PREPARING FOR THE UNTHINKABLE
CBRN AND HAZMAT MEDICAL TRAINING
ACKNOWLEDGEMENTS

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FOREWORD

In 2017, a group of seven people, two women and five children, were admitted to Rozhawa Hospital in Iraq with symptoms of exposure to a blistering chemical agent. Their symptoms included coughing, vomiting, skin and mucous membrane irritation, blisters and bloodshot eyes. Fortunately, the International Committee of the Red Cross (ICRC) had previously trained hospital staff and equipped several clinics in Iraq to ensure that victims in such incidents could be safely decontaminated and treated. The deliberate use of hazardous material (HAZMAT) or chemical warfare agents is prohibited under international conventions and is – thankfully – not common, but it does occur.

Chemical, biological, radiological or nuclear (CBRN) incidents occur during conflicts when, for example, chemical warfare agents are used or missiles are targeted at chemical pipelines, factories or nuclear power stations. However, accidental HAZMAT releases, e.g. of toxic industrial chemicals, can also pose significant threats. For example, because of ongoing conflict, the supervisor of a factory production line may be exhausted or absent, which can lead to safety lapses with devastating consequences. In the Bhopal incident, when substandard operating and safety procedures at an understaffed plant in India resulted in the escape of methyl isocyanate, killing between 15,000 and 20,000 people and causing permanent injury to around half a million others.¹ HAZMAT incidents can happen anytime and anywhere, even in peacetime: For example, disasters can stem from natural hazards, as seen in the Fukushima reactor meltdown in 2011. The earthquake and subsequent tsunami led to the contamination of nearly eight per cent of the Japanese mainland, and the total cost of managing the disaster, estimated in 2016, was around 75.7 billion US dollars.²

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² T. Hornyak, “Clearing the radioactive rubble heap that was Fukushima Daiichi, 7 years on”, Scientific American, 9 Mar. 2018: https://www.scientificamerican.com/article/clearing-the-radioactive-rubble-heap-that-was-fukushima-daiichi-7-years-on/.
Irrespective of the root cause, managing contaminated victims is an immense challenge for first responders. To expand response capabilities, the ICRC has developed the CBRN/HAZMAT medical course as an extension of decontamination training. This handbook serves as pre-course reading material and, together with the course, presents the foundation for safely managing contaminated victims. Rather than setting out mandatory regulations, it presents examples of best practices to aid members of emergency services who will be the first to arrive at the scene of CBRN/HAZMAT incidents, as well as second responders who will care for patients during the initial hospital phases.

A note on using the handbook: The intended audience is broad, with a diverse knowledge base. To keep the text concise but still accessible, a glossary has been compiled with technical and medical terms; throughout the handbook, terms that can be found in the glossary have been emphasized and, for readers using the digital version, contain links back to their definitions.
# GLOSSARY

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<tr>
<th>Term</th>
<th>Description</th>
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<td>4-DMAP</td>
<td>4-dimethylaminophenol, an antidote to cyanide poisoning</td>
</tr>
<tr>
<td>Acetylcholine</td>
<td>A neurotransmitter (chemical messenger in the central nervous system)</td>
</tr>
<tr>
<td>Acetylcholinesterase (AChE)</td>
<td>Acetylcholinesterase, the enzyme that breaks down acetylcholine</td>
</tr>
<tr>
<td>ADR</td>
<td>European Agreement concerning the International Carriage of Dangerous Goods by Road; an ADR hazard label indicates various properties of a HAZMAT for transport purposes</td>
</tr>
<tr>
<td>AEGL</td>
<td>Acute exposure guideline limits; graded 1 to 3 depending on the level of harm caused</td>
</tr>
<tr>
<td>Aerosol</td>
<td>A fine mist of liquid or solids</td>
</tr>
<tr>
<td>ALARA</td>
<td>As low as reasonably achievable, referring to radiation exposure</td>
</tr>
<tr>
<td>ARDS</td>
<td>Adult respiratory distress syndrome</td>
</tr>
<tr>
<td>ATP</td>
<td>Adenosine triphosphate</td>
</tr>
<tr>
<td>BAL</td>
<td>British anti-lewisite, antidote to lewisite</td>
</tr>
<tr>
<td>Blister agent</td>
<td>Caustic chemical warfare agent that damages eyes, skin and lungs; also known as a vesicant</td>
</tr>
<tr>
<td>Blood agent</td>
<td>Chemical warfare agent (such as hydrogen cyanide and cyanogen chloride) that blocks the carriage of oxygen and production of ATP</td>
</tr>
<tr>
<td>BZ</td>
<td>An incapacitating agent</td>
</tr>
<tr>
<td>CBRN</td>
<td>Chemical, biological, radiological, nuclear</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
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<tr>
<td>CDA</td>
<td>Casualty decontamination area, where dangerous substances, radioactivity or germs are neutralized or removed from an area, object or person</td>
</tr>
<tr>
<td>Chemical warfare agent (CWA)</td>
<td>A toxic chemical that can be purposefully used to cause injury or death</td>
</tr>
<tr>
<td>Chemical weapon</td>
<td>Chemical Weapons&quot;means the following, together or separately:</td>
</tr>
<tr>
<td></td>
<td>(a) Toxic chemicals and their precursors, except where intended for purposes not prohibited under this Convention, as long as the types and quantities are consistent with such purposes;</td>
</tr>
<tr>
<td></td>
<td>(b) Munitions and devices, specifically designed to cause death or other harm through the toxic properties of those toxic chemicals specified in subparagraph (a), which would be released as a result of the employment of such munitions and devices;</td>
</tr>
<tr>
<td></td>
<td>(c) Any equipment specifically designed for use directly in connection with the employment of munitions and devices specified in subparagraph (b).</td>
</tr>
<tr>
<td>Choking agent</td>
<td>Chemical warfare gas that damages the lung and exposed mucosal surfaces</td>
</tr>
<tr>
<td>Cold chain</td>
<td>System for the movement of refrigerated items</td>
</tr>
<tr>
<td>Cold zone</td>
<td>Area free of contamination by hazardous material</td>
</tr>
<tr>
<td>CN</td>
<td>Chloroacetophenone, an irritant gas/lacrimator used for riot control</td>
</tr>
<tr>
<td>CR</td>
<td>Dibenzoxazepine, an irritant gas/lacrimator used for riot control</td>
</tr>
<tr>
<td>CS</td>
<td>O-chlorobenzylidene malononitrile, an irritant gas/lacrimator used for riot control</td>
</tr>
<tr>
<td>Cyclosarin</td>
<td>A nerve agent, also known as “GF”</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
<tr>
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<tr>
<td>Decontamination</td>
<td>The process of removing or deactivating toxic substances</td>
</tr>
<tr>
<td>Deterministic effect</td>
<td>An effect of radiation, the incidence and severity of which are both predicted by the dose of radiation absorbed</td>
</tr>
<tr>
<td>Doffing</td>
<td>The process of removing personal protective equipment</td>
</tr>
<tr>
<td>Donning</td>
<td>The process of putting on personal protective equipment</td>
</tr>
<tr>
<td>DU</td>
<td>Depleted uranium</td>
</tr>
<tr>
<td>EDTA</td>
<td>Ethylenediaminetetraacetic acid, a drug administered intravenously that binds and removes some metal ions</td>
</tr>
<tr>
<td>Fasciculations</td>
<td>Involuntary muscle contraction and relaxation involving fine muscle fibres</td>
</tr>
<tr>
<td>GCS</td>
<td>Glasgow Coma Scale, a commonly used system to measure a person’s level of consciousness following brain injury</td>
</tr>
<tr>
<td>Gy</td>
<td>Gray, an energy-based SI unit of ionizing radiation; 1 Gy is the amount of radiation that results in 1 J of energy being absorbed by 1 kg of matter; replaces the rad (1 Gy = 100 rad)</td>
</tr>
<tr>
<td>HAZMAT</td>
<td>Hazardous materials, any material or substance that can be damaging to health</td>
</tr>
<tr>
<td>HCN</td>
<td>Hydrogen cyanide, a blood agent</td>
</tr>
<tr>
<td>HIN</td>
<td>Hazard identification number, two to three numbers indicating various properties, e.g. flammability or radioactivity</td>
</tr>
<tr>
<td>Hot zone</td>
<td>An area contaminated with hazardous material; see also “warm zone” and “cold zone”</td>
</tr>
<tr>
<td>I-131</td>
<td>Radioactive iodine</td>
</tr>
<tr>
<td><strong>IAEA</strong></td>
<td>International Atomic Energy Agency</td>
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<tr>
<td><strong>Incapacitating agent</strong></td>
<td>A.k.a. incapacitating chemical agent (ICA); a non-lethal agent that causes confusion; best-known example is the riot-control agent BZ</td>
</tr>
<tr>
<td><strong>IDLH</strong></td>
<td>Immediately dangerous to life and health, refers to the concentration of an industrial toxic chemical</td>
</tr>
<tr>
<td><strong>Incorporation</strong></td>
<td>Process by which contaminants are imbedded in or absorbed by the human body</td>
</tr>
<tr>
<td><strong>IN</strong></td>
<td>Intranasal, intranasally</td>
</tr>
<tr>
<td><strong>IO</strong></td>
<td>Intraosseous, intraosseously</td>
</tr>
<tr>
<td><strong>IV</strong></td>
<td>Intravenous, intravenously</td>
</tr>
<tr>
<td><strong>Lacrimation</strong></td>
<td>The secretion of tears</td>
</tr>
<tr>
<td><strong>Lacrimator</strong></td>
<td>A chemical that causes irritation to the eyes and moist mucous membranes</td>
</tr>
<tr>
<td><strong>Lewisite</strong></td>
<td>A blister agent, also known as “L”</td>
</tr>
<tr>
<td><strong>Nerve agent</strong></td>
<td>Chemical warfare agent that disrupts the central nervous system by inhibiting acetylcholinesterase, an enzyme critical for nerve function</td>
</tr>
<tr>
<td><strong>NFPA</strong></td>
<td>US National Fire Processing Agency; publishes a hazard classification system for chemicals including fire diamonds, graphics indicating properties of HAZMAT</td>
</tr>
<tr>
<td><strong>Nitrogen mustard</strong></td>
<td>A blister agent; also known as “HN”</td>
</tr>
<tr>
<td><strong>Non-persistent</strong></td>
<td>Refers to a HAZMAT or CBRN agent that evaporates relatively quickly in the environment, often within hours, days or weeks</td>
</tr>
<tr>
<td><strong>OPIDN</strong></td>
<td>Organophosphorus–induced delayed neuropathy</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
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</tr>
<tr>
<td><strong>Organophosphates</strong></td>
<td>A class of chemical compounds containing phosphorus that are commonly used as insecticides, herbicides and fungicides but can also be highly toxic to humans and animals owing to their ability to inhibit acetylcholinesterase, an enzyme critical for nerve function.</td>
</tr>
<tr>
<td><strong>PEEP</strong></td>
<td>Positive end-expiratory pressure</td>
</tr>
<tr>
<td><strong>Pepper spray</strong></td>
<td>Oleoresin capsicum or “OC”, a riot-control agent</td>
</tr>
<tr>
<td><strong>Persistent</strong></td>
<td>Describing a HAZMAT or CBRN agent that remains in the environment for a long time, often years or even decades.</td>
</tr>
<tr>
<td><strong>Phosgene</strong></td>
<td>A choking agent, also known as “CG”</td>
</tr>
<tr>
<td><strong>Phosgene oxime</strong></td>
<td>A blister agent, also known as “CX”</td>
</tr>
<tr>
<td><strong>PPE</strong></td>
<td>Personal protective equipment</td>
</tr>
<tr>
<td><strong>ppm</strong></td>
<td>Parts per million, a measure of gas concentration</td>
</tr>
<tr>
<td><strong>Rad</strong></td>
<td>An energy-based unit of ionizing radiation; 1 rad is the amount of radiation that results in 0.01 J of energy being absorbed by 1 kg of matter; has been replaced by the gray in the SI unit system (1 rad = 0.01 Gy)</td>
</tr>
<tr>
<td><strong>RIID</strong></td>
<td>Radiation isotope identification device</td>
</tr>
<tr>
<td><strong>Riot-control agent</strong></td>
<td>A non-lethal agent used for dispersing crowds, also known as “RCA”</td>
</tr>
<tr>
<td><strong>Rinse-in effect</strong></td>
<td>Potentiation of symptoms or effect by washing with water</td>
</tr>
<tr>
<td><strong>Sarin</strong></td>
<td>A nerve agent; also known as “GB”</td>
</tr>
<tr>
<td><strong>Soman</strong></td>
<td>A nerve agent; also known as “GD”</td>
</tr>
<tr>
<td><strong>Stochastic</strong></td>
<td>An effect of radiation, the incidence of which is directly related to radiation dose but whose severity cannot be predicted.</td>
</tr>
<tr>
<td><strong>Sulfur mustard</strong></td>
<td>A blister agent; also known as “HD”</td>
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</tr>
<tr>
<td><strong>Sv</strong></td>
<td>Sievert; the SI unit of measure for equivalent radiation dose; used to express the radiation dose that is absorbed by a human or other living organism</td>
</tr>
<tr>
<td><strong>Tabun</strong></td>
<td>A nerve agent; also known as “GA”</td>
</tr>
<tr>
<td><strong>TIC</strong></td>
<td>Toxic industrial chemical</td>
</tr>
<tr>
<td><strong>TIM</strong></td>
<td>Toxic industrial material; can be biological, chemical or radiological</td>
</tr>
<tr>
<td><strong>Toxicity</strong></td>
<td>The degree or level of poisonous or otherwise harmful effects that a chemical, biological, radiological or nuclear agent can have on living organisms; a measure of the agent’s capacity to cause damage to human health or the environment</td>
</tr>
<tr>
<td><strong>Triage</strong></td>
<td>The process sorting casualties by the severity of their injuries</td>
</tr>
<tr>
<td><strong>UIN/UN</strong></td>
<td>Unique identification number for chemicals issued by the United Nations</td>
</tr>
<tr>
<td><strong>Vesicant</strong></td>
<td>Caustic chemical warfare agent that damages eyes, skin and lungs; also known as a blister agent</td>
</tr>
<tr>
<td><strong>Warm zone</strong></td>
<td>Area between hot and cold zones; contamination in this area is brought in by traffic from the hot zone to the cold zone</td>
</tr>
<tr>
<td><strong>VX</strong></td>
<td>A nerve agent</td>
</tr>
</tbody>
</table>
Figure 1: First-aid post on the Isonzo front line during a chemical weapon attack, World War I
1. HISTORY OF CBRN AGENTS

