposure:</p> <p>Inhalation/ingestion</p> <ul style="list-style-type: none">• Inhalation or ingestion may cause headache, weakness, syncope, coma and seizures.• Nausea, vomiting, diarrhoea and polydipsia may occur.• Nasal irritation, rhinorrhoea, dyspnoea, tachypnoea, cough, chest pain, pulmonary oedema and respiratory failure can develop. <p>Dermal</p> <ul style="list-style-type: none">• Burns or blisters may be seen. <p>Ocular</p> <ul style="list-style-type: none">• Conjunctivitis, mydriasis and blurred vision may be noted.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC, serum lactate.
- Monitor ECG and blood pressure.
- Sodium azide poisoning can cause high anion gap metabolic acidosis.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Decontamination should be undertaken in a well-ventilated area. Contaminated clothes should be removed, double-bagged, sealed and stored safely. Decontaminate open wounds. Pay particular attention to mucous membranes-skin folds, fingernails and ears.
- Patients with significant exposures should be admitted and observed for at least 24-48 hours.
- Exclude cyanide exposure, as there are specific antidotes for the treatment of cyanide poisoning.
- Commence intravenous fluids.
- Correct acidosis.
- Vasopressors may be required.

Inhalation

- Administer humidified oxygen.
- Relieve bronchospasm with bronchodilators.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert clinical toxicology advice.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin injury.

Sodium chlorate

Alternative names	B-herbttox Chlorate of soda Chloric acid, sodium salt Natrium chlorat Chlorate salt of sodium
Properties of the agent	<ul style="list-style-type: none">• Sodium chlorate is a colourless, odourless crystal or granule• It is used as a herbicide.• Chlorates are very potent oxidising agents.
Routes of exposure	<ul style="list-style-type: none">• Oral – readily absorbed by ingestion• Inhalation – systemic effects may be seen with prolonged exposure• Eye• Dermal – not readily absorbed by this route
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Chlorate oxidises haemoglobin and glutathione, and inactivates G6PD. The resultant red cell membrane rigidity causes methaemoglobinemia and haemolysis. The rate of conversion to metHb is insidious.• Effects result from hypoxia secondary to methaemoglobinemia, haemolysis and disseminated intravascular coagulation.• Chlorate poisoning is characterised by a latent period of up to 12 hours, followed initially with nausea, vomiting and diarrhoea. This is followed by arterial hypotension, cyanosis, and haemolysis (with jaundice, dark urine and secondary hyperkalaemia).• Renal failure may result from a direct nephrotoxic effect or be secondary to acute tubular necrosis or haemolysis.• Elevated LFTS, hepatomegaly and jaundice have also been described.• CNS effects are the consequence of hypoxia.• Death may occur within 4 hours or after 34 days. Death in the early stage of chlorate poisoning is due to anoxia from methaemoglobinaemia or to DIC. Later death is generally due to renal failure. <p>Ingestion/dermal/ocular</p> <ul style="list-style-type: none">• There is an irritant effect on the gut with nausea, vomiting and diarrhoea.• The effect on the skin and eyes is irritant

Laboratory/radiographic testing

For minimal or mild exposures:

- Based on history and clinical picture.

For significant exposures:

- Monitor EUC, LFTs, FBC, COAGS.
- Monitor methaemoglobin levels, haemoglobin, haematocrit and plasma free haemoglobin in patients with methaemoglobinemia. Methaemoglobinemia interferes with pulse oximetry reading, resulting in falsely high values.
- Urinalysis positive for blood with few or no red blood cells (RBC's) is an early indication of haemolysis.
- Closely monitor urinary output.
- If respiratory tract irritation or respiratory depression is evident, monitor ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 72 hours.
- Cardiac monitoring for 24 hours is recommended.
- Correct fluid, electrolyte, acid-base imbalances and coagulation abnormalities.

Inhalation

- Administer humidified oxygen.
- Ventilatory support may be required.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert clinical toxicology advice. Activated charcoal is thought to be of limited value.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin irritation.

Antidote

- Consider administering Sodium thiosulphate to symptomatic patients to inactivate the chlorate ion by conversion to the chloride ion, following expert clinical toxicology advice.
Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.
- Chlorate-induced metHb is refractory to treatment with Methylene Blue, because the chlorate inactivates the pathway to enzymatically reduce metHb.
- Special Situations: Exchange transfusion combined with haemodialysis should be considered in severely intoxicated patients.

Sodium fluoroacetate

Alternative names	Fluoroacetate-1080 Compound 1080 Ratbane SMFA 1080
Properties of the agent	<ul style="list-style-type: none">• It is an odourless, tasteless, highly water-soluble and readily absorbed, white powder. It is usually mixed with a black dye. Mixture with water tastes similar to vinegar. It remains stable for long periods of time due to its carbon-fluorine bond.• In Australia, it is widely used as a pesticide especially in the rural regions. One of the commonly manufactured products is known as 'Foxoff' baits.
Routes of exposure	<ul style="list-style-type: none">• Oral – most common• Inhalation – common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• It is rapidly absorbed and may cause systemic toxicity after any route of exposure.• Clinical effects usually develop within 30 minutes to 2.5 hours of exposure, but may be delayed for as long as 20 hours. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Respiratory depression and pulmonary oedema have been reported.• In severe cases, pneumonitis and ARDS can also occur and may be delayed for several hours. <p>Ingestion</p> <ul style="list-style-type: none">• Early systemic features may include agitation, confusion, apprehension, tachypnoea and sweating.• Tachycardia, bradycardia, prolonged QTc interval (hypocalcaemia), ventricular tachycardia or fibrillation and asystole may occur.• Hyperactive behaviour, auditory hallucinations, cerebellar dysfunction, loss of speech, paraesthesia, seizures, coma, carpopedal spasm and neurological impairment may be noted.• Nausea, vomiting, excessive salivation and diarrhoea may be noted. Mild hepatic dysfunction has been reported.• Metabolic acidosis, hyperglycaemia, hypocalcaemia and hyperuricaemia may be noted.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing required.

For significant exposures:

- Monitor EUC, LFTs, CMP, glucose, FBC.
- Obtain an ECG and institute continuous cardiac monitoring. Monitor QTc.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Fluoroacetate levels are not clinically useful.
- Samples for analysis should include suspected baits, vomitus, stomach contents, liver and kidney.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information 131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Carry out decontamination in well-ventilated area. Remove, double-bag and seal away any contaminated clothing. Patient should be washed with water for at least 10-15 minutes.
- Patients with significant exposures should be admitted and observed for at least 24 hours as it has delayed effects.
- Cardiac monitoring is required for at least 24 hours.
- Correct fluid, electrolyte and acid-base imbalances.
- Control seizures with benzodiazepines.

Inhalation

- Administer humidified oxygen.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following advice from a clinical toxicologist.
- Treat hypocalcaemia with intravenous calcium.
- Treat agitation and convulsions with titrated benzodiazepines.
- Manage hypotension and metabolic acidosis with adequate fluid resuscitation in addition to sodium bicarbonate if required following discussion with a clinical toxicologist.

Sodium sulphide (hydrated)

Alternative names	Sodium sulphide anhydrous or containing < 30% water crystallization. Sodium monosulphide Sodium sulfuret Disodium sulphide
Properties of the agent	<ul style="list-style-type: none">• It is in the form of yellow or brick-red lumps or flakes or deliquescent crystals and has an odour of rotten eggs.• It is highly flammable. Reacts with water, steam, or acid to produce toxic and corrosive gasses e.g. H₂S.
Routes of exposure	<ul style="list-style-type: none">• Oral – common• Inhalation – most common• Eye• Dermal
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Sodium Sulphide (hydrated) has a primary irritant and corrosive effect on skin, eyes and mucous membranes. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Stridor, dyspnoea, upper airway injury and pulmonary oedema may occur. <p>Ingestion</p> <ul style="list-style-type: none">• Ingestion may result in burns to the lips, tongue, oral mucosa and upper airway.• Burns of the oesophagus and less commonly the stomach may occur after caustic ingestion; the absence of oral mucosal injury does not reliably exclude oesophageal burns. Patients with stridor, drooling or vomiting are more likely to have oesophageal burns.• In severe cases gastrointestinal bleeding or perforated viscus with mediastinitis or peritonitis may develop.• Systemic hydrogen sulphide toxicity is a possibility following reaction with stomach acid. <p>Dermal</p> <ul style="list-style-type: none">• Severe skin irritation and / or burns may be noted. <p>Ocular</p> <ul style="list-style-type: none">• Inhalation / ocular exposure may lead to severe burns to the eyes.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain an upright CXR if GIT perforation is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.

Note: This agent requires consultation with a clinical toxicologist. Please call 13 11 26 for advice regarding management.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 48 hours.
- Correct fluid and electrolytes imbalances.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Administer humidified oxygen.
- Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Keep patient nil by mouth.
- Consider gastroenterology review and endoscopy in patients suspected of having corrosive burns of the GIT.
- If systemic symptoms – consider hydrogen sulphide toxicity.

Dermal

- Irrigate with copious amounts of water.
- Manage skin injury as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmological review for all patients with persistent eye symptoms following irrigation.

Sulphur dioxide

Alternative names	SO ₂ Bisulphite Sulfur oxide Sulfurous acid anhydride Sulfurous anhydride Sulfurous oxide
Properties of the agent	<ul style="list-style-type: none">• Non-flammable, colourless gas that is heavier than air at room temperature.• It has a strong pungent odour detectable at 0.3–1 ppm by most people. The odour threshold is 5 times lower than the OSHA permissible exposure limit of 5 ppm.• Liquid sulphur dioxide corrodes iron, copper and brass and some plastics and rubber.• Many metals ignite in unheated sulphur dioxide.
Routes of exposure	<ul style="list-style-type: none">• Oral exposure is unlikely, as sulphur dioxide is a gas at room temperature.• Inhalation is the major route of exposure (up to 90% absorbed).• Eye exposure to 10-20 ppm causes irritation of mucous membranes.• Dermal absorption is negligible.

Human toxicity

General:

- Sulphur dioxide reacts with moisture on mucous membranes to form sulphurous acid (H₂SO₃), a severe irritant.