Descriptions of chemical warfare are almost as old as writing itself. Indian and Chinese texts report the use of toxic smoke as a weapon as far back as 2000 BC. Similar tactics were used by the Spartans against the Athenians and their allies during the Peloponnesian War (ca. 400 BC). Even Leonardo da Vinci (1452–1519) proposed a poisonous mixture of arsenic, sulfur and verdigris for use against ships.

Examples of biological attacks are equally numerous. The most famous is probably at Caffa in 1346. Infected bodies catapulted into the town are reputed to have started an outbreak of plague that decimated the defenders and broke the siege.

Most historians believe that, pre-industrialization, the fear created by these attacks was significantly greater than the damage they caused. The agents used were crude and the means of dispersal rudimentary. Although chlorine was discovered in 1774, cyanide in 1782, phosgene in 1812 and mustard agent in 1822, it was not until the 1900s that advances in chemistry and engineering enabled these highly toxic agents to be produced and delivered in any quantity. By the start of the First World War, in 1914, most of the compounds now classified as chemical warfare agents had been identified. The only exception was nerve agents, which were not discovered or produced until the 1930s, owing to the complexity of their manufacture.

The dangers posed by these weapons did not escape the notice of the international community. In 1675, France and Germany concluded an agreement banning the use of poison bullets. The 1874 International Declaration Concerning the Laws and Customs of War included a prohibition against the use of poisons and poisoned arms in warfare. This was reinforced by Hague Convention IV of 1899, which sought to ban “the use of projectiles the sole object of which is the diffusion of asphyxiating or deleterious gases”. These noble aspirations were shattered by the carnage of the First World War – 1.3 million chemical casualties, 250,000 of whom were civilians.
Figure 2: Nuclear explosion

The public outrage triggered by these horrors prompted the League of Nations to draft the Geneva Protocol of 1925, which limited the use, but not the storage and production, of chemical and biological weapons. The Geneva Protocol was strengthened in 1975 by the Biological Weapons Convention and in 1997 by the Chemical Weapons Convention, which banned the use, storage and production of these weapons. Treaties aimed at preventing the proliferation of nuclear weapons entered into force in 1970 (the Treaty on the Non-Proliferation of Nuclear Weapons) and 2021 (the Treaty on the Prohibition of Nuclear Weapons).

Despite the efforts to limit the use of these weapons, the United Nations Office for Disarmament Affairs estimates that chemical weapons alone have been responsible for more than one million casualties globally since the end of the First World War.
Figure 3: Volunteers from the Red Cross Society of the Democratic Republic of the Congo learn how to bury Ebola victims safely

Chemical, radiation and biological threats are not limited to weapons. The world is becoming more populous, industrialized and in need of power. The risks posed by chemical production and storage, nuclear power and pathogenic organisms are increasing. The task of mitigating these dangers in times of peace is difficult – in times of conflict, it becomes colossal.
2. INTRODUCTION

2.1 HAZMAT VS. CBRN AGENTS

“HAZMAT” stands for “hazardous material”, which is any material or substance that can be damaging to health. “CBRN” stands for “chemical, biological, radiological and nuclear”, sometimes with an added “E” for “explosives” (CBRNE). The term is generally used to describe agents deliberately used as a means of warfare or terrorism or for criminal ends. “HAZMAT” has traditionally been used to describe an accidental release of hazardous material, such as toxic industrial chemicals. But the line has become somewhat blurred, as HAZMAT events can occur in armed conflict when conventional weapons cause unintended (or even sometimes intended) damage to facilities resulting in the release of a hazardous agent.

2.2 HOW TO USE THIS HANDBOOK

This handbook is intended to help a static medical facility prepare for significant CBRN or HAZMAT threats. Chapters 1 to 5 cover concepts and general principles. Annexes A to H address practical and technical details.

The handbook should be used in conjunction with the ICRC’s pre-hospital decontamination guidelines.³

Medical systems, geographical features, legal requirements, resources and threats all vary enormously around the world. This handbook provides links to useful sources of information that will enable facilities to formulate solutions to CBRN and HAZMAT threats according to their unique constraints.

2.3 CLASSIFICATION OF MEDICAL FACILITIES DURING A CBRN/HAZMAT INCIDENT

Medical facilities involved in most CBRN/HAZMAT incidents can be divided into two types:
- facilities outside an area of contamination (the “clean” zone)
- facilities within an area of contamination (the “dirty” zone).

Contamination may be persistent (long-lasting) or non-persistent (transient). Persistent contamination comes from liquids, powders or radioactive particles. Pure gases and vapours tend to disperse rapidly and result in only non-persistent contamination.
A large spill of a liquid agent (e.g. the rupture of a tank containing 500 tonnes of ammonia) will produce an area of heavy liquid contamination and a downwind plume of hazardous vapour. (More information about downwind hazard distances can be found in Annex B, p. 107.)