Symptoms are based on route of exposure:

Inhalation

- Increased airway resistance may occur at < 0.1 ppm in asthmatics, and 5 ppm in healthy individuals. Above 10 ppm there is cough and sneezing, and > 20 ppm bronchospasm. 50–100 ppm may be tolerated for up to 1 hour, but higher or longer exposures may cause death.
- Some individuals develop severe symptoms due to hyper-reactivity at very low exposure concentrations.
- Symptoms include sneezing, sore throat, wheezing, shortness of breath, chest tightness, and a feeling of suffocation.
- Bronchospasm, pneumonitis, pulmonary oedema, and acute airway obstruction due to laryngeal oedema and reflex spasm may occur.
- Hypotension, arrhythmias, coma, seizures and death may occur.
- Exposure to high concentrations can lead to pulmonary fibrosis, chronic bronchitis and reactive airway disorder, or chemically-induced asthma subsequently.
- Children are more vulnerable because of smaller airway diameters, higher minute volumes per kilogram, and lower likelihood of taking avoidance measures.

Ingestion

- Highly sensitive individuals may develop bronchospasm after exposure to trace amounts of sulphur dioxide used as a preservative in foods and wine.
- Nausea, vomiting and abdominal pain may occur following moderate inhalational exposures.

Dermal

- Stinging pain, erythema and blisters, especially on mucous membranes.
- Exposure to compressed gas may result in frostbite injury.

Ocular

- Irritation of mucous membranes occurs at 10-20 ppm.
- Lacrimation, conjunctivitis, corneal burns, erosion and necrosis resulting in blindness may occur.
- Exposure to compressed gas may result in frostbite injury.

Laboratory/radiographic testing

For minimal or mild exposures

- Obtain baseline PEFr, pulse oximetry and ECG.

For significant exposures

- Monitor pulse oximetry and PEFr.
- Perform continuous ECG monitoring. Monitor blood pressure.
- Obtain chest X-ray.
- Obtain ABG if indicated.
- Severely affected patients should have long-term follow-up lung function tests.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- As sulphur dioxide is a potent irritant, asymptomatic patients only need a short period of observation.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients exposed only to sulphur dioxide gas with no symptoms of eye or skin irritation do not need decontamination.
- Decontamination may be required to relieve skin and eye irritation, or following exposure to liquid sulphur dioxide. DO NOT apply neutralising chemicals as heat produced may cause thermal burns and increase injury.
- Observe for 24-48 hours. Respiratory symptoms may take up to 18-24 hours to develop.
- Consider admission for patients with persistent respiratory symptoms or chemical burns. Respiratory symptoms may progress over the following 18 to 24 hours.
- Smoking exacerbates respiratory symptoms and patients should be advised to avoid smoking for 72 hours after exposure.
- Discharged patients should be reviewed at 24 hours if they had initial skin or eye symptoms.
- Please Note: This agent requires consultation with a clinical toxicologist.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Provide humidified oxygen.
- Treat bronchospasm with bronchodilators
- Consider racemic adrenaline for children who develop stridor.
- Support respiration as required. PEEP or CPAP may be required for pulmonary oedema.
- Administer fluids for hypotension and treat arrhythmias appropriately.

Dermal

- Flush affected areas thoroughly.
- Manage burns as for thermal burns.
- Handle frostbitten areas with caution. Place frostbitten skin in tepid water and allow circulation to re-establish spontaneously.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange urgent ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Sulphuric acid

Alternative names

Battery acid
BOV
Chamber acid
Fertiliser acid
Oil of vitriol
Hydrogen sulphate
Oleum

Properties of the agent

- It is a colourless, non-flammable oily liquid when pure and brownish when impure.
- It is odourless but has a choking odour when hot.
- Pure sulphuric acid exists as a solid at temperatures below 10.5°C.

Routes of exposure

- Oral
- Inhalation
- Dermal
- Eye

Human toxicity

General:

- Sulphuric acid is corrosive to the skin, eyes and mucous membranes.
- May be fatal following ingestion, inhalation or dermal exposure.

Symptoms are based on route of exposure:

Inhalation

- Inhalation of sulphuric acid mist causes a reflex increase in respiratory rate with bronchospasm. Exposure to strong mineral acids may produce circulatory collapse with clammy skin, weak and rapid pulse and shallow respirations.
- Initial symptoms include sneezing, sore throat, cough, headache, confusion and ataxia.
- Dyspnoea develops 3 to 30 hours later. Pneumonitis, laryngeal and pulmonary oedema may occur.
- Bronchitis, pulmonary fibrosis and emphysema are late effects.

Ingestion

- Epigastric pain, haematemesis, nausea and vomiting can occur.
- Corrosion, necrosis and perforation of the oesophagus or stomach especially at the pylorus may develop.
- Delayed complications may include strictures (especially of the pylorus) and fistula formation.
- Metabolic acidosis may be noted following severe ingestion.

Dermal

- Immediate, severe penetrating burns occur with deep ulceration, necrosis and scarring.
- Circulatory collapse may occur following extensive dermal injury.
- Dermatitis may be noted.

Ocular

- The eyes are especially sensitive to the corrosive and irritant effects of sulphuric acid. Its vapour or mist is a strong irritant and can cause lacrimation and conjunctivitis. Splash contact may cause corneal burns, visual loss and rarely perforation of the globe.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Perform continuous ECG monitoring. Monitor blood pressure.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Obtain an upright CXR if perforation of the GIT is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- Asymptomatic patients and those who experience only minor sensations of burning of the nose, throat, eyes and respiratory tract can be discharged.
- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours.
- Ensure adequate analgesia is prescribed.

Inhalation

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Administer humidified oxygen. Inhaled beta agonists for bronchospasm. Non-cardiogenic pulmonary oedema should be managed with PEEP or CPAP.

Ingestion

- Do not induce vomiting.
- Make the patient nil by mouth.
- Administer intravenous fluids.
- Consider gastroenterology review and endoscopy in patients suspected of having corrosive burns of the GIT.

Dermal

- Flush affected areas thoroughly.
- Manage skin injury as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Sulphuryl fluoride

Alternative names

F₂O₂S
O₂SF₂
SO₂F₂
Sulfonyl fluoride
Sulfuric oxyfluoride
Sulfuryl fluoride
Sulphur difluoride dioxide
Sulphuryl difluoride
Profume[®]
Vikane[®]

Properties of the agent

- Non-flammable, colourless, odourless gas that is heavier than air at room temperature.
- Transported as liquefied, compressed gas.
- Used as a fumigant for the control of insect pests.
- Frequently combined with chloropicrin as a warning agent. Chloropicrin is irritant and a lachrymator.

Routes of exposure

- Inhalation is the major route of exposure.
- Eye exposure.
- There is no data on oral or dermal absorption.

Human toxicity

General:

- Sulphuryl fluoride is rapidly absorbed and widely distributed, with highest concentrations in lungs, spleen and kidneys. It is excreted rapidly, predominantly in urine. Sulphuryl fluoride is metabolised to sulphate and fluoride ions. Fluoride toxicity is the primary mode of injury.

Symptoms are based on route of exposure:

Inhalation

- Symptoms of mild exposure include eye and respiratory irritation, sore throat, wheeze and cough.
- Anorexia, nausea, vomiting, diarrhoea, cramping abdominal pain, fever and headache may occur.
- Hypocalcaemia, hypomagnesaemia and hyperkalaemia may occur in association with elevated serum fluoride levels.
- Prolonged exposure or exposure to high concentrations can lead to hypoxaemia, cardiac arrhythmias (including torsade de pointes), hypotension, salivation, lachrymation, rhinorrhoea, chest pain, respiratory distress due to non-cardiogenic pulmonary oedema, respiratory depression, paraesthesias, carpopedal tetany, agitation, confusion, collapse, convulsions and death.
- The onset of arrhythmias and non-cardiogenic pulmonary oedema may be delayed up to 24 hours.
- Severe exposure can cause brain, renal and hepatic injury.
- Children are more vulnerable because of smaller airway diameters, higher minute volumes per kilogram, and lower likelihood of taking avoidance measures.

Ingestion

- Not applicable.

Dermal

- Pruritus has been reported.
- Exposure to compressed gas may result in frostbite injury.

Ocular

- Irritation of mucous membranes.
- Exposure to compressed gas may result in frostbite injury.

Laboratory/radiographic testing

- Monitor EUC, CMP and fluoride (normal fluoride levels are < 20µg/L).
- Monitor ECG and blood pressure continuously. Measure QTc interval.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

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Asymptomatic patients:

- Exposed persons may have little evidence of intoxication initially, unless exposed to very high concentrations of sulphuryl fluoride. Respiratory symptoms may progress over the following 18 to 24 hours. Observe all exposed persons for 24 hours with strict bed rest.

Symptomatic patients:

General advice

- Patients exposed only to sulphuryl fluoride gas do not need decontamination.
- Observe all symptomatic, exposed patients for 24 hours with strict bed rest.
- Correct electrolyte imbalances, particularly hypocalcaemia.

Inhalation

- Establish a patent airway via intubation if necessary.
- Provide humidified oxygen for hypoxia and bronchodilators for bronchospasm.
- Support respiration as required. PEEP or CPAP may be required for pulmonary oedema.
- Administer fluids for hypotension and treat arrhythmias appropriately. Torsade de pointes may require overdrive pacing or cardioversion.
- Treat convulsions with benzodiazepines.

Dermal

- Handle frostbitten areas with caution. Place frostbitten skin in tepid water and allow circulation to re-establish spontaneously.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange urgent ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Thallium sulphate

Alternative names

Nil known

Properties of the agent

- It is a colourless to white crystalline solid.
- It is used as a rodenticide and an insecticide.

Routes of exposure

- Oral – most common
- Inhalation
- Eye
- Dermal

Human toxicity

General:

- Thallium is a cellular poison, blocking ATP formation and sulfhydryl cross-linking. Its distribution is similar to potassium.
- Symptoms are delayed 12 to 24 hours following exposure.
- Insidious onset with a variety of toxic manifestations.
- Anaemia and thrombocytopenia may occur.

Symptoms are based on route of exposure

Ingestion

- Mild symptoms include anorexia, stomatitis, salivation, nausea and diarrhoea.
- Severe exposures cause severe paroxysmal abdominal pain, vomiting haematemesis and massive diarrhoea resulting in hypovolaemia.
- Survivors develop sensory neuropathy 1 to 5 days after exposure followed by motor neuropathy, cranial nerve palsies, paralysis and respiratory failure. Convulsions, delirium and coma may occur. Fever is a poor prognostic feature.
- Death may be caused by respiratory paralysis, circulatory disturbances and / or pneumonia. Ventricular arrhythmias, toxic cardiomyopathy and ARDS may occur.
- Alopecia is a common feature but may take 2-3 weeks to occur.
- Neurological damage resolves slowly and may be permanent.