The speed at which a chemical disperses depends on:

- the volatility and the volume of the chemical
- weather conditions (e.g. wind, ambient temperature, temperature gradient, rain)
- any additives to the chemical.

Highly volatile chemicals can be dissolved in oils or gels to prolong their duration of effect, a process known as “thickening”.

Some biological agents, e.g. anthrax spores, can also render areas hazardous for prolonged periods of time.

Medical facilities inside an area of persistent contamination cannot function without huge resources. They must be presumed inoperable until the threat clears. The priority, then, is to protect staff and patients. If possible, new patients should be directed to facilities outside the contaminated zone. It may prove necessary to relocate the staff and current patients as well.

The main principle of managing chemical and nuclear incidents is the clean/dirty line. Casualties from the hot zone are brought to a decontamination point on the line, where pollutants are cleaned off. The decontaminated patients can then be moved into a clean facility and treated. Patients from the hot zone are not treated in the clean facility without decontamination. Treatment in the hot zone should be limited to a few basic, pre-determined interventions. Medical assets should not be moved from the cold zone into the hot zone except for very specific tasks.

These concepts are discussed in more detail in Chapter 4 (p. 37).

Note that biological incidents may require a different approach.
2.4 ADAPTING TO THE TYPE OF INCIDENT

2.4.1 PLANNING
There is a vast body of literature on the management of HAZMAT incidents worldwide. Most of it originates from countries with well-equipped emergency services responding to the release of a single, known toxin. Management plans are often in place and stockpiles of monitoring equipment and antidotes available. The principles and challenges described in that literature are useful and valid. Unfortunately, the solutions proposed often require more resources than can be found in a conflict zone, where you may not know the hazard or the population at risk.

2.4.2 PERSONAL PROTECTIVE EQUIPMENT
There are many considerations when determining the appropriate personal protective equipment (PPE) for different situations. The ICRC recommends that staff working in a casualty decontamination area wear level C PPE (see Annex F for details, p. 143). This is the same level of protection that is worn by most military organizations and civilian response agencies.

Level C PPE is designed to protect the wearer against moderate concentrations of environmental contamination. In a casualty decontamination area, one would expect to find a lower level of threat because it is set up outside of the hot zone, so staff have to protect themselves against secondary contamination. This is the case for contamination from both toxic industrial chemicals and chemical warfare agents, even if chemical warfare agents are some of the most dangerous chemicals on earth.

Industrial HAZMAT incidents usually result in the release of large amounts of less toxic chemicals – often as a liquid in a small area. It is generally recommended to wear level A or B PPE in the hot zone of such an accident. Level C overalls and respirator filters may be overwhelmed by the sheer volume of chemical pollution and will not provide reliable protection. When accepting casualties outside of the hot zone, however, the risk is again only secondary contamination, so level C PPE is appropriate.
The PPE recommended in this manual will provide a good level of protection for health-care staff working in a casualty decontamination area, where the risk is only secondary contamination.

It is not designed for use at the scene of an industrial accident or for work in the hot zone beyond stabilizing and decontaminating patients at the edge of the hot zone (in the contamination reduction area) so they can enter the cold zone.

See Annex F for more details (p. 143).
3. PREPARATORY PHASE

The aim of this phase is to:

- identify key threats and needed resources
- develop plans and medical protocols to deal with identified threats
- ensure adequate resources are available to implement those plans.

3.1 SOURCES OF ADVICE AND INFORMATION


CBRN/HAZMAT incidents fall outside the experience of most medical practitioners and hospitals. It is unrealistic to expect hospital administrators to predict the casualties that would result from, say, a large chemical spill. Expert guidance is needed, and organizations and individuals who can provide such guidance should be identified.

A national CBRN/HAZMAT disaster can have serious consequences around the globe. So, resources may also be sought outside national borders, e.g. from other states or confederations.

Possible sources are:

- the host nation government (the optimal solution) – e.g. the ministry of health, state emergency services, civil defence or medical services of the ministry of defence
- national industrial, nuclear or chemical regulatory bodies
- universities, national laboratories or energy agencies
- the US Centers for Disease Control and Prevention
- the WHO
- the Organisation for the Prohibition of Chemical Weapons
- the International Atomic Energy Agency (IAEA)
- the ICRC.
The IAEA is the world's central intergovernmental forum for scientific and technical cooperation in the nuclear field. The Organisation for the Prohibition of Chemical Weapons can provide advice with regard to chemical warfare agents.

### 3.2 RISK ASSESSMENT

The principles of planning for a CBRN/HAZMAT incident are identical to those for any major incident. A few issues require serious and concerted reflection:
- What is the threat or risk?
- How many casualties could it cause and over what period?
- How many casualties can the facility actually treat?
- What other facilities are available?
- What medical protocols are to be used?
- What resources are required, and what are available?

This topic is covered in more detail in the ICRC’s [pre-hospital decontamination guidelines](#) (Section 2, on assessments, planning and management).

As previously mentioned, the process of predicting casualty numbers from a potential CBRN/HAZMAT event is a specialist skill and very foreign to most staff working in a hospital. The principles are:
1. identify a potential hazard
2. plot the geographical area implicated and calculate the population in that area
3. apply models to predict how many casualties would result depending on the prevailing winds.

Hazards can be predicted by considering:
- geographic information system (GIS) mapping
- historical events
- the availability of chemical warfare agents and toxic industrial chemicals (TICs)
- the intentions of the parties to a conflict
- the potential for unintentional TIC release.

Expert advice should be sought for these evaluations.
The areas around known hazards – such as a storage site for a known quantity of a specific TIC – can be mapped out as shown in Figure 5. Based on known lethal doses, the effects on the population in the surrounding area can be predicted. In the outermost zone, C, the population is expected to show mild, transient health effects. In zone B, there will be serious, perhaps irreversible health effects that could impair the casualties’ ability to take protective action. In the innermost zone, A, there will be life-threatening health effects.

There is software available to help with this task, such as ALOHA (Areal Locations of Hazardous Atmospheres), which is widely used to plan for and respond to chemical emergencies, and NUKEMAP, which can calculate the effects of the detonation of a nuclear bomb.

Approximate numbers are essential for logistical planning. While initial assessments of capacity and demand will almost certainly be incorrect, they can be adjusted later. An erroneous estimate is better than no estimate at all.
3.3 COMMUNICATION AND REPORTING

A system must be established to direct and coordinate the activities of neighbouring medical facilities. Areas of responsibility must be explicitly agreed in advance to minimize the risk of organizational power struggles during a crisis.

Official methods of communication should be created to enable the passage of accurate information. While personal electronic communications are now universal, even in conflict zones, they may be non-operational in some conflict situations or in the immediate aftermath of certain disasters. Without official channels, organizations are at the mercy of rumour and misinformation.

The minimum data needed for reports should also be discussed. Ideally, reporting should be carried out in a standardized electronic format to enable rapid pooling of reports about large numbers of patients from several locations.

3.4 PREPARING EMERGENCY RESPONDERS

3.4.1 TRAINING

The importance of training cannot be overemphasized.

Preparing staff to receive CBRN/HAZMAT casualties is an enormous undertaking. Significant resources and time must be allocated to it. Every staff member must have an idea of how the whole process works and how they fit into it.

Every stage of casualty movement should be rehearsed, with particular emphasis on the decontamination area. Medical protocols should be updated to include treatments for exposure to CBRN/HAZMAT agents. Biomedical technicians (those who take care of medical equipment in the health-care facility) must be instructed in the use and maintenance of any new detectors or decontamination equipment. Administrative staff must understand their roles and the decisions they may need to make. Coordination with outside organizations (e.g. civil defence) must be practised and methods of communication, established.

Ideally, a team should be selected to manage and monitor training. Every member of the medical facility should receive training appropriate to their role.
3.4.2 PROTECTION

Physical
There are many different types of PPE. Consideration must be given to the type of PPE needed based on the type and amount of hazardous agent and the tasks to be undertaken. Donning and doffing (correctly putting on and safely taking off) PPE used to protect against hazardous agents requires significant training. See Annex F for more information concerning PPE (p. 143).

Once patients have been decontaminated and enter the clean zone, staff can wear the standard PPE for the work that they are engaging in.

Immunological
Staff may need to be vaccinated against specific pathogens.

Chemical
A variety of drugs have been proposed to mitigate the effects of hazardous agents before or during exposure. Examples are:
• potassium iodide following a nuclear incident
• antibiotics for pre- or post-exposure prophylaxis pyridostigmine
pre-treatment for nerve agent exposure.

However, pre-treatment therapies are not without their problems:
• All of the medications are very hazard-specific. They should only be considered if that particular hazard is anticipated.
• It is not clear how effective many of the treatments are.
• Prophylactic treatments can cause adverse reactions in otherwise healthy people.
• Prophylaxis consumes a lot of resources for potentially little gain.
3.5 LOGISTICS

This topic is covered in more detail in the ICRC’s pre-hospital decontamination guidelines (Section 2, on assessments, planning and management).

In an ideal world, consumable and hardware requirements follow from the risk assessment and incident-response plan. In the real world, equipment constraints often determine the incident-response plan.

3.5.1 MEDICAL SUPPLIES

The quantity of drugs, oxygen, PPE and other medical supplies that the facility requires will depend on the number of casualties predicted by the risk assessment and the therapies to be used. Some antidotes to hazardous agents may already be available. An example is atropine, which is administered to treat exposure to organophosphates (p. 175) or nerve agents (p. 190). Atropine is routinely used by anaesthetists and other physicians, but the quantity required for nerve-agent poisoning is much larger than the doses used in standard clinical practice. These specific treatments should be requested well in advance of an incident to ensure appropriate quantities are in stock and ready for use.

Often, special treatments will not be available. For most CBRN/HAZMAT casualties, proper basic resuscitation is more important than specific antidotes, and patients can be managed without them.

A number of toolkits are available for predicting drug, PPE and other consumable requirements. Planning guides to help predict PPE needs can be found in the ICRC’s pre-hospital decontamination guidelines.
3.5.2 COLD CHAIN

Special consideration must be given to items that require refrigerated transport or storage. The best example of this is vaccines.

Most medical supply systems already provide for fridge items, but contingency planning is required to ensure the cold chain can survive any disruption from a CBRN or HAZMAT incident.

3.5.3 SAFE STORAGE

Consumables and medical equipment must be stored correctly to minimize the risk of damage from the usual environmental hazards. Finding suitable space or storage facilities can be difficult.

Exposure to hazardous agents can also contaminate stores of valuable medical supplies, rendering them useless. Chemical-agent-resistant materials (e.g. polypropylene or PVC) can usually be sourced in most parts of the world. Wrapping boxes in an appropriate plastic is simple and will prevent valuable supplies from having to be discarded after CBRN/HAZMAT exposure.

3.5.4 MOVEMENT OF SUPPLIES

Moving supplies is often difficult in a conflict zone owing to problems with roads and lack of vehicles. The chaos and confusion that follows the release of a hazardous agent makes this even harder. Contingency plans must be made to address any transport issues that could occur.

Possible solutions are positioning medical supplies in advance, coordinating with other organizations and requesting assistance from police or civil-defence organizations.
3.5.5 STOCK CONTROL
A medical facility needs drugs and other consumables to function. Proper management of medical stores is a vital but unglamorous task. It is often overlooked and under-resourced – much to the facility’s detriment.

Medical stock control often fails during a crisis. The extra demands placed on the system mean that:
- unusually large quantities of medical items need to be processed
- unusual items need to be ordered
- increasing storage needs may make stock control chaotic
- unusual sources of supplies can cause confusion, e.g. because of unfamiliar drug names.

Most of these problems can be solved with extra manpower and regular discussions between the medical, nursing and supply teams.

3.5.6 OXYGEN

Suggested reading: The WHO provides comprehensive advice on all aspects of oxygen use at [www.who.int/health-topics/oxygen](http://www.who.int/health-topics/oxygen).

Provision of oxygen is extremely complicated. Bottled oxygen is often unavailable. Oxygen concentrators and air compressors are more common.

The first step when addressing oxygen logistics is to make an oxygen calculation.

Estimated oxygen use will be determined by the casualty numbers predicted by the risk assessment and the agreed treatment protocols for them.

Some questions must be considered if bottled oxygen is used:
- Where can bottles be refilled and is the oxygen supplied free of impurities?
- What are the dangers of moving and storing pressurized oxygen cylinders, especially in a conflict area?
- What connectors and adaptors are needed to use oxygen cylinders?
3.6 DETECTION EQUIPMENT

Detection equipment exists for chemical, radioactive and biological hazards.

These devices have several functions:
- to warn of agent release (alarms)
- to identify agents (i.e. analysis)
- to determine how much of an agent is in the atmosphere (i.e. quantification)
- to check specific areas or objects (handheld devices)
- to measure radiation exposure (e.g. with individual dosimeters).

Detection, identification and monitoring are essential steps when responding to any event involving a hazardous agent. For emergency responders in a civilian setting, the absolute priority is to save lives while minimizing risks to their own safety.
A huge range of detectors with an array of different technologies are available commercially. There is no universal detector. All have advantages and disadvantages. The simplest detectors are paper strips that change colour when exposed to specific liquid chemical agents. As devices become more sophisticated, they also become better at detecting hazards but require maintenance and calibration that may not be available in a conflict zone.

See Annex E for more information about detection equipment (p. 133).

Operators of detection devices require training and time to get to know the equipment. Even the small, handheld detectors are complicated electrical devices that are prone to inaccurate results if not used properly.

Two examples of detectors used by the ICRC are the AP4C and the Dräger X-am 5000.

**AP4C**

![AP4C handheld detector](image)

The AP4C can detect chemical warfare agents in vapours and aerosols. It can be combined with a separate scraper unit, the SP4E, to enable sampling and detection of solid, liquid and absorbed chemicals. Particularly useful for identifying contaminated casualties and clearing patients after successful decontamination.
Dräger X-am 5000

The Dräger X-am 5000 is a portable gas-detection instrument for the continuous monitoring of the concentration of several gases in the ambient air within the working area and in explosion-hazard areas. The X-am 5000 can (depending on the device type and sensor configuration) measure up to five gases independently.

3.7 DISEASE SURVEILLANCE

Disease surveillance entails the continuous and systematic collection, analysis and interpretation of health data. Its aim is to detect disease outbreaks, such as from a biological agent, quickly, before they spread and become difficult to control.

An effective disease surveillance system is essential. See Annex C for further details (p. 113).
3.8 COORDINATING WITH POLICE AND CIVIL DEFENCE

Successfully managing a medical facility during a CBRN/HAZMAT incident involves effective communication and strict control of the flow of pedestrians and vehicles. This level of crowd control, during a period of great stress, necessitates help from the police or similar agencies. The agency that is responsible for policing must be notified well in advance to plan and rehearse their response.
4. INCIDENT MANAGEMENT

4.1 CLEAN VS. DIRTY MEDICAL FACILITIES

Medical facilities involved in CBRN incidents fall into two categories: “clean” facilities, outside the hot zone, and “dirty” facilities, inside the zone. Their roles and priorities must be considered separately.

The demands of operating inside an area contaminated with HAZMAT or CBRN agents are beyond the capabilities of most civilian medical facilities. The primary goal of a hospital unfortunate enough to be inside a contaminated zone is the protection of staff and patients.

A facility that is exposed to a transient threat (such as a cloud of gas that rapidly disperses) may be able to continue functioning once the threat has cleared.

A facility that is subjected to a more prolonged hazard (such as being located directly downwind of a damaged tank of industrial phosgene) must evacuate and relocate until the threat has passed. Upon exiting the contaminated zone, everyone must be assessed and decontaminated if necessary. In the worst-case scenario, where immediate evacuation is not feasible, the facility should be secured, and everyone should shelter in place until it becomes safe to evacuate.

Once a CBRN/HAZMAT incident has been detected, the role of a clean facility is to receive and treat casualties from contaminated and non-contaminated areas. Where and how to do this is covered in the decontamination training course.
Figure 9: Sheltering in place in the event of a CBRN/HAZMAT incident at a medical facility
4.1.1 HAZMAT/CBRN RELEASE ON A MEDICAL FACILITY: IMMEDIATE RESPONSE


• Use all available PPE.
• Reduce the number of potential casualties by ensuring that:
  – injured people are removed from the site of any chemical release, if possible, without risk to rescuers
  – uninjured people outdoors are moved upwind of any chemical release
  – uninjured people indoors remain in shelter with doors and windows shut, decontamination is done promptly where injuries from caustic or irritating agents are present or organophosphate/nerve agent poisoning is suspected.
• Decontamination is not needed if the chemical agent released is a gas.
• If decontamination is necessary, disrobe the patient, and perform improvised dry or wet emergency decontamination, as appropriate.
• Manage injuries and clinical symptoms using standard cABCD guidance for resuscitation.
• If indicated, use appropriate medical countermeasures for first-line treatment at the scene of the incident, such as auto-injectors.
• Record any treatment given on the triage tags attached to patients.
• Report the incident.

4.2 CRITERIA FOR ACTIVATING EMERGENCY PLANS

Activating emergency plans will significantly disrupt the normal functioning of the hospital and should not be undertaken lightly. Plans for receipt of HAZMAT/CBRN casualties should be activated if:
• you have been instructed to do so by appropriate authorities
• there has been a confirmed HAZMAT/CBRN release in the vicinity
• symptoms of HAZMAT/CBRN exposure have been detected in humans or animals
• reports have been received from patients indicating a HAZMAT/CBRN release. These reports may be:
  – subjective – based on smoke, vapours, colours, smells, etc.
  – objective – based on safety data sheets, colour coding of gas bottles, information from staff at site, transport papers, etc.

4.3 TRIAGE

Patients need to be triaged at two stages (at least) to determine priority for:
• decontamination (dirty triage)
• treatment/admission to the facility after decontamination (clean triage).

More information about triage is available in Annex G and the ICRC’s pre-hospital decontamination guidelines.

Figure 10: Decontamination and triage
There is no universally accepted system for triaging patients prior to decontamination.

A casualty decontamination area is made up of a series of lanes; each lane is designated for patients with a specific level of functioning (walking, on a stretcher, etc.). Patients are triaged and directed to the appropriate lane. An example of possible categories is the following:

- able to mobilize and can self-decontaminate
- able to mobilize but needs some assistance with decontamination (e.g. patient with an eye injury) or has difficulty mobilizing but cooperative (e.g. patient in a wheelchair)
- unable to mobilize and unable to cooperate (e.g. unconscious patient)
- fatally injured or dead.

Managing patients classified as dead or dying at the scene requires a high level of sensitivity and cultural awareness. The optimal approach for handling these situations will depend on the context and location. It is important to carefully consider the needs of both the patients and their families when developing strategies for managing such incidents.