Dermal

- Diaphoresis, dry and scaly skin and eruptions may be noted. Black pigmentation of the hair root becomes apparent within four days. Mee's lines appear after two to four weeks.

Ocular

- Decreased visual acuity and colour vision along with ophthalmoplegia and optic neuritis are common. Optic nerve atrophy may occur long term.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Monitor ECG and blood pressure continuously for first 24 hours.
- If respiratory tract irritation or respiratory depression is evident, monitor ABGs, CXR and PFTs.
- Thallium is radio-opaque and an abdominal film should be obtained whenever thallium ingestion is suspected.
- Thallium is excreted in the urine for many weeks following ingestion or dermal exposure. The most reliable test for thallium is a 24-hour urine quantitative assay, however it is not useful in acute management. Normal value is less than 10 mcg/litre/24 hours.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 24 to 48 hours.
- Resuscitate with IV fluids. Vasopressors may be required.
- Ensure adequate analgesia is prescribed.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert clinical toxicology advice.

Antidote

- **Oral Prussian blue** (not commercially available in Australia) and haemodialysis are effective in clearing thallium, reducing the elimination half-life from 8 to 1.4 days.
- The adult dose of Prussian blue is 3 g orally three times daily until urinary thallium excretion is less than 0.5 mg/day. The child dose is 1 g orally three times daily. Laxatives may be required to treat constipation associated with Prussian blue therapy.

Note: For indications, contraindications, dosing regimens and clinical end points, seek expert clinical toxicology advice.

Toluene

Alternative names	Methyl benzene Toluol
Properties of the agent	<ul style="list-style-type: none">• Toluene (methyl benzene), an aromatic hydrocarbon and benzene derivative, is a highly volatile and colourless, clear refractive liquid with a sweet, pungent aromatic odour.• Its odour has also been described as sour or burnt.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation – most common• Eye• Dermal
Human toxicity	<p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Inhalation may cause irritation, acute bronchitis, bronchospasm, pulmonary oedema, pneumonitis and asphyxia. Chronic abusers may develop respiratory failure.• In large concentrations, toluene can cause dizziness, weakness and confusion. <p>Ingestion</p> <ul style="list-style-type: none">• Ingestion or inhalation may cause vomiting, abdominal cramps and diarrhoea. Rarely, hepato-renal failure has been attributed to toluene abuse or occupational exposure. Hepatomegaly and impaired liver function have also been reported.• Transient distal renal tubular acidosis (with hyperchloraemic metabolic acidosis, hypokalaemia and urine pH >5.5) is common in paint sniffers who have been hospitalised. Isolated cases of irreversible renal insufficiency, glomerulonephritis, focal segmental glomerulosclerosis, acute interstitial nephritis and renal failure secondary to myoglobinuria have been reported. <p>Dermal</p> <ul style="list-style-type: none">• Prolonged contact may cause drying and superficial burns. <p>Ocular</p> <ul style="list-style-type: none">• Splash exposure of the eye causes transient irritation and superficial injury. Chronic abuse is associated with decreased visual acuity, impaired colour vision and optic atrophy.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Obtain an ECG.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed for at least 12-24 hours.
- Cardiac monitoring is essential for first 12- 24 hours following ingestion.
- Correct fluid, electrolyte and acid-base imbalances.

Inhalation

- Provide humidified oxygen.
- Treat bronchospasm with bronchodilators.
- Support respiration as required. PEEP or CPAP may be required for pulmonary oedema.

Ingestion

- Do not induce vomiting.
- Activated charcoal may be indicated in selected cases (alert, cooperative adults who present within 1 hour of ingestion) following expert clinical toxicology advice.

Dermal

- Flush affected areas thoroughly.
- Manage skin injury as for thermal burns.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Triethanolamine

Alternative names	Nitrilo-2, 2', 2"-triethanol Sterolamide TEA Triethanolamin Triethylolamine Trolamine
Properties of the agent	<ul style="list-style-type: none">• Triethanolamine is a hygroscopic, viscous liquid, which has a slight odour of ammonia. It turns brown upon exposure to light and air.• It is miscible with water, methanol and acetone and is soluble in chloroform, benzene and ether.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation• Eye• Dermal – most common
Human toxicity	<p>General:</p> <ul style="list-style-type: none">• Triethanolamine is a strong irritant with low acute toxicity; toxic effects are due chiefly to its alkalinity. Degree of irritation is dependent on its concentration, exposure duration and site of exposure. Large oral doses in animals have produced minimal toxicity.• Little human data is available concerning adverse effects, although there is considerable opportunity for exposure to triethanolamine in occupational settings and in consumer products. <p>Inhalation</p> <ul style="list-style-type: none">• Bronchospasm has been reported.• Irritant gases (nitrogen oxides, sulphur dioxide) and carbon monoxide can be produced when triethanolamine is heated to decomposition. <p>Ingestion</p> <ul style="list-style-type: none">• Alkali burns of mouth, pharynx and oesophagus may occur. <p>Dermal</p> <ul style="list-style-type: none">• Contact dermatitis has been reported.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Monitor vital signs and replace fluids.
- If the patient has been exposed to triethanolamine heated to decomposition, there is a risk of inhalation exposure to nitrogen oxides, carbon monoxide and carbon dioxide. Monitor carboxyhaemoglobin.
- Determine carboxyhaemoglobin (COHb) level when the patient is first seen and repeat every 2 to 4 hours until patient is asymptomatic or level is within the normal range.
Note: COHb levels correlate poorly with signs and symptoms of toxicity. Interpretation may be confounded by delays in obtaining blood samples and therapeutic interventions (oxygen administration). The so called classic 'cherry-red skin' of carbon monoxide poisoning is rare.
- Monitor ECG, EUC, CK, ABGs if symptomatic or if the COHb level is greater than 20%. Pulse oximetry is not a reliable estimate of oxyhaemoglobin saturation.
- If respiratory tract irritation is evident, monitor pulse oximetry and PEFr.
- Obtain an upright CXR if GIT perforation is suspected.
- Investigations for oesophageal or gastric burns would be dependent on the institution, either CT chest and abdomen, or endoscopy.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
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Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed until asymptomatic.

Inhalation

- Provide humidified oxygen.
- Treat bronchospasm with bronchodilators.

Ingestion

- Establish a patent airway via intubation if necessary. A surgical airway may be required.
- Do not induce vomiting.
- Make the patient nil by mouth.
- Administer intravenous fluids.
- Consider gastroenterology review.
- Consider endoscopy in patients suspected of having corrosive burns of the GIT.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin irritation.

Vanadium oxysulphate

Alternative names	Vanadyl sulphate
Properties of the agent	<ul style="list-style-type: none">• Vanadium oxysulphate is a blue crystalline powder.• Vanadium is ubiquitous in the environment. It is concentrated in fatty and oily foods (e.g. milk, food oils), seafood, cereals and vegetables. Drinking water may contain trace amounts.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation – most common• Eye• Dermal
Human toxicity	<p>General</p> <ul style="list-style-type: none">• Vanadium oxysulphate inhibits a number of cellular enzymes.• Irritant to eyes, skin and mucous membranes. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Dry mouth, rhinitis, epistaxis, cough, dyspnoea, tracheitis, haemoptysis bronchitis, bronchospasm, metallic taste, greenish-black tongue and irritated eyes have been reported in workers exposed to vanadium compound dust. Occupational asthma has been reported.• Dysrhythmias and bradycardia may occur after prolonged exposure.• Pulmonary irritation leading to pulmonary oedema is a possible effect with high concentrations.• Vanadate is toxic to alveolar macrophages and may therefore impair pulmonary resistance to infection. <p>Ingestion</p> <ul style="list-style-type: none">• Abdominal cramping, nausea, vomiting and diarrhoea may occur.• CNS depression and tremor may occur, usually with large exposures.• Hepatotoxic and nephrotoxic with haematuria and albuminuria. <p>Dermal</p> <ul style="list-style-type: none">• Dermatitis and green discolouration of the skin may be noted with exposure to vanadium compounds. <p>Ocular</p> <ul style="list-style-type: none">• Irritation, conjunctivitis and corneal burns may occur.

Laboratory/radiographic testing

For minimal or mild exposures:

- Nil testing is required.

For significant exposures:

- Monitor EUC, LFTs, CMP, FBC.
- Continuous ECG monitoring.
- If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.
- Perform urinalysis.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information
131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed until asymptomatic.

Inhalation

- Provide humidified oxygen.
- Treat bronchospasm with bronchodilators.
- Support respiration as required. PEEP or CPAP may be required for pulmonary oedema.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin irritation.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

Vanadium pentoxide

Alternative names	Vanadic acid Vanadium oxide
Properties of the agent	<ul style="list-style-type: none">• Vanadium pentoxide is a pentavalent vanadium and is one of the most toxic vanadium compounds.• It is an odourless, non-combustible solid.
Routes of exposure	<ul style="list-style-type: none">• Oral• Inhalation – most common• Eye• Dermal
Human toxicity	<p>General</p> <ul style="list-style-type: none">• Vanadium pentoxide inhibits a number of cellular enzymes, including ATPase, calcium ATPase, adenylate kinase and ribonuclease.• Irritant to eyes, skin and mucous membranes. <p>Symptoms are based on route of exposure:</p> <p>Inhalation</p> <ul style="list-style-type: none">• Dry mouth, rhinitis, epistaxis, cough, dyspnoea, tracheitis, haemoptysis bronchitis, bronchospasm, metallic taste, greenish-black tongue and irritated eyes have been reported in workers exposed to vanadium compound dust. Occupational asthma has been reported.• Dysrhythmias and bradycardia may occur after prolonged exposure.• Pulmonary irritation leading to pulmonary oedema is a possible effect with high concentrations.• Vanadate is toxic to alveolar macrophages and may therefore impair pulmonary resistance to infection. <p>Ingestion</p> <ul style="list-style-type: none">• Abdominal cramping, nausea, vomiting and diarrhoea may occur.• CNS depression and tremor may occur, usually with large exposures.• Hepatotoxic and nephrotoxic with haematuria and albuminuria. <p>Dermal</p> <ul style="list-style-type: none">• Dermatitis and green discolouration of the skin may be noted with exposure to vanadium compounds. <p>Ocular</p> <ul style="list-style-type: none">• Irritation, conjunctivitis and corneal burns may occur.
Laboratory/radiographic testing	<p>For minimal or mild exposures:</p> <ul style="list-style-type: none">• Nil testing is required. <p>For significant exposures:</p> <ul style="list-style-type: none">• Monitor EUC, LFTs, CMP, FBC.• Continuous ECG monitoring.• If respiratory tract irritation or respiratory depression is evident, monitor pulse oximetry, ABGs, CXR and PFTs.• Perform urinalysis.

Treatment

See [chapter 2.1](#) for basic management principles that apply to any chemical agent.

Poisons Information

131126

Asymptomatic patients:

- See section on [patient discharge and follow-up](#).

Symptomatic patients:

General advice

- Patients with significant exposures should be admitted and observed until asymptomatic.

Inhalation

- Provide humidified oxygen.
- Treat bronchospasm with bronchodilators.
- Support respiration as required. PEEP or CPAP may be required for pulmonary oedema.

Dermal

- Flush exposed areas thoroughly.
- Provide symptomatic care of skin irritation.

Ocular

- Affected eyes should be irrigated for at least 15 minutes.
- Assess visual acuity and fluorescein uptake.
- Arrange ophthalmologic review for all patients with persistent eye symptoms following irrigation.

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Appendix 1 – Index of chemical agent synonyms

CW – Chemical Weapons

ICCA – Industrial and Commercial Chemical Agent

NLCW – Non-lethal Chemical Weapons

CHEMICAL NAME	SYNONYM	CLASSIFICATION
1,1'-Dimethyl-4,4'-bipyridinium	PARAQUAT	ICCA
1,1'-Dimethyl-4,4'-bipyridinium dichloride	PARAQUAT	ICCA
1,1'-Dimethyl-4,4'-bipyridinium dimethylsulfate	PARAQUAT	ICCA
1,1'-Dimethyl-4,4'-dipyridinium di(methyl sulphate)	PARAQUAT	ICCA
1,1'-Dimethyl-4,4'-dipyridylium chloride	PARAQUAT	ICCA
1,1-Thiobis(2-chloroethane)	MUSTARD	CW – Blister Agent
1,2,2-Trimethylpropoxyfluoromethylphosphine oxide	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
1,2,2-Trimethylpropyl methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
1,2-Dihydroxyethane	ETHYLENE GLYCOL	ICCA
1,2-Ethanediol	ETHYLENE GLYCOL	ICCA
1080	SODIUM FLUOROACETATE	ICCA
1-Azabicyclo(2.2.2)octan-3-ol, benzilate (9CI)	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
1-Chloro-2-(beta-chloroethylthio)ethane	MUSTARD	CW – Blister Agent
1-Chloro-2-di-chloroarsinoethane	LEWISITE	CW – Blister Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
1-Chloroacetophenone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
1-Methyl-2,2-dimethylpropylmethylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
1-Propene, 1,1,3,3,3-pentafluoro-2-trifluoromethyl-	PERFLUROISOBUTENE	ICCA
2,2,2-Nitrioltriethanol	TRIETHANOLAMINE	ICCA
2,2-Dichlorodiethyl sulfide	MUSTARD	CW – Blister Agent
2,2-Dichloroethyl sulfide	MUSTARD	CW – Blister Agent
2-Butanol, 3,3-dimethyl-, methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
2-Chloro-1-phenyl ethone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
2-Chloroacetophenone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
2-Chloroethenylarsonous dichloride	LEWISITE	CW – Blister Agent
2-Chloroethenyldichloroarsine	LEWISITE	CW – Blister Agent
2-Chlorovinylidichloroarsine	LEWISITE	CW – Blister Agent
2-Fluoro-1-ethanol	FLUROETHYL ALCOHOL	ICCA
2-Fluoroethanol	FLUROETHYL ALCOHOL	ICCA
2-Fluoro-ethyl alcohol	FLUROETHYL ALCOHOL	ICCA
2-Hydroxyethanol	ETHYLENE GLYCOL	ICCA
3-(2,2-Diphenyl-2-hydroxyethanoyloxy)-quinuclidine	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
3,3-Dimethyl-2-butanol methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
3,3-Dimethyl-2-butyl methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
3,3-Dimethyl-N-but-2-yl methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
3,3-Dimethyl-N-but-2-yl methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
3-Chinuclidylbenzilate	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
3-Quinuclidinol benzilate	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
3-Quinuclidinyl benzilate	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
3-Quinuclidyl benzilate	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
4,4'-Bipyridinium, 1,1'-dimethyl-	PARAQUAT	ICCA
4,4'-Bipyridinium, 1,1'-dimethyl-,bis(methyl sulphate)	PARAQUAT	ICCA
4,4'-Bipyridinium, 1,1'-dimethyl-,dichloride	PARAQUAT	ICCA
4,4'-Dimethyldipyridyl dichloride	PARAQUAT	ICCA
4-Nitro-N-phenyl	ANILINE	ICCA
A13-01804	PHENOL	ICCA
Abate	ORGANOPHOSPHATES	ICCA
Acetaldehyde	ACETALDEHYDE	ICCA
Acetic acid, fluoro-	SODIUM FLUOROACETATE	ICCA
Acetic acid, fluoro-, methyl ester	METHYL FLUOROACETATE	ICCA
Acetic aldehyde	ACETALDEHYDE	ICCA
Acide chlorhydrique	HYDROCHLORIC ACID	ICCA
100Acido chloridico	HYDROCHLORIC ACID	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Acidum salis	HYDROCHLORIC ACID	ICCA
Acquinite	CHLOROPICRIN	NLCW – Vomiting Agent
Aero liquid HCN	CYANIDE	CW – Cyanides
Agent 3	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
Agent buzz	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
Agent CX	PHOSGENE OXIME	CW – Blister Agent
Agent GA	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Agent GD	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Ah 501	PARAQUAT	ICCA
Alcohol of sulfur	CARBON DISULPHIDE	ICCA
Aldehyde (acetaldehyde)	ACETALDEHYDE	ICCA
Alpha – Chloroacetophenone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Alpha-chloroacetophenone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Aluminium phosphide	PHOSPHINE	CW – Pulmonary Agent
Aminobenzene	ANILINE	ICCA
Aminophen	ANILINE	ICCA
Ammonia	AMMONIA	ICCA
Ammonia anhydrous	AMMONIA	ICCA
Ammonia gas	AMMONIA	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Ammonium perchlorate	PERCHLORIC ACID	ICCA
Anhydrous ammonia	AMMONIA	ICCA
Anhydrous hydrofluoric acid	HYDROFLUORIC ACID	ICCA
Anilin	ANILINE	ICCA
Anilina	ANILINE	ICCA
Aniline	ANILINE	ICCA
Aniline oil	ANILINE	ICCA
Antifreeze	ETHYLENE GLYCOL	ICCA
Anyvim	ANILINE	ICCA
Aqua fortis	NITRIC ACID	ICCA
Aqueous hydrogen chloride	HYDROCHLORIC ACID	ICCA
Aqueous hydrogen fluoride	HYDROFLUORIC ACID	ICCA
Arsenic (III) oxide	ARSENIC TRIOXIDE	ICCA
Arsenic (V) oxide	ARSENIC PENTOXIDE	ICCA
Arsenic acid anhydride	ARSENIC PENTOXIDE	ICCA
Arsenic anhydride	ARSENIC PENTOXIDE	ICCA
Arsenic pentaoxide	ARSENIC PENTOXIDE	ICCA
Arsenic pentoxide, solid	ARSENIC PENTOXIDE	ICCA
Arsenic sesquioxide	ARSENIC TRIOXIDE	ICCA
Arsenic trihydride	ARSINE	ICCA
Arsenic trioxide	ARSENIC TRIOXIDE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Arsenicum album	ARSENIC TRIOXIDE	ICCA
Arsenious acid	ARSENIC TRIOXIDE	ICCA
Arsenious anhydride	ARSENIC TRIOXIDE	ICCA
Arsenious oxide	ARSENIC TRIOXIDE	ICCA
Arsenious trioxide	ARSENIC TRIOXIDE	ICCA
Arsenite	ARSENIC TRIOXIDE	ICCA
Arseniuretted hydrogen	ARSINE	ICCA
Arsenolite	ARSENIC TRIOXIDE	ICCA
Arsenous acid	ARSENIC TRIOXIDE	ICCA
Arsenous acid anhydride	ARSENIC TRIOXIDE	ICCA
Arsenous anhydride	ARSENIC TRIOXIDE	ICCA
Arsenous hydride	ARSINE	ICCA
Arsenous oxide	ARSENIC TRIOXIDE	ICCA
Arsenous oxide anhydride	ARSENIC TRIOXIDE	ICCA
Arsine	ARSINE	ICCA
Arsine, (2-chlorovinyl)dichloro-	LEWISITE	CW – Blister Agent
Arsine, dichloro(2-chlorovinyl)-	LEWISITE	CW – Blister Agent
Arsodent	ARSENIC TRIOXIDE	ICCA
Arsonous dichloride (2-chloroethenyl)-(90)	LEWISITE	CW – Blister Agent
Arsonous dichloride, (-chloroethenyl)-	LEWISITE	CW – Blister Agent
Athylenglykol	ETHYLENE GLYCOL	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Azide	SODIUM AZIDE	ICCA
Azium	SODIUM AZIDE	ICCA
Azotic acid	NITRIC ACID	ICCA
Baker's P and S liquid	PHENOL	ICCA
Baker's P and S ointment	PHENOL	ICCA
Basic copper chloride	COPPER OXYCHLORIDE	ICCA
Basic cupric chloride	COPPER OXYCHLORIDE	ICCA
Battery acid	SULPHURIC ACID	ICCA
Benzenamine	ANILINE	ICCA
Benzene, amino-	ANILINE	ICCA
Benzene, methyl-	TOLUENE	ICCA
Benzenol	PHENOL	ICCA
Benzidam	ANILINE	ICCA
Benzilic acid, 3-quinuclidinyl ester	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
Bertholite	BERYLLIUM OXIDE	ICCA
Bertholite	CHLORINE	CW – Pulmonary Agent
Beryllium difluoride	BERYLLIUM FLUORIDE	ICCA
Beryllium fluoride	BERYLLIUM FLUORIDE	ICCA
Beryllium oxide	BERYLLIUM OXIDE	ICCA
Beryllium sulphate	BERYLLIUM SULPHATE	ICCA
Beta-chlorovinylbichloroarsine	LEWISITE	CW – Blister Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Beta-fluoroethanol	FLUOROETHYL ALCOHOL	ICCA
B-Herbtox	SODIUM CHLORATE	ICCA
Bichloride of mercury	MERCURIC CHLORIDE	ICCA
Bipyridinium, 1,1'-dimethyl-4,4'-dichloride	PARAQUAT	ICCA
Bis (2-chloroethyl)sulphide	MUSTARD	CW – Blister Agent
Bisulfite	SULPHUR DIOXIDE	ICCA
Bleaching agents	HYDROCHLORIC ACID	ICCA
Blister agent	LEWISITE or MUSTARD or PHOSGENE OXIME	CW – Blister Agent
Blue copper	COPPER OXYCHLORIDE	ICCA
Blue oil	ANILINE	ICCA
Blue vitriol	COPPER SULPHATE	ICCA
Bluestone	COPPER SULPHATE	ICCA
BOV	SULPHURIC ACID	ICCA
Bromine	BROMINE	ICCA
Bromine cyanide (cyanogen bromide)	CYANIDE	CW – Cyanides
Bromocyan (cyanogen bromide)	CYANIDE	CW – Cyanides
Bromocyanide (cyanogen bromide)	CYANIDE	CW – Cyanides
Bromocyanogen (cyanogen bromide)	CYANIDE	CW – Cyanides
BZ	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
C.I. 76000	ANILINE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
C.I. oxidation base 1	ANILINE	ICCA
Caboneum sulfuratum	CARBON DISULPHIDE	ICCA
Cadmium	CADMIUM	ICCA
Calcid (calcium cyanide)	CYANIDE	CW – Cyanides
Calcium cyanide	CYANIDE	CW – Cyanides
Calcium phosphide	PHOSPHINE	CW – Pulmonary Agent
0Calcyan (calcium cyanide)	CYANIDE	CW – Cyanides
Calcyanide (calcium cyanide)	CYANIDE	CW – Cyanides
Campilit (cyanogen bromide)	CYANIDE	CW – Cyanides
Carbolic acid	PHENOL	ICCA
Carbolic oil	PHENOL	ICCA
Carbon bisulfide	CARBON DISULPHIDE	ICCA
Carbon disulfide	CARBON DISULPHIDE	ICCA
Carbon disulphide	CARBON DISULPHIDE	ICCA
Carbon hydride nitride	CYANIDE	CW – Cyanides
Carbon monoxide	CARBON MONOXIDE	ICCA
Carbon monoxide monosulfide	CARBONYL SULPHIDE	ICCA
Carbon nitride (cyanogen)	CYANIDE	CW – Cyanides
Carbon nitride ion	CYANIDE	CW – Cyanides
Carbon oxide	CARBON MONOXIDE	ICCA
Carbon oxide sulphide	CARBONYL SULPHIDE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Carbon oxychloride	PHOSGENE	CW – Pulmonary Agent
Carbon oxysulfide	CARBONYL SULPHIDE	ICCA
Carbon sulfide	CARBON DISULPHIDE	ICCA
Carbonei sulfidum	CARBON DISULPHIDE	ICCA
Carboneum sulfuratum	CARBON DISULPHIDE	ICCA
Carbonic acid dichloride	PHOSGENE	CW – Pulmonary Agent
Carbonic dichloride	PHOSGENE	CW – Pulmonary Agent
Carbonic oxide	CARBON MONOXIDE	ICCA
Carbonyl chloride	PHOSGENE	CW – Pulmonary Agent
Carbonyl sulphide	CARBONYL SULPHIDE	ICCA
Carbonyl sulphide	CARBONYL SULPHIDE	ICCA
CCRIS 3417	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Celphos (aluminium phosphide)	PHOSPHINE	CW – Pulmonary Agent
Ceresan	ETHYL MERCURY CHLORIDE	ICCA
Chamber acid	SULPHURIC ACID	ICCA
Chemical mace	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Chlorate of soda	SODIUM CHLORATE	ICCA
Chlorcyan (cyanogen chloride)	CYANIDE	CW – Cyanides
Chloric acid, sodium salt	SODIUM CHLORATE	ICCA
Chlorine	CHLORINE	CW – Pulmonary Agent
Chlorine cyanide (cyanogen chloride)	CYANIDE	CW – Cyanides