Once patients have been decontaminated, a second triage is performed to determine the order in which patients are moved into the clean medical facility for treatment. See Annex G for more details on triage (p. 155).
4.4 CASUALTY DECONTAMINATION AREA

Overview of disrobing and decontamination procedure

<table>
<thead>
<tr>
<th>Warm zone</th>
<th>Cold zone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Station 1</td>
<td>Station 6</td>
</tr>
<tr>
<td>Decontamination triage</td>
<td>Re-robing</td>
</tr>
<tr>
<td>Station 2</td>
<td>Station 7</td>
</tr>
<tr>
<td>Disrobing</td>
<td>Medical triage</td>
</tr>
<tr>
<td>Station 3</td>
<td></td>
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<tr>
<td>Immediate decontamination – wet or dry</td>
<td></td>
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<tr>
<td>Station 4</td>
<td></td>
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<tr>
<td>Technical decontamination</td>
<td></td>
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<tr>
<td>Station 5</td>
<td></td>
</tr>
<tr>
<td>Drying</td>
<td></td>
</tr>
<tr>
<td>Combined Station 3 and 4 (non-ambulatory)</td>
<td></td>
</tr>
<tr>
<td>Immediate decontamination &amp; technical decontamination</td>
<td></td>
</tr>
</tbody>
</table>

Figure 11: Disrobing and decontamination procedure

Detailed instructions of how to set up and run a casualty decontamination area (CDA) can be found in the ICRC’s pre-hospital decontamination guidelines.

Decontamination entails removing residue or other toxins from the patient’s surface. This will:
- stop ongoing damage to the patient by the toxin
- ensure that the patient cannot contaminate others.

A CDA is set up in the warm zone, the junction between clean/cold and hot/dirty areas. As previously mentioned, it consists of a series of lanes, with each lane designated for patients with a specific level of functioning (walking, on a stretcher, etc.). Patients are triaged and directed to the appropriate lane. As they travel down the lane, their clothing is removed, the toxic agent is cleaned from their skin and hair. A variety of cleaning techniques are available.
Casualties who have been decontaminated do not pose a health risk to staff or other patients and can proceed into the clean zone for treatment after they have been clothed in clean clothes or single-use coveralls.

Please read the pre-hospital decontamination guidelines for a detailed description about designing and managing a CDA.

The CDA should be prepared and its use rehearsed before the first patients arrive. The size and capacity of the CDA will depend on the number of casualties expected and the rate at which they are expected to arrive.

All contaminated patients who arrive from a dirty area must be decontaminated before entering the clean area.
Casualties from a dirty area may have been decontaminated prior to arrival at the facility. A policy needs to be set on whether these casualties are to be decontaminated again or admitted directly to the clean facility.

The facility may receive casualties from contaminated and uncontaminated zones simultaneously. Ideally, the two groups are kept separate until contaminated patients have been decontaminated. (see Figure 10, p. 40).

Large numbers of survivors from the dirty zone who are uninjured but need reassurance from medical staff (the so-called worried well) may disrupt the operation of the CDA. They should be directed after triage to a survivor reception centre.

4.5 MEDICAL ASPECTS OF DECONTAMINATION

The primary goal of decontamination is to remove contaminants from casualties as quickly as possible so they may be quickly transferred and treated in a clean facility. Reports from the Tokyo sarin release, Iran–Iraq War and even the UK confirm that contaminated patients do present a significant risk to medical staff not wearing PPE. Decontamination is most important for liquid chemicals and radioactive fallout. Patients exposed to a pure toxic gas (e.g. carbon monoxide) do not require decontamination.

Medical interventions in the decontamination area should be kept to a minimum. Attempting complex medical interventions will cause organizational chaos and gridlock and delay the delivery of definitive care.

Treatment should probably be limited to:

- simple airway manoeuvres
- intramuscular administration of specific antidotes (if available)
- replacing contaminated dressings, etc., as necessary (see next).

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Patients may arrive bearing wound dressings, tourniquets or splints. For such cases, observe the following guidelines:

- It is best to keep any dressings on to avoid washing contaminant into the wounds. If a dressing needs to be removed because it is contaminated, do so carefully using aseptic technique and irrigate the wound with saline.
- If the patient suffered from catastrophic haemorrhage and has a tourniquet, it is best to try to decontaminate the tourniquet. If it must be changed because it is grossly contaminated, decontaminate the area, place a new tourniquet, and then remove the old one.
- Splints should be decontaminated but left in place where possible.

The risk posed to health-care workers by contamination in wounds is not as high as previously thought. However, if a patient has gone through decontamination and arrives for treatment with potentially contaminated dressings, splints or tourniquets in place, certain precautions must be taken. When removing potentially contaminated dressings, they should be disposed of in a 5% bleach solution. Similarly, foreign bodies or dressing material from wounds that have been packed should also be disposed of in the same solution. Any splints, instruments or linens that may have been exposed to contaminants must be soaked in a 5% bleach solution before being cleaned, sterilized or disposed of. Afterwards, wounds should be thoroughly irrigated with saline.

Prolonged irrigation of contaminated eyes may be necessary to prevent long-term damage and vision loss. Such irrigation may be facilitated by application of topical anaesthetic eye drops and the use of a specific device such as a Morgan Lens.

Butyl rubber gloves provide better protection than vinyl rubber, and both are better than latex gloves. Double gloving is recommended for any procedure involving contaminated wounds, and gloves should be changed frequently. If feasible, wear gloves of different colours so it is easy to notice any break in a glove.

The use of handheld chemical monitors (detectors) for checking wounds in the operating theatre has been found impractical, but radiation detectors is possible.
4.6 MEDICAL MANAGEMENT OF HAZMAT/CBRN INJURIES

Figure 14: Rinse with copious amounts of water

Once decontaminated, patients are treated in much the same way as any other casualty. Primary and secondary surveys should be conducted as usual, bearing in mind that the symptoms and signs of HAZMAT/CBRN toxicity may complicate diagnosis. Injuries should be managed using standard algorithms, although a few specific antidotes exist.

4.6.1 CHEMICAL AGENTS

Specific antidotes exist for some chemical warfare agents, such as nerve agents (p. 190), cyanide (p. 212), lewisite (p. 224) and opioids (p. 194). Antidotes also exist for some toxic industrial chemicals, such as hydrofluoric acid (p. 186) and hydrazines (p. 178).

4.6.2 RADIATION AND NUCLEAR AGENTS

Potassium iodide (p. 198), Prussian blue, diethylenetriamine pentaacetate (DTPA) and haemopoietic stimulants have been used to mitigate the toxicity of radiation or radioactive contaminants. Potassium iodide is effective only in the early phase, but most other specific therapies are not yet needed in that phase. This leaves time to get advice and resources from specialized facilities.

4.6.3 BIOLOGICAL PATHOGENS

Vaccination, guided antimicrobial therapy, antivirals and antitoxins are the main adjuncts to supportive care.

See Annexes A (p. 55), B (p. 83) and C (p. 113) for technical details on managing specific CBRN agents and some toxic industrial chemicals.
4.7 ORGANIZATIONAL CONSIDERATIONS

4.7.1 MIXING OF CASUALTIES
Ideally casualties arriving from a clean zone should not mix with casualties from a dirty zone until they have been decontaminated. See Figure 10, p. 40.

4.7.2 CONTAMINATION OF THE CLEAN ZONE
Movement of casualties and staff in and out of the facility should be kept to a minimum. It is almost inevitable that some contamination will get through. Ideally detectors should be used to monitor the environment in the facility for the build-up of toxins.

Handheld devices can be used to screen individuals as they leave the decontamination point or as they enter the facility. Screening reduces the risk of CBRN agents being carried into a clean facility but is laborious and disrupts movement. It is important for monitoring radiation and nuclear contamination, but it is probably impractical for chemical agents.

4.7.3 COMMUNICATION
Communication is difficult while wearing PPE, communicating with staff wearing PPE at a decontamination point even more so. A robust local solution needs to be devised that will enable communication between key areas and individuals in the complex.

Patients requiring decontamination will be scared and confused by the environment of the CDA. Staff wearing PPE appear very intimidating, and communication difficulties can rapidly lead to chaos. Patients may not understand what is happening or why they have to be decontaminated. They may resist being separated from their clothing or belongings. Explain that not cooperating will put others’ lives at risk. Use loudspeakers if available. Practical demonstrations and gestures may be useful for explaining the decontamination process. Consider adding pictures. Note that children will pose particular difficulties.

An information area or office should be established away from the main complex. This will reduce the number of relatives or other members of the public entering the facility with questions or looking for family members. When providing information be open and honest about what is known about the incident and what actions are being taken to resolve the situation.
4.7.4 CROWD CONTROL

Figure 15: Crowd control at all stages is of utmost importance to avoid cross-contamination and ensure a response that is as quick and efficient as possible.

Crowd control at all stages is of utmost importance to avoid cross-contamination and ensure that the response is as quick and efficient as possible.

Organizing the survivors of a CBRN incident will be at best difficult and at worst utter chaos. Patients and their families may attempt to enter the medical facility directly, ignoring any attempts at triage and decontamination.

Movement around the decontamination point and the medical facility must be strictly supervised. Assistance from police or a similar agency will be needed to maintain control. This should be discussed and planned well before any incident occurs.

A large number of relatives and uninjured or minimally injured people will try to congregate around the facility. They must be encouraged to leave the immediate vicinity — crowding will hamper the facility’s smooth functioning and increase the risk of clean areas becoming contaminated.

Patients may need to be evacuated to other hospitals to create space. Plans for evacuation need to be made in advance.
4.7.5 SPECIAL POPULATIONS

There are several considerations that need to be given to particular groups in a CBRN or HAZMAT incident, including people with functional impairments, elderly people and children.

Overall, it is important to consider these people’s unique needs and vulnerabilities when developing CBRN or HAZMAT incident response plans. The plans should consider the specific challenges faced by each group and ensure that appropriate measures are in place to address their needs.

People with functional impairments

Figure 16: Special considerations must be given to vulnerable groups when formulating response plans

People with reduced mobility may need specialized equipment, such as wheelchairs or stretchers. It is important to ensure that evacuation routes and decontamination lanes are accessible and that appropriate assistance is available. Those with visual or hearing impairments may require special guidance through the decontamination process.
**Elderly people**
Elderly people may have reduced mobility or cognitive impairment. Many may also be more susceptible to the effects of exposure to HAZMAT/CBRN agents owing to pre-existing medical conditions and frailty.