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Chlorine gas	CHLORINE	CW – Pulmonary Agent
Chloroacetophenone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Chloroacetophenone, liquid	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Chloroacetophenone, solid	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Chlorocyan (cyanogen chloride)	CYANIDE	CW – Cyanides
Chlorocyanide (cyanogen chloride)	CYANIDE	CW – Cyanides
Chlorocyanogen (cyanogen chloride)	CYANIDE	CW – Cyanides
Chloroethylmercury	ETHYL MERCURY CHLORIDE	ICCA
Chloroform, nitro-	CHLOROPICRIN	NLCW – Vomiting Agent
Chloroformyl chloride	PHOSGENE	CW – Pulmonary Agent
Chlorohydric acid	HYDROCHLORIC ACID	ICCA
Chloromethyl phenyl ketone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Chloropicrin	CHLOROPICRIN	NLCW – Vomiting Agent
Chlorovinylarsine dichloride	LEWISITE	CW – Blister Agent
Chlorowodor	HYDROCHLORIC ACID	ICCA
Chlorpyrifos	ORGANOPHOSPHATES	ICCA
Chlorwasserstoff	HYDROCHLORIC ACID	ICCA
Citrine ointment	MERCURIC NITRATE	ICCA
Claudelite	ARSENIC TRIOXIDE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Claudetite	ARSENIC TRIOXIDE	ICCA
CN	CHLOROACETOPHENONE	NLCW – Riot Control Agent
CN	CYANIDE	CW – Cyanides
CO	CARBON MONOXIDE	ICCA
Colloidal cadmium	CADMIUM	ICCA
Colloidox	COPPER OXYCHLORIDE	ICCA
Compound 1080	FLUOROACETIC ACID, NA SALT	ICCA
Copper basic sulphate	COPPER SULPHATE	ICCA
Copper chloride oxide	COPPER OXYCHLORIDE	ICCA
Copper chloride oxide, hydrate	COPPER OXYCHLORIDE	ICCA
Copper hydroxide sulfate	COPPER SULPHATE	ICCA
Copper monosulphate	COPPER SULPHATE	ICCA
Copper oxychloride	COPPER OXYCHLORIDE	ICCA
Copper sulphate	COPPER SULPHATE	ICCA
Copper sulphate, basic	COPPER SULPHATE	ICCA
Corrosive mercury chloride	MERCURIC CHLORIDE	ICCA
Corrosive sublimate	MERCURIC CHLORIDE	ICCA
COS	CARBONYL SULPHIDE	ICCA
Coumaphos	ORGANOPHOSPHATES	ICCA
Crisquat	PARAQUAT	ICCA
Crude arsenic	ARSENIC TRIOXIDE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Cruformate	ORGANOPHOSPHATES	ICCA
Cryptodine	ETHYL MERCURY CHLORIDE	ICCA
CS 4030	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
Cupric oxide chloride	COPPER OXYCHLORIDE	ICCA
Cupric oxychloride	COPPER OXYCHLORIDE	ICCA
Cupric sulfate	COPPER SULPHATE	ICCA
CX	PHOSGENE OXIME	CW – Blister Agent
Cyanide	CYANIDE	CW – Cyanides
Cyanide anion	CYANIDE	CW – Cyanides
Cyanide ion	CYANIDE	CW – Cyanides
Cyanide of potassium	CYANIDE	CW – Cyanides
Cyanide of sodium	CYANIDE	CW – Cyanides
Cyanide salts (K, Hg, Na, Ca, Zn)	CYANIDE	CW – Cyanides
Cyanobromide (cyanogen bromide)	CYANIDE	CW – Cyanides
Cyanogas (calcium cyanide)	CYANIDE	CW – Cyanides
Cyanogen	CYANIDE	CW – Cyanides
Cyanogen azide (cyanogen)	CYANIDE	CW – Cyanides
Cyanogen azide (cyanogen)	CYANIDE	CW – Cyanides
Cyanogen bromide	CYANIDE	CW – Cyanides
Cyanogen chloride	CYANIDE	CW – Cyanides
Cyanogen fluoride	CYANIDE	CW – Cyanides