**Children**
Children are physiologically different from adults and can be more vulnerable to harmful materials. Their body surface area is proportionately larger, leaving them more susceptible to skin exposure, and younger children may put contaminated objects in their mouth. Their airways are smaller, which increases their risk of respiratory consequences of exposure. Their organ systems are also immature, which may make them more susceptible to the toxic effects of certain agents. Finally, children may need specific psychological support and have different communication needs than adults.

**4.7.6 CLOTHING FOR DECONTAMINATED PATIENTS**
Casualties are asked to disrobe or stripped naked during the decontamination process and provided with clean clothing upon its completion. Stores of replacement clothing will be needed.

**4.7.7 EVACUATION**
Patients may need to be evacuated to prevent a facility from being overwhelmed or to get them a higher level of care. Plans for evacuation need to be made well before any incident and key issues agreed on. The following questions must be considered:
- Who will coordinate evacuation and how?
- Which patients will be moved?
- Where will they go?
- How will they be moved?
- Who will look after them in transit?
4.8 BIOLOGICAL AGENTS

Figure 17: A live pathogen, in the form of an ebola virus particle, as seen under an electronmicroscope

Biological agents take the form of live pathogens (viral and bacterial), spores and toxins. Symptoms and signs of exposure often take time to appear and a whole community may be infected before the first case is detected.

It is not possible to decontaminate already-infected patients. Treatment is supportive and may be enhanced by vaccination and specific antimicrobial therapy or antitoxins. The priority is to prevent the agent’s spread. Casualties may need to be isolated in some way, and the best location for this may be in the contaminated zone.

Keep in mind that biological agents may also affect livestock or crops.

Figure 18: Biological agents may also affect livestock or crops. On this image, the animals did not die as a consequence of a biological agent, but is from a destocking operation.
5. POST-INCIDENT PHASE

5.1 DECONTAMINATING EQUIPMENT

A wide variety of powders, gels, oxidants, solutions and other liquids are available for decontaminating equipment. Most of these specialist items are not available in conflict zones.

The most readily available materials are hypochlorite (bleach), hydrogen peroxide and dry, adsorbent powders. See the ICRC’s pre-hospital decontamination guidelines for further recommendations.

5.2 DISPOSING OF HAZARDOUS WASTE

Laws and regulations for waste disposal are country-specific. See the pre-hospital decontamination guidelines (link above) for further recommendations.

5.3 MANAGING THE DEAD

The local systems for managing the dead will need to be modified to cover contaminated bodies. This is a very sensitive topic and must be approached in line with local laws and customs. If available, use chemical-resistant body bags to preserve dead bodies for a dignified decontamination at the end, minimizing panic for those going through decontamination first. In some locations, it may be appropriate to decontaminate only the outside of chemical-resistant body bags, enabling subsequent sampling by officials in full PPE.

More advice is available in the publication Management of Dead Bodies after Disasters: A Field Manual for First Responders (2020).
5.4 REPORTING

Reports of casualty numbers and bed occupancy are essential for directing resources between facilities. Patient registries may be needed for long-term health surveillance, e.g. to follow up on effects from radiation or long-term cancer risk after exposure to chemical agents.

Samples will be required for analysis by national or international laboratories. This is a complicated process if the incident concerns the intentional release of an agent that contravenes multiple international conventions. Definitive proof of its use may have global repercussions; hence there is a stringent process for collection and analysis by independent bodies. The Organisation for the Prohibition of Chemical Weapons has resources for sampling and analysis of chemical warfare agents.

5.5 MENTAL HEALTH

The actual or threatened use of CBRN weapons or even accidental HAZMAT exposure generates huge stress for the casualties, first responders and the community at large. Survivors of such incidents show high rates of post-traumatic stress. Appropriate plans must be devised to address it through mental health and psychosocial support.

For more information, refer to the ICRC’s Guidelines on Mental Health and Psychosocial Support (2020).

5.6 DEBRIEFING AND LESSONS LEARNED

Debriefing and lessons learned are essential components of the response to a CBRN/HAZMAT incident. After an incident, it is important to conduct a debriefing session to gather information and feedback from all those involved in the response effort. The debriefing should focus on identifying what worked well during the response and what didn’t, and highlight any areas for improvement. The lessons learned should then be documented and shared with relevant stakeholders, including response personnel, other organizations and the public. The action points and lessons learned should be used to inform future response efforts and ensure a more effective and efficient response.
INTRODUCTION

Nuclear or radiological incidents cause casualties by three main mechanisms: blast, irradiation and contamination through the spread of radioactive materials.

BLAST

Blast injuries from nuclear weapons are characterized in the same way as blast injuries from other causes (primary, secondary, tertiary, quaternary).

IRRADIATION

Pure exposure to alpha, beta, gamma or neutron radiation may be very damaging, but it does not render a person radioactive. Such patients pose no risk to emergency responders or health-care staff. The source of the radiation is the hazard, not the patients.
CONTAMINATION BY RADIOACTIVE MATERIALS

Radioactive material released into the environment can cover large areas and may irradiate anyone who comes into contact with it.

- External (non-fixed) contamination occurs when a structure, surface or patient is coated with radioactive material. Such external contamination can be removed by physical means (i.e. washing/cleaning).
- Internal contamination (incorporation) occurs when radioactive material is internalized by inhalation, ingestion or wound contamination.

Incorporation is far more dangerous to the patient than external contamination and poses more clinical challenges. External contamination is more difficult from an incident-response perspective, however, and requires an integrated response from all emergency services to prevent contamination from being spread to first responders and the public.

The relative impact of each mechanism of injury will depend on the incident’s specifics, but there are patterns:

**Predominantly irradiation**

Example: A procedural or equipment failure in a nuclear plant. Workers may be accidentally exposed to high levels of radiation without any explosions or release of radioactive material into the environment. The threat is limited to the immediate area where radiation is present, and the casualties pose no risk to rescuers or health-care workers.
**Predominantly contamination**
Examples include:
- escape of radioactive waste from a nuclear plant into the environment
- detonation of a conventional explosive device packed with radioactive materials (a so-called dirty bomb) in a heavily populated area.

**Blast, irradiation and contamination**
Example: Detonation of a nuclear weapon. Most of the energy produced by a nuclear explosion is released as blast and heat. A much smaller amount is released as radiation.

![Approximate energy release following detonation of a nuclear weapon](image)

Figure 20: Approximate energy release following detonation of a nuclear weapon

In the case of a nuclear weapon being detonated, the effect depends on the altitude at which the detonation has occurred:

**High-altitude burst**
Nuclear explosions at very high altitudes (e.g. 150,000 feet) disrupt electronic equipment and communications. The nuclear blast itself poses no direct threat to human health, but the effect on electronic equipment (an electromagnetic pulse) does.
**Air-burst**

An air-burst is an explosion where the fireball created by the detonation fails to touch the surface of the earth. The area below the detonation is subject to blast, heat and irradiation. Unless it rains, fallout (particles that are made radioactive by the effects of the explosion) is minimal, as the fission products (small, radioactive nuclei produced as by-products during the nuclear fission reactions) are dispersed over a wide area. The area directly below the blast may remain radioactive for a short period of time (owing to neutron-induced activity in the ground) but this resolves surprisingly quickly. The detonation over Hiroshima is an example of an air-burst. The area below the detonation was intensely radioactive for a period of hours but was habitable within a few days. The acute radiation injuries were caused by the initial explosion, and there was almost no fallout.

**Ground burst**

A ground burst is a detonation where the fireball touches the surface of the earth. This diminishes the area affected by blast and heat but produces significantly more downwind contamination from radioactive fallout.

**MEASURING RADIATION**

Understanding radiation and its effects is crucial to ensuring people’s safety during emergencies involving radioactive materials. Radiation affects the human body by ionizing atoms and damaging cells, which can lead to various health issues, including radiation sickness, cancer and death. The degree of exposure to radiation (the dose) is directly related to the severity of health outcomes, with higher doses increasing the risk of adverse effects. By measuring radiation levels, emergency responders can assess the potential dangers, establish evacuation zones, and implement appropriate safety measures to protect themselves and others from unnecessary exposure.
TYPES OF RADIATION

There are four types of radiation: alpha, beta, neutrons and electromagnetic waves such as gamma rays or X-rays. They each have different properties and pose different levels of risk to humans.

Alpha particles only travel short distances (three centimetres in air) and cannot penetrate the skin. That means that alpha emitters (sources of alpha particles) are only a hazard if you internalize them by ingesting them, inhaling them or getting them into a wound.

Beta particles can travel around three metres in air, but only five millimetres in tissue. They can damage the skin and can also damage cells from inside the body. Beta emitters are therefore a hazard if they get on your skin or inside your body.

Neutrons are non-charged particles and usually only arise during the fission (nuclear) process. They are highly penetrating and damaging when they interact with the body.

Electromagnetic waves such as gamma rays and X-rays can travel directly through the body, damaging cells in the skin and other organs along the way.

![Diagram of Types of Radiation and Penetration](image)

Figure 21: Types of radiation and how penetrating they are

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ANNEX A: NUCLEAR AND RADIOLOGICAL INCIDENTS
UNITS
There are two measures of radiation which are relevant to radiation-induced tissue damage: the sievert (Sv) and the gray (Gy). The gray describes the amount of energy that an object will absorb if radiation is directed at it. One gray is equal to one Joule absorbed per kilogram of mass.