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Cyanogen gas	CYANIDE	CW – Cyanides
Cyanogen iodide	CYANIDE	CW – Cyanides
Cyanogen monobromide (cyanogen bromide)	CYANIDE	CW – Cyanides
Cyanogene gas	CYANIDE	CW – Cyanides
Cyanol	ANILINE	ICCA
Cycloheximide	PHOSGENE OXIME	CW – Blister Agent
Cyclohexyl sarin	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Cyclon (hydrocyanic acid)	CYANIDE	CW – Cyanides
Cyclone B (hydrocyanic acid)	CYANIDE	CW – Cyanides
Cymag (sodium cyanide)	CYANIDE	CW – Cyanides
Delicia (aluminium phosphide)	PHOSPHINE	CW – Pulmonary Agent
Demeton	ORGANOPHOSPHATES	ICCA
Desolat	SODIUM CHLORATE	ICCA
Detia (aluminium phosphide)	PHOSPHINE	CW – Pulmonary Agent
Detia gas EX-B (aluminium phosphide)	PHOSPHINE	CW – Pulmonary Agent
Dextrone	PARAQUAT	ICCA
Dextrone-X	PARAQUAT	ICCA
Dexuron	PARAQUAT	ICCA
Di-2-chloroethyl sulphide	MUSTARD	CW – Blister Agent
Diarsenic oxide	ARSENIC TRIOXIDE	ICCA
Diarsenic pentoxide	ARSENIC PENTOXIDE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Diarsenic trioxide	ARSENIC TRIOXIDE	ICCA
Diazinon	ORGANOPHOSPHATES	ICCA
Dichloro (2-chlorovinyl) arsine	LEWISITE	CW – Blister Agent
Dichlorodiethylsulfide	MUSTARD	CW – Blister Agent
Dichloroformoxime	PHOSGENE OXIME	CW – Blister Agent
Dichlorvos	ORGANOPHOSPHATES	ICCA
Dicyan (cyanogen)	CYANIDE	CW – Cyanides
Dicyanogen (cyanogen)	CYANIDE	CW – Cyanides
Dihydrogen monosulfide	HYDROGEN SULPHIDE	ICCA
Dihydrogen sulfide	HYDROGEN SULPHIDE	ICCA
Dimethoate	ORGANOPHOSPHATES	ICCA
Dimethyl ester of sulfuric acid	DIMETHYL SULPHATE	ICCA
Dimethyl sulfate	DIMETHYL SULPHATE	ICCA
Dimethyl sulfoxide	DIMETHYL SULPHOXIDE	ICCA
Dimethyl sulphate	DIMETHYL SULPHATE	ICCA
Dimethyl sulphoxide	DIMETHYL SULPHOXIDE	ICCA
Dimethyl Viologen	PARAQUAT	ICCA
Dimethyl viologen chloride	PARAQUAT	ICCA
Dimethylamidoethoxyphosphoryl cyanide	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Dimethylaminoethocyphosphine oxide	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Dimethylphosphoramidocyanidic acid, ethyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Dioxonium perchlorate	PERCHLORIC ACID	ICCA
Dipping acid	SULPHURIC ACID	ICCA
Disodium sulphide	SODIUM SULPHIDE (HYDRATED)	ICCA
Distilled mustard	MUSTARD	CW – Blister Agent
Disulfoton	ORGANOPHOSPHATES	ICCA
Dithiocarbonic anhydride	CARBON DISULPHIDE	ICCA
DMS	DIMETHYL SULPHATE	ICCA
DMSO	DIMETHYL SULPHOXIDE	ICCA
Dojyopicrin	CHLOROPICRIN	NLCW – Vomiting Agent
Dolochlor	CHLOROPICRIN	NLCW – Vomiting Agent
EA 1195	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
EA 1208	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
EA 1210	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
EA 1701	NERVE AGENTS (DERMAL)	CW – Nerve Agent
EA 2278	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
EG	ETHYLENE GLYCOL	ICCA
EMC	ETHYL MERCURY CHLORIDE	ICCA
Emcon D	ETHYL MERCURY CHLORIDE	ICCA
Engraver's Acid	NITRIC ACID	ICCA
Esgram	PARAQUAT	ICCA
Ethanal	ACETALDEHYDE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Ethanedinitrile (cyanogen)	CYANIDE	CW – Cyanides
Ethanol, 2-fluoro	FLUOROETHYL ALCOHOL	ICCA
Ethyl aldehyde	ACETALDEHYDE	ICCA
Ethyl dimethylamidocyanophosphate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Ethyl dimethylphosphoramidocyanidate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Ethyl mercury chloride	ETHYL MERCURY CHLORIDE	ICCA
Ethyl N,N- dimethylphosphoramidocyanidate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Ethyl N,N-dimethylamino cyanophosphate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Ethyl N-dimethylphosphoramidocyanidate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Ethyl phosphorodimethylamidocyanidate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Ethyl S-2-diisopropylaminoethyl methylphosphonothiolate	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Ethyl S-diisopropylaminoethyl methylthiophosphonate	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Ethylene alcohol	ETHYLENE GLYCOL	ICCA
Ethylene dihydrate	ETHYLENE GLYCOL	ICCA
Ethylene fluorhydrin	FLUOROETHYL ALCOHOL	ICCA
Ethylene fluorhydrine	FLUOROETHYL ALCOHOL	ICCA
Ethylene glycol	ETHYLENE GLYCOL	ICCA
Ethylmercuric chloride	ETHYL MERCURY CHLORIDE	ICCA
Ethyl-N,N- dimethylphosphoramidocyanidate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Ethyl-S-dimethylaminoethyl methylphosphonothiolate	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Exhaust gas	CARBON MONOXIDE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Famphur	ORGANOPHOSPHATES	ICCA
Fenitrothion	ORGANOPHOSPHATES	ICCA
Fenosmolin	PHENOL	ICCA
Fenosmoline	PHENOL	ICCA
Fensulfothion	ORGANOPHOSPHATES	ICCA
Fenthion	ORGANOPHOSPHATES	ICCA
Fermenicide liquid	SULPHUR DIOXIDE	ICCA
Fermenicide powder	SULPHUR DIOXIDE	ICCA
Fertiliser acid	SULPHURIC ACID	ICCA
Flue gas	CARBON MONOXIDE	ICCA
Fluohydric acid gas	CYANIDE	CW – Cyanides
Fluoric acid, solution	HYDROFLUORIC ACID	ICCA
Fluoroacetate – 1080	SODIUM FLUOROACETATE	ICCA
Fluoroacetic acid, methyl ester	METHYL FLUOROACETATE	ICCA
Fluoroacetic acid, sodium salt	SODIUM FLUOROACETATE	ICCA
Fluoroethanol	FLUOROETHYL ALCOHOL	ICCA
Fluoroethyl alcohol	FLUOROETHYL ALCOHOL	ICCA
Fluorohydric acid	HYDROFLUORIC ACID	ICCA
Fluoromethyl(1,2,2-trimethylpropoxy)phosphine oxide	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Fluoromethylpinacolylxyphosphine	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Fonophos	ORGANOPHOSPHATES	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Formonitrile (hydrogen cyanide)	CYANIDE	CW – Cyanides
G 25	CHLOROPICRIN	NLCW – Vomiting Agent
GA (military designation)	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Garmoxone methyl sulphate	PARAQUAT	ICCA
Gas EX-B (aluminium phosphide)	PHOSPHINE	CW – Pulmonary Agent
GB (military designation for sarin)	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
GD	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
GE (military designation)	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Gelan I	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Gelbkreuz	MUSTARD	CW – Blister Agent
GF (military designation)	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Glycol	ETHYLENE GLYCOL	ICCA
Glycol alcohol	ETHYLENE GLYCOL	ICCA
Glycol fluorohydrin	FLUOROETHYL ALCOHOL	ICCA
Goldquat 276	PARAQUAT	ICCA
Gramixel	PARAQUAT	ICCA
Gramonol	PARAQUAT	ICCA
Gramoxon	PARAQUAT	ICCA
Gramoxone	PARAQUAT	ICCA
Gramoxone D	PARAQUAT	ICCA
Gramoxone dichloride	PARAQUAT	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Gramoxone inteon	PARAQUAT	ICCA
Gramoxone S	PARAQUAT	ICCA
Gramoxone W	PARAQUAT	ICCA
Gramuron	PARAQUAT	ICCA
Granosan	ETHYL MERCURY CHLORIDE	ICCA
H	MUSTARD	CW – Blister Agent
HCN	CYANIDE	CW – Cyanides
HD	MUSTARD	CW – Blister Agent
Herbaxon	PARAQUAT	ICCA
Herboxone	PARAQUAT	ICCA
Hexafluorosilicic acid	HYDROFLUORIC ACID	ICCA
HF	HYDROFLUORIC ACID	ICCA
HT	MUSTARD	CW – Blister Agent
Huile d'aniline	ANILINE	ICCA
Hydrochloric acid	HYDROCHLORIC ACID	ICCA
Hydrochloric acid gas	HYDROCHLORIC ACID	ICCA
Hydrochloric acid, anhydrous	HYDROCHLORIC ACID	ICCA
Hydrochloric acid, trimer	HYDROCHLORIC ACID	ICCA
Hydrochloride	HYDROCHLORIC ACID	ICCA
Hydrocyanic acid	CYANIDE	CW – Cyanides
Hydrocyanic acid, liquefied	CYANIDE	CW – Cyanides

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Hydrocyanic acid, potassium salt	CYANIDE	CW – Cyanides
Hydrocyanic acid, sodium salt	CYANIDE	CW – Cyanides
Hydrofluoric acid	HYDROFLUORIC ACID	ICCA
Hydrofluoride	HYDROFLUORIC ACID	ICCA
Hydrogen arsenide	ARSINE	ICCA
Hydrogen chloride	HYDROCHLORIC ACID	ICCA
Hydrogen chloride dimer	HYDROCHLORIC ACID	ICCA
Hydrogen chloride gas	HYDROCHLORIC ACID	ICCA
Hydrogen chloride solution	HYDROCHLORIC ACID	ICCA
Hydrogen chloride, anhydrous	HYDROCHLORIC ACID	ICCA
Hydrogen cyanide	CYANIDE	CW – Cyanides
Hydrogen cyanide gas	CYANIDE	CW – Cyanides
Hydrogen cyanide, anhydrous	CYANIDE	CW – Cyanides
Hydrogen fluoride	HYDROFLUORIC ACID	ICCA
Hydrogen nitrate	NITRIC ACID	ICCA
Hydrogen phosphide	PHOSPHINE	CW – Pulmonary Agent
Hydrogen sulfate	SULPHURIC ACID	ICCA
Hydrogen sulfide	HYDROGEN SULPHIDE	ICCA
Hydrogen sulphide	HYDROGEN SULPHIDE	ICCA
Hydrogen sulphuric acid	HYDROGEN SULPHIDE	ICCA
Hydronium perchlorate	PERCHLORIC ACID	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Hydrosulfide	HYDROGEN SULPHIDE	ICCA
Hydrosulfuric acid	HYDROGEN SULPHIDE	ICCA
Hydroxyanic acid, liquified	CYANIDE	CW – Cyanides
Hydroxybenzene	PHENOL	ICCA
IMPF	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Isobutene, octafluoro	PERFLUROISOBUTENE	ICCA
Isocyanide	CYANIDE	CW – Cyanides
Isopropoxymethylphosphonyl fluoride	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Isopropoxymethylphosphoryl fluoride	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Isopropyl methanefluorophosphonate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Isopropyl methylfluorophosphate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Isopropyl methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Isopropyl-methyl-phosphoryl fluoride	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
KLOP	CHLOROPICRIN	NLCW – Vomiting Agent
Krystallin	ANILINE	ICCA
Kyanol	ANILINE	ICCA
L	LEWISITE	CW – Blister Agent
Le-100	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Lewisite	LEWISITE	CW – Blister Agent
Mace	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Magnesium perchlorate	PERCHLORIC ACID	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Malathion	ORGANOPHOSPHATES	ICCA
Matting acid	SULPHURIC ACID	ICCA
MCE	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
MEG	ETHYLENE GLYCOL	ICCA
Mercuric chloride	MERCURIC CHLORIDE	ICCA
Mercuric nitrate	MERCURIC NITRATE	ICCA
Mercuric oxide	MERCURIC OXIDE	ICCA
Mercurous nitrate	MERCUROUS NITRATE	ICCA
Mercurous nitrate monohydrate	MERCUROUS NITRATE	ICCA
Mercury (1+), methyl-	METHYL MERCURY	ICCA
Mercury (1+), methyl ion	METHYL MERCURY	ICCA
Mercury (2+) chloride	MERCURIC CHLORIDE	ICCA
Mercury (2+) nitrate	MERCURIC NITRATE	ICCA
Mercury (2+) oxide	MERCURIC OXIDE	ICCA
Mercury (I) nitrate	MERCUROUS NITRATE	ICCA
Mercury (I) nitrate monohydrate	MERCUROUS NITRATE	ICCA
Mercury (II) chloride	MERCURIC CHLORIDE	ICCA
Mercury (II) nitrate	MERCURIC NITRATE	ICCA
Mercury (II) oxide	MERCURIC OXIDE	ICCA
Mercury (organo) alkyl compounds	ETHYL MERCURY CHLORIDE	ICCA
Mercury bichloride	MERCURIC CHLORIDE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Mercury dichloride	MERCURIC CHLORIDE	ICCA
Mercury dinitrate	MERCURIC NITRATE	ICCA
Mercury monoxide	MERCURIC OXIDE	ICCA
Mercury nitrate	MERCUROUS NITRATE	ICCA
Mercury oxide	MERCURIC OXIDE	ICCA
Mercury perchloride	MERCURIC CHLORIDE	ICCA
Mercury pernitrate	MERCURIC NITRATE	ICCA
Mercury protonitrate	MERCUROUS NITRATE	ICCA
Methacide	TOLUENE	ICCA
Methane, phenyl-	TOLUENE	ICCA
Methane, trichloronitro-	CHLOROPICRIN	NLCW – Vomiting Agent
Methyl benzene	TOLUENE	ICCA
Methyl fluoroacetate	METHYL FLUOROACETATE	ICCA
Methyl mercury	METHYL MERCURY	ICCA
Methyl pinacolyl phosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methyl pinacolyl oxy phosphoryl fluoride	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methyl sulfate	DIMETHYL SULPHATE	ICCA
Methyl sulphate	DIMETHYL SULPHATE	ICCA
Methyl sulphoxide	DIMETHYL SULPHOXIDE	ICCA
Methyl Viologen	PARAQUAT	ICCA
Methyl viologen dichloride	PARAQUAT	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Methylbenzol	TOLUENE	ICCA
Methylester kyseliny fluoroctove	METHYL FLUOROACETATE	ICCA
Methylfluorophosphonic acid, isopropyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylfluoropinacolylphosphonate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylisopropoxyfluorophosphine oxide	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylmercury (II) cation	METHYL MERCURY	ICCA
Methylmercury ion (1+)	METHYL MERCURY	ICCA
Methylphosphonofluoride acid isopropyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylphosphonofluoridic acid 1,2,2-trimethylpropyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylphosphonofluoridic acid 1-methyl-ethyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylphosphonofluoridic acid isopropyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylphosphonofluoridic acid, 3,3-dimethyl-2-butyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylphosphonothioic acid S-(2-(bis(1-methylethyl)amino)ethyl)	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Methylpinacolyloxyfluorophosphine oxide	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylpinacolyloxyphosphonyl fluoride	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Methylviologen	PARAQUAT	ICCA
Mevinphos	ORGANOPHOSPHATES	ICCA
MFA	SODIUM FLUOROACETATE	ICCA
MFI	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Millon's reagent	MERCURIC NITRATE	ICCA
Mono-chloroacetone	CHLOROACETOPHENONE	NLCW – Riot Control Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Monoethylene glycol	ETHYLENE GLYCOL	ICCA
Monoethylmercury chloride	ETHYL MERCURY CHLORIDE	ICCA
Monohydroxybenzene	PHENOL	ICCA
Monomercury nitrate	MERCUROUS NITRATE	ICCA
Monomethyl mercury	METHYL MERCURY	ICCA
Monophenol	PHENOL	ICCA
Muriatic acid	HYDROCHLORIC ACID	ICCA
Mustard	MUSTARD	CW – Blister Agent
Mustard gas	MUSTARD	CW – Blister Agent
Mustard HD	MUSTARD	CW – Blister Agent
Mustard vapour	MUSTARD	CW – Blister Agent
Mustard, sulphur	MUSTARD	CW – Blister Agent
N,N'-Dimethyl-4,4'-bipyridinium	PARAQUAT	ICCA
N,N'-Dimethyl-4,4'-dipyridinium dication	PARAQUAT	ICCA
N,N'-Dimethyl-4,4'-dipyridinium dichloride	PARAQUAT	ICCA
N,N'-Dimethyl-4,4'-dipyridylium dichloride	PARAQUAT	ICCA
N,N'-Dimethyl-4,4'-dipyridylium dichloride	PARAQUAT	ICCA
N,N'-Dimethyl-gamma,gamma'-dipyridinium	PARAQUAT	ICCA
N,N'-Dimethyl-gamma,gamma'-dipyridylium	PARAQUAT	ICCA
Sodium chlorate	SODIUM CHLORATE	ICCA
Nerve agents (dermal)	NERVE AGENTS (DERMAL)	CW – Nerve Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Nerve agents (Volatile)	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Nettle agent	PHOSGENE OXIME	CW – Blister Agent
Nettle rush gas	PHOSGENE OXIME	CW – Blister Agent
Nickel (2+) chloride	NICKEL CHLORIDE (HYDRATED)	ICCA
Nickel chloride hydrated	NICKEL CHLORIDE (HYDRATED)	ICCA
Nickel dichloride	NICKEL CHLORIDE (HYDRATED)	ICCA
Nital	NITRIC ACID	ICCA
Nitric Acid	NITRIC ACID	ICCA
Nitrilacetonitrile (cyanogen)	CYANIDE	CW – Cyanides
Nitrilo-2, 2', 2''-triethanol	TRIETHANOLAMINE	ICCA
Nitrochloroform	CHLOROPICRIN	NLCW – Vomiting Agent
Nitrotrichloromethane	CHLOROPICRIN	NLCW – Vomiting Agent
Nitrous fumes	NITRIC ACID	ICCA
Nordhausen acid	SULPHURIC ACID	ICCA
NSC-763	DIMETHYL SULPHOXIDE	ICCA
O-1,2,2-trimethylpropyl methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Octafluoroisobutylene	PERFLUOROISOBUTENE	ICCA
Octafluoro-sec-butene	PERFLUOROISOBUTENE	ICCA
O-ethyl N,N-dimethylphosphoramidocyanidate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
O-ethyl-S-(2-(diisopropylamino)ethyl) methylphosphonothioate	NERVE AGENTS (DERMAL)	CW – Nerve Agent
O-ethyl-S-(2-diisopropylaminoethyl) methylthiophosphonoate	NERVE AGENTS (DERMAL)	CW – Nerve Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
O-ethyl-S-2-diisopropylaminoethyl methylphosphonothioate	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Oil of vitriol	SULPHURIC ACID	ICCA
O-isopropyl methylisopropoxfluorophosphine oxide	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
O-isopropyl methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
OK 622	PARAQUAT	ICCA
Oleum	SULPHURIC ACID	ICCA
Omega-chloroacetophenone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
OP	ORGANOPHOSPHATES	ICCA
o-pinalcolyl methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Organic mercury	METHYL MERCURY	ICCA
Organophosphates	ORGANOPHOSPHATES	ICCA
Ortho paraquat CL	PARAQUAT	ICCA
Orthoarsenic acid	ARSENIC PENTOXIDE	ICCA
Oxalic acid dintrile (cyanogen)	CYANIDE	CW – Cyanides
Oxalonitrile (cyanogen)	CYANIDE	CW – Cyanides
Oxalyl cyanide (cyanogen)	CYANIDE	CW – Cyanides
Oxide of mercury	MERCURIC OXIDE	ICCA
Oxides of sulfur	SULPHUR DIOXIDE	ICCA
Oxybenzene	PHENOL	ICCA
Oxycarbon sulfide	CARBONYL SULPHIDE	ICCA
Oxyisobutyric nitrile	CYANIDE	CW – Cyanides

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Para-col	PARAQUAT	ICCA
Paraquat	PARAQUAT	ICCA
Paraquat bis(methyl sulphate)	PARAQUAT	ICCA
Paraquat dication	PARAQUAT	ICCA
Paraquat dimethyl sulphate	PARAQUAT	ICCA
Paraquat dimethyl sulphate	PARAQUAT	ICCA
Paraquat, bis(methylsulfate) salt	PARAQUAT	ICCA
Paraquat, dichloride salt	PARAQUAT	ICCA
Parathion	ORGANOPHOSPHATES	ICCA
Pathclear	PARAQUAT	ICCA
Pentafluoro-(2-trifluoromethyl)-propene	PERFLUOROISOBUTENE	ICCA
Perchloric acid	PERCHLORIC ACID	ICCA
Perfluoroisobutene	PERFLUOROISOBUTENE	ICCA
Perfluoroisobutylene	PERFLUOROISOBUTENE	ICCA
PFIB	PERFLUOROISOBUTENE	ICCA
PFMP	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Phenacyl chloride	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Phenic acid	PHENOL	ICCA
Phenol	PHENOL	ICCA
Phenolum	PHENOL	ICCA
Phenosmolin	PHENOL	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Phenoxyarsine, 10,10'-oxydi-	ARSINE	ICCA
Phenyl alcohol	PHENOL	ICCA
Phenyl dichloroarsine	ARSINE	ICCA
Phenyl hydrate	PHENOL	ICCA
Phenyl hydroxide	PHENOL	ICCA
Phenylamine	ANILINE	ICCA
Phenyl-chloromethyl phenone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Phenylchloromethylketone	CHLOROACETOPHENONE	NLCW – Riot Control Agent
Phenylic acid	PHENOL	ICCA
Phenylic alcohol	PHENOL	ICCA
Phenylmethane	TOLUENE	ICCA
Phorate	ORGANOPHOSPHATES	ICCA
Phosgene	PHOSGENE	CW – Pulmonary Agent
Phosgene oxime	PHOSGENE OXIME	CW – Blister Agent
Phosphine	PHOSPHINE	CW – Pulmonary Agent
Phosphine oxide, fluoroisopropoxymethyl	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Phosphine oxide, fluoromethyl(1,2,2-trimethylpropoxy)-	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Phosphonodifluoridic acid, methyl-	HYDROFLUORIC ACID	ICCA
Phosphonofluoridic acid, methyl-, 1-methylethyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Phosphonofluoridic acid, methyl-, isopropyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Phosphonofluoridic acid, methyl-, 1,2,2-trimethylpropyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Phosphonothioic acid, methyl-, S-(2-(diisopropylamino)ethyl) O-ethyl ester	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Phosphonothioic acid, methyl-, S-(2-bis(1-methylethyl)amino)ethyl) O-ethyl ester (9CI)	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Phosphoramidocyanidic acid, dimethyl-, ethyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Phosphorated hydrogen	PHOSPHINE	CW – Pulmonary Agent
Phosphoretted hydrogen	PHOSPHINE	CW – Pulmonary Agent
Phosphoric acid, methylfluoro-, isopropyl ester	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Phosphoroychloride	PHOSPHORUS OXYCHLORIDE	ICCA
Phosphorus chloride	PHOSPHORUS OXYCHLORIDE	ICCA
Phosphorus chloride oxide	PHOSPHORUS OXYCHLORIDE	ICCA
Phosphorus hydride	PHOSPHINE	CW – Pulmonary Agent
Phosphorus oxide trichloride	PHOSPHORUS OXYCHLORIDE	ICCA
Phosphorus oxychloride	PHOSPHORUS OXYCHLORIDE	ICCA
Phosphorus oxytrichloride	PHOSPHORUS OXYCHLORIDE	ICCA
Phosphorus trihydride	PHOSPHINE	CW – Pulmonary Agent
Phosphoryl chloride	PHOSPHORUS OXYCHLORIDE	ICCA
Phosphoryl trichloride	PHOSPHORUS OXYCHLORIDE	ICCA
Photophor (calcium phosphide)	PHOSPHINE	CW – Pulmonary Agent
Picfume	CHLOROPICRIN	NLCW – Vomiting Agent
Picride	CHLOROPICRIN	NLCW – Vomiting Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Pillarquat	PARAQUAT	ICCA
Pillarxone	PARAQUAT	ICCA
Pinacoloxymethylphosphoryl fluoride	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Pinacolyl methanefluorophosphonate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Pinacolyl methylfluorophosphonate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Pinacolyl methylfluorophosphonate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Pinacolyl methylphosphonefluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Pinacolyl methylphosphonofluoridate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Pinacolyl methylphosphonofluoride	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Pinacolyloxy methylphosphoryl fluoride	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Pinacolyloxymethylphosphonyl fluoride	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
PMFP	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
PnOH	PHENOL	ICCA
Potassium cyanide	CYANIDE	CW – Cyanides
Potassium cyanide, solution	CYANIDE	CW – Cyanides
Potassium perchlorate	PERCHLORIC ACID	ICCA
PP147	PARAQUAT	ICCA
PP910	PARAQUAT	ICCA
Priglone	PARAQUAT	ICCA
Profume A	CHLOROPICRIN	NLCW – Vomiting Agent
Prussic acid (hydrocyanic acid)	CYANIDE	CW – Cyanides