Different types of radiation vary in how much damage they cause to tissue. The sievert combines the energy present in radiation (measured in grays) with how dangerous that type of radiation is (represented by the weighting factor).

\[ \text{Sievert} = \text{gray} \times \text{weighting factor} \]

<table>
<thead>
<tr>
<th>Type of radiation</th>
<th>Weighting factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gamma</td>
<td>1</td>
</tr>
<tr>
<td>Beta</td>
<td>1</td>
</tr>
<tr>
<td>Alpha</td>
<td>20</td>
</tr>
<tr>
<td>Neutron</td>
<td>2.5–5</td>
</tr>
</tbody>
</table>

Table 1: Radiation weighting factors

DETECTORS
A large range of radiation detectors are commercially available. Manufacturers use a variety of measurement technologies. Detectors fall into one of three main types:

Survey meters
These devices measure the amount of radiation in the environment at the moment of measurement. Not all devices measure all types of radiation.

Dosimeters
Dosimeters monitor accumulated radiation exposure, usually specific to the type of radiation. Only some dosimeters have a display for real-time readings.

Radiation isotope identification device (RIID)
Different radioactive materials produce unique radiation fingerprints, so to speak. RIIDs identify the radioactive materials from the radiation they emit. This is important because identifying which radioactive materials are present enables a targeted response.
Figure 22: Dosimeters can be worn on a belt, around the neck or attached to shirt

Figure 23: Example of an RIID

Make sure to choose a detector that:

• is the right type for the task at hand and has been calibrated.
• can measure radiation accurately within the necessary range of radiation energies or intensities. The necessary range for a radiation detection device depends on the specific application and the type of radiation being detected.
• is suitable for the intended location (e.g. by being portable) and that available staff know how to operate.
DOSE REFERENCE LEVELS AND RADIATION PROTECTION FOR HEALTH-CARE WORKERS

For hospital staff, it is crucial to adhere to the principle of “as low as reasonably achievable” (ALARA) when dealing with radiation. This means minimizing your exposure to radiation where possible by following the protective measures of time, distance and shielding (see below).

Things that can be done to follow the ALARA principle:
- remove all contaminated materials (maximizes distance from the source of radiation and minimizes the total amount of radiation)
- work precisely but quickly (minimizes time near the source)
- switch out personnel if possible (minimizes time near the source)
- keep your distance from patents if you’re not needed (maximizes distance from the source)
- use PPE to avoid spreading contamination further (minimizes the amount)
- use shielding if possible – put something between you and the source.\(^7\)

![Distance! Time! Shielding!](image)

Figure 24: Following the ALARA principle protects workers from unnecessary exposure to nuclear radiation

It is also important to follow the national guidelines on dose reference levels (DRLs) which specify the maximum amount of radiation exposure that is considered safe for workers in various industries, including health care. If none exist, follow those established by organizations such as the IAEA or the WHO. In general, the IAEA provides guidance on radiation protection related to nuclear energy, while the WHO focuses more broadly on radiation protection in the

context of public health. Both the IAEA and the WHO recommend an annual whole-body dose limit of 20 mSv for radiation workers (people who routinely work around sources of radiation) and 1 mSv for members of the public. For staff involved in decontamination, the annual limit is also 20 mSv, while pregnant staff should not exceed a dose of 1 mSv during their pregnancy.\(^8\) In situations where there are multiple casualties and limited resources, it may be necessary to exceed these DRLs to save lives.

**MEDICAL CONSEQUENCES OF A NUCLEAR OR RADIOLOGICAL INCIDENT**

Most injuries produced by the detonation of a nuclear bomb are from the initial intense blast wave and thermal radiation. Both effects are localized to the immediate area of the incident.

Unlike a nuclear bomb, a nuclear power plant will not explode. The fuel is only enriched to around 5% (that is, only 5% of the fuel is the specific form of uranium needed for a nuclear chain reaction). A much higher enrichment is needed for explosives. The primary danger in a nuclear power plant incident is the release of radioactive materials, causing a relatively lower number of immediate fatalities from irradiation as compared with a nuclear explosion.

Nuclear contamination and fallout from the incident may require large areas of land to be evacuated, as seen with the exclusion zone around Chernobyl, but the actual harm caused to humans long-term appears to be surprisingly modest.

Nuclear hazards are associated with the process of splitting the atom (fission). This process results in many different types of isotopes (fission products), as well as very high levels of radiation, including neutrons. Medical treatment to reduce the effects of contamination with radioactive isotopes is specific to the various isotopes, just like antidotes are specific to various toxic substances. An example is potassium iodide, which is used when radioactive iodine is released into the environment after a nuclear accident. See the [factsheet](#) for more information (p. 198).

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Most radiation incidents involve no explosion (either nuclear or conventional) but are what are referred to as “orphan source” incidents, where radiation sources are lost or stolen. These are likely to only involve one isotope. Even if radiological material is used in an explosive device to create a radiological dispersion device (RDD), often referred to as a “dirty bomb”, it is unlikely to pose a significant radiological problem. The proximity needed to an improvised RDD to cause any significant radiation dose would likely lead to death from blast injuries.

**INJURIES CAUSED BY RADIATION**

Ionizing radiation affects the human body by interacting with DNA and potentially damaging it. Whether that particular piece of damaged DNA will lead to a mutation that causes cancer is not certain. This is what is called a stochastic effect, which means it is random. However, the greater the dose of radiation, the greater the risk of cancer.

A simple analogy: If you go outside during a lightning storm, you don’t know whether you will be struck by lightning. But the more you do it, and the more you go to the mountain tops with an umbrella during the storm, the higher the likelihood that you will be struck by lightning.

At a certain high level of radiation, a threshold is passed, and the damage goes from being random to deterministic. This is because, at that very high level, the radiation causes direct cell death.

Thus, injuries caused by radiation are classified as stochastic or deterministic:

**Stochastic injury**

Health effect whose incidence but not severity is determined by the absorbed radiation dose. Cancer is an example. The chance of developing cancer is directly related to radiation exposure. The prognosis from that tumour, however, is not related to radiation dose.

---

Deterministic injury

A complication whose incidence and severity are both directly related to the absorbed radiation dose. Radiation sickness is an example. Both the chance of developing radiation sickness and the severity of symptoms can be predicted by the amount of radiation received.

HIROSHIMA BOMB

The number of people who died at Hiroshima will never be known. Estimates vary from 90,000 to 200,000. Of the deaths, 70% occurred on the day of the explosion, with the remainder occurring over the following weeks. Cases of radiation sickness began about a week after the bombing, peaked at three to four weeks and disappeared by nine weeks. Long-term follow-up has shown an increased lifetime risk of cancers.

In 1950 the Life Span Study (LSS) identified 120,000 survivors of the bombings at Hiroshima and Nagasaki for long-term follow-up. The study produces regular reviews of cancer rates and other health problems.

<table>
<thead>
<tr>
<th>Period</th>
<th>Cancer</th>
<th>Number in group</th>
<th>Cancers Observed</th>
<th>Excess over expected</th>
<th>Radiation-associated excess risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>1958–1998</td>
<td>Solid</td>
<td>44,635</td>
<td>7,851</td>
<td>848</td>
<td>10.7%</td>
</tr>
<tr>
<td>1950–2000</td>
<td>Leukaemia</td>
<td>49,204</td>
<td>204</td>
<td>94</td>
<td>46%</td>
</tr>
</tbody>
</table>

*Types of blood cancers

<table>
<thead>
<tr>
<th>Neoplasm</th>
<th>Radiation-associated excess risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute myeloid leukaemia*</td>
<td>41%</td>
</tr>
<tr>
<td>Chronic myeloid leukaemia*</td>
<td>36%</td>
</tr>
<tr>
<td>Acute lymphocytic leukaemia*</td>
<td>23%</td>
</tr>
<tr>
<td>Solid tumours</td>
<td>10%</td>
</tr>
</tbody>
</table>

Table 2: Data from the LSS register of Hiroshima and Nagasaki survivors, released by the Radiation Effects Research Foundation

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Figure 25: A seriously wounded hospital patient after the explosion of the atom bomb at Hiroshima

**CHERNOBYL REACTOR ACCIDENT**

In 1986, in Chernobyl, poorly designed experiment resulted in the worst nuclear accident in history. It was a Level-7 nuclear accident, according to the classification used by the IAEA to organize major events with widespread health and environmental effects. Fukushima is the only other accident to reach a Level 7, which is the top of this scale.\(^{11}\) The accident in Chernobyl, however, resulted in greater amounts of radioactive material being ejected into the atmosphere directly from the reactor core. 120,000 people had to be rapidly evacuated from a 30-kilometre-radius exclusion zone. In subsequent years, a further 220,000 people were moved as new areas of radioactivity were identified. Two workers were killed immediately and 237 were hospitalized. 134 of those patients developed acute radiation sickness, of whom 28 died within three months of injury.\(^{12}\)


The long-term health effects of the accident are unclear and contested. The population potentially affected by the accident is enormous. An increase in cancer rates of a few per cent would be undetectable by current epidemiological studies but produce tens of thousands of extra neoplasms (abnormal growths). Initial predictions of excess cancer deaths caused by the Chernobyl contamination vary from 5,000\(^\text{13}\) to 50,000.\(^\text{14}\) A definite increase in the incidence of thyroid cancer in the areas around Chernobyl has been detected, but the resulting death toll has been small – possibly only 15.\(^\text{15}\) As of yet, no other significant threats to health have been identified in any group other than those admitted to hospital immediately after the accident.


Figure 27: The New Safe Confinement was installed in 2016 to confine the remains of the Chernobyl plant’s number 4 reactor unit

**DIAGNOSIS AND TREATMENT OF NUCLEAR/RADIATION INJURIES**

**GENERAL CONSIDERATIONS**

The immediate life-threatening injuries resulting from a nuclear/radiation incident will be from conventional causes, such as blast, burns, fragments etc. The damage from radiation will not manifest for several hours or days. The conventional injuries are managed in the usual way using standard trauma protocols.

Figure 28: Irradiation is not the same as contamination!
Most casualties from a nuclear/radiation incident do not pose much threat to medical staff. Having been irradiated does not make a patient radioactive. Do not delay the treatment of seriously injured patients with lengthy decontamination procedures.