CHEMICAL NAME	SYNONYM	CLASSIFICATION
PS (Military designation)	CHLOROPICRIN	NLCW – Vomiting Agent
Pynacolyl methylfluorophosphonate	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
QNB	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
Ratbane	SODIUM FLUOROACETATE	ICCA
Red fuming nitric acid	NITRIC ACID	ICCA
Red oxide of mercury	MERCURIC OXIDE	ICCA
Red precipitate	MERCURIC OXIDE	ICCA
Ro 2-3308	3-QUINUCLIDINYL BENZILATE	NLCW – Incapacitating Agent
Ronnel	ORGANOPHOSPHATES	ICCA
S lost	MUSTARD	CW – Blister Agent
S mustard	MUSTARD	CW – Blister Agent
S-(2-diisopropylaminoethyl)-O-ethyl methyl phosphonothiolate	NERVE AGENTS (DERMAL)	CW – Nerve Agent
S-2((2-diisopropylamino) ethyl) O-ethyl methylphosphonothiolate	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Sarin	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Sarin II	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Schwefel-Lost	MUSTARD	CW – Blister Agent
Sewer gas	HYDROGEN SULPHIDE	ICCA
Sodium azide	SODIUM AZIDE	ICCA
Sodium chlorate	SODIUM CHLORATE	ICCA
Sodium cyanide, solid	CYANIDE	CW – Cyanides

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Sodium fluoacetic acid	SODIUM FLUOROACETATE	ICCA
Sodium fluoactetate	SODIUM FLUOROACETATE	ICCA
Sodium monofluoroacetate	SODIUM FLUOROACETATE	ICCA
Sodium monosulphide	SODIUM SULPHIDE (HYDRATED)	ICCA
Sodium perchlorate	PERCHLORIC ACID	ICCA
Sodium sulfuret	SODIUM SULPHIDE (HYDRATED)	ICCA
Sodium sulphide (hydrated)	SODIUM SULPHIDE (HYDRATED)	ICCA
Sodium sulphide anhydrous	SODIUM SULPHIDE (HYDRATED)	ICCA
Soman	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Sour gas	HYDROGEN SULPHIDE	ICCA
Spirit of Hartshorn	AMMONIA	ICCA
Spirit of sulfur	SULPHURIC ACID	ICCA
Spirits of salts	HYDROCHLORIC ACID	ICCA
SQ-9353	DIMETHYL SULPHOXIDE	ICCA
Sterolamide	TRIETHANOLAMINE	ICCA
Stink damp	HYDROGEN SULPHIDE	ICCA
Sublimate	MERCURIC CHLORIDE	ICCA
Sulfane	HYDROGEN SULPHIDE	ICCA
Sulfate dimethylique	DIMETHYL SULPHATE	ICCA
Sulfur hydride	HYDROGEN SULPHIDE	ICCA
Sulfur mustard	MUSTARD	CW – Blister Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Sulfur mustard gas	MUSTARD	CW – Blister Agent
Sulfurated hydrogen	HYDROGEN SULPHIDE	ICCA
Sulfureted hydrogen	HYDROGEN SULPHIDE	ICCA
Sulfuretted hydrogen	HYDROGEN SULPHIDE	ICCA
Sulfuric acid methyl ester	DIMETHYL SULPHATE	ICCA
Sulfuric acid, copper (2+)	COPPER SULPHATE	ICCA
Sulfuric acid, dimethyl ester	DIMETHYL SULPHATE	ICCA
Sulphur dioxide	SULPHUR DIOXIDE	ICCA
Sulphur fluoride (tetrafluoride)	HYDROFLUORIC ACID	ICCA
Sulphur hydride	HYDROGEN SULPHIDE	ICCA
Sulphur oxide	SULPHUR DIOXIDE	ICCA
Sulphuric acid	SULPHURIC ACID	ICCA
Sulphurous acid anhydride	SULPHUR DIOXIDE	ICCA
Sulphurous anhydride	SULPHUR DIOXIDE	ICCA
Sulphurous oxide	SULPHUR DIOXIDE	ICCA
Swamp gas	HYDROGEN SULPHIDE	ICCA
Sweep	PARAQUAT	ICCA
T.2107	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
T-144	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
T-2104	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
T-2106	NERVE AGENTS (VOLATILE)	CW – Nerve Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Taboon A	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Tabun	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
TEA	TRIETHANOLAMINE	ICCA
Tear gas	CHOLOROACETOPHENONE	NLCW – Riot Control Agent
Teflon	PERFLUOROISOBUTENE	ICCA
TEPP	ORGANOPHOSPHATES	ICCA
Terraklene	PARAQUAT	ICCA
Thallium sulphate	THALLIUM SULPHATE	ICCA
TL 1587	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
TL 1618	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
TL 551	METHYL FLUOROACETATE	ICCA
Toluene	TOLUENE	ICCA
Toluol	TOLUENE	ICCA
Tolu-sol	TOLUENE	ICCA
Totacol	PARAQUAT	ICCA
Total	PARAQUAT	ICCA
Toxer total	PARAQUAT	ICCA
Tri-chlor	CHLOROPICRIN	NLCW – Vomiting Agent
Trichlorfon	ORGANOPHOSPHATES	ICCA
Trichloronitromethane	CHLOROPICRIN	NLCW – Vomiting Agent
Trichlorophosphine oxide	PHOSPHORUS OXYCHLORIDE	ICCA

CHEMICAL NAME	SYNONYM	CLASSIFICATION
Trichlorophosphorus oxide	PHOSPHORUS OXYCHLORIDE	ICCA
Triethanolamine	TRIETHANOLAMINE	ICCA
Trihydroxytriethylamine	TRIETHANOLAMINE	ICCA
Trilon 83	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Trilone 46	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Trinagle	COPPER SULPHATE	ICCA
Tris(hydroxyethyl)amine	TRIETHANOLAMINE	ICCA
Trolamine	TRIETHANOLAMINE	ICCA
TX 60	NERVE AGENTS (DERMAL)	CW – Nerve Agent
V-agent	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Vanadium oxysulphate	VANADIUM OXYSULPHATE	ICCA
Vanadium pentoxide	VANADIUM PENTOXIDE	ICCA
Vanadyl sulphate	VANADIUM OXYSULPHATE	ICCA
VE (military designation)	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Vesicant	LEWISITE	CW – Blister Agent
VG (military designation)	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Viologen, methyl-	PARAQUAT	ICCA
Vitriol brown oil	SULPHURIC ACID	ICCA
VM (military designation)	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
VS (military designation)	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
VX	NERVE AGENTS (DERMAL)	CW – Nerve Agent

CHEMICAL NAME	SYNONYM	CLASSIFICATION
VX (VAN)	NERVE AGENTS (DERMAL)	CW – Nerve Agent
Weedol	PARAQUAT	ICCA
White arsenic	ARSENIC TRIOXIDE	ICCA
White fuming nitric acid	NITRIC ACID	ICCA
Yellow cross liquid	MUSTARD	CW – Blister Agent
Yellow oxide of mercury	MERCURIC OXIDE	ICCA
Yellow precipitate	MERCURIC OXIDE	ICCA
Yperite	MUSTARD	CW – Blister Agent
Zarin	NERVE AGENTS (VOLATILE)	CW – Nerve Agent
Zinc cyanide	CYANIDE	CW – Cyanides
Zinc phosphide	PHOSPHINE	CW – Pulmonary Agent
Zoman	NERVE AGENTS (VOLATILE)	CW – Nerve Agent

Appendix 2 – Useful telephone numbers

This table is provided for individual hospitals to note any important phone numbers for easy reference e.g. Local Disaster Unit, Poison's Centre, the Department of Health, On-call Toxicologists, other hospitals etc.

Name	Contact details
Poisons Information Centre	
Local Disaster Preparedness and Management Unit	
On-call Toxicologists	
Local Hospitals	
Department of Health	

Health.gov.au

All information in this publication is correct as at September 2015