Operating inside an area with radioactive contamination will, however, pose a threat. The usual limit of exposure to radiation for a member of the public (including health-care workers) is 1 mSv in a year. This will limit how long first responders can be active on the scene.

**ACUTE RADIATION POISONING**
Damage from acute radiation exposure evolves through a series of well described stages. (See Table 4 at the end of this annex for an overview.)

**Radiation dose**
Exposure of at least 0.5 Sv is required to cause acute damage.

**Initial illness**
Initial symptoms develop a few hours after exposure. The speed and severity of the initial illness is a good predictor of severity. See Table 3 below for a list of initial symptoms.

A fatal prognosis is suggested by:
- symptoms less than one hour after radiation exposure
- progression from initial illness to secondary syndromes with no latent period.

**Latent period**
The initial illness usually lasts less than 48 hours. This is followed by a latent period (in which the patient has no symptoms). The latent period usually lasts days or weeks before the patient relapses into full radiation sickness. The length of the latent period is also a predictor of mortality, i.e. the shorter it is, the more severe the injury is.
Manifest illness
After the latent period, the patient relapses. Four classical syndromes have been described: cerebrovascular, gastrointestinal, haemopoietic and cutaneous.

Cerebrovascular
Cerebrovascular syndrome usually results from high-dose exposure (more than 20 Sv) but has been observed with exposures of 10 Sv upwards. Initial symptoms manifest in minutes and progress with no latent period. Patients become disorientated and ataxic and then show signs of raised intracranial pressure. This syndrome is uniformly fatal within hours or days.

Gastrointestinal
Gastrointestinal syndrome results from exposure of 6 to 30 Sv. Initial symptoms develop within about an hour and last about 48 hours. A latent period of four to six days precedes severe acute mucositis (inflammation of mucous membranes), enteritis (inflammation of intestines), necrotic gut, perforation and septic shock. Patients may die early of cerebrovascular syndrome; survivors will develop haemopoietic syndrome.

The diarrhoea of the initial illness may be due to neurohormonal factors and is not associated with overt mucosal damage.

Haemopoietic
Haemopoietic syndrome results from exposure of 1 to 6 Sv. Initial illness starts one to six hours after exposure and lasts 24 to 48 hours. A latent period of one week to one month precedes bone marrow failure. (The bone marrow produces all the cells in the blood.) A drop in lymphocyte count is detectable by 24 to 48 hours.

Neutropenia manifests at two to four weeks, thrombocytopenia at three to six weeks and anaemia thereafter. Survivors have a 50% increase in their lifetime leukaemia risk.

Cutaneous (skin)
Cutaneous syndrome may follow exposure of 2 Sv upwards. An initial illness of erythema (redness) and itchy urticaria develops minutes to hours after exposure. This lasts six to 36 hours and is followed by worsening erythema, scaling,
desquamation (flaking) and even blisters with ulceration (wounds). The skin lesions may take weeks to heal. The damaged areas often remain fragile and may suffer ongoing problems, including frequent skin neoplasms (abnormal growths).

Whole-body irradiation does not usually produce skin lesions that do not heal; these are a feature of localized radiation or radiotherapy. This is because the amount of radiation required to produce non-healing lesions would be lethal if applied to the whole body.

**MANAGEMENT OF RADIATION INJURY**

Management of radiation injury can be divided into three phases: triage, treatment, and sampling and follow-up.

1. **TRIAGE OF RADIATION INJURY**

When there are multiple casualties with radiation injuries, triage is carried out at several stages. The initial triage identifies who will get priority for decontamination (if applicable), first aid and/or transfer to a clinic or hospital. This is referred to as “dirty triage” in Figure 10 (p. 40). Serious medical problems take priority over radiological concerns. In a high-resource setting with appropriate systems in place, that means that, if a patient is triaged as red according to the medical triage system in place, their life-threatening injuries should be assessed and treated immediately, before decontamination takes place; however, this will not be the case in most places where the ICRC works.

After a patient has been initially decontaminated and stabilized, radiological triage is performed. At this stage, triage looks only at radiological injuries and is based both on clinical signs and laboratory tests.

The European Society for Blood and Marrow Transplantation (EBMT) has produced a guide to be used in the first 48 hours to clinically score patients after exposure to radiation – see Table 3, below.
Patients are divided into three groups:

- **Group 1**
  Very likely to survive. Do not need immediate admission to hospital for radiation injury. Need observation and blood tests for 30 days. May develop cutaneous or haemopoietic syndromes. Lifetime cancer risk increased by 5%.

- **Group 2**
  Very likely to develop radiation syndromes. Admit to hospital and await deterioration. Likely to develop cutaneous, alimentary or haemopoietic syndromes. Lifetime cancer risk increased by 10%. Significant mortality – especially if untreated.

- **Group 3**
  Severe exposure. Initial illness likely to develop into radiation syndromes with no latent period. Survival very unlikely.

**Combined injuries**
The mortality and morbidity of conventional injuries are significantly worsened by radiation exposure. This is most noticeable with burns. Radiation exposure of 1.5 Gy will approximately double the predicted mortality of a burn.\(^{16}\) Infection and wound healing are also adversely affected.\(^{17}\)

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16 Headquarters, US Departments of the Army, the Navy and the Airforce and Commandant, Marine Corps, *Treatment of Nuclear and Radiological Casualties*, Headquarters, Department of the Army, Falls Church, Virginia, 2001: [https://irp.fas.org/doddir/army/fm4-02-283.pdf](https://irp.fas.org/doddir/army/fm4-02-283.pdf).

<table>
<thead>
<tr>
<th>Evaluation of radiation exposure</th>
<th>First 48 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Probable dose</strong></td>
<td>Group 1</td>
</tr>
<tr>
<td>&lt; 2 Sv</td>
<td>2–20 Sv</td>
</tr>
<tr>
<td><strong>Time to onset</strong></td>
<td>of symptoms</td>
</tr>
<tr>
<td>&lt; 12 hr</td>
<td>&lt; 5 hr</td>
</tr>
<tr>
<td><strong>Erythema (redness)</strong></td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Weakness</strong></td>
<td>+</td>
</tr>
<tr>
<td><strong>Nausea</strong></td>
<td>+</td>
</tr>
<tr>
<td><strong>Vomiting</strong></td>
<td>0–1/day</td>
</tr>
<tr>
<td><strong>Diarrhoea (stools/day)</strong></td>
<td>2–3, bulky</td>
</tr>
<tr>
<td><strong>Abdominal pain</strong></td>
<td>Minimal</td>
</tr>
<tr>
<td><strong>Headache</strong></td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Temperature</strong></td>
<td>&lt; 38˚C</td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Consciousness</strong></td>
<td>Normal/no temporary loss</td>
</tr>
<tr>
<td><strong>24-hour lymphocytes</strong></td>
<td>&gt; 1.5 x 10⁹/L</td>
</tr>
<tr>
<td><strong>48-hour lymphocytes</strong></td>
<td>&gt; 1.5 x 10⁹/L</td>
</tr>
<tr>
<td><strong>Plan</strong></td>
<td>• Keep under review with regular blood tests</td>
</tr>
<tr>
<td></td>
<td>• Admission to hospital not necessary unless patient deteriorates</td>
</tr>
<tr>
<td></td>
<td>• Treat as appropriate</td>
</tr>
</tbody>
</table>

Table 3: EBMT clinical scoring guide, 2018

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2. TREATMENT OF RADIATION INJURY

Treatment can be broken down into three areas:
• decontamination
• management of radiation sickness
• management of combined conventional and radiation injuries.

DECONTAMINATION AND DECORPORATION

All patients should enter the facility through the decontamination point, as previously described. Patients may be contaminated externally (surface contamination) or internally (incorporation).

Surface decontamination
Surface contamination can be easily removed by:
• removing clothing
• washing hair
• wiping the patient down (first dry, then wet).

Treatment of severely injured patients should not be delayed by prolonged decontamination. It may be enough to remove the outermost layer of clothing or rapidly survey the patient with an appropriate detector (if available).

Internal decontamination
Incorporation, i.e. internal contamination, is more serious to the casualty than external contamination. Internal contamination happens when radioactive materials are ingested, inhaled or absorbed through the skin or enter the body via an open wound. Risk to health-care staff is small.

Wounds are decontaminated by debridement and saline irrigation. Cover the skin surrounding open wounds (with or without foreign bodies) with waterproof dressings or drapes in order to limit the spread of radioactivity from water run-off during wound irrigation. If a detector is available, continue debridement and irrigation until the reading is less than two to three times the background radiation level. Consult with medical and health physics experts before excising vital tissue.

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Certain types of radioactive materials remain in the body and may be deposited in various organs, while others are excreted from the body in blood, sweat, urine, faeces, etc.

**Decorporation therapy**
Decorporation therapy consists of medical treatments aimed at removing radioactive or toxic materials from the body after exposure. These treatments may include the use of chelating agents or other medications to promote the excretion of the radioactive or toxic material from the body.

Examples of agents used for decorporation of specific radionuclides in order to reduce their effects:
- Prussian blue aids in gut decontamination; it binds with caesium and thallium before they can enter the rest of the body. The drug is administered orally. It is not systemically absorbed and is eliminated in the faeces.
- Diethylenetriamine pentaacetic acid (DTPA) binds plutonium, americurium and curium. It is administered intravenously. Elimination is through the kidneys.
- Potassium iodide taken before exposure to radioactive iodine can reduce damage to the thyroid. See the [factsheet](#) for more information (p. 198).

![Figure 29: Potassium iodide tablets](image)
PPE
For protection against internal and external contamination, staff working at the decontamination point should wear the protective clothing recommended in the ICRC’s pre-hospital decontamination guidelines, which will consist of a respirator, a protective suit, and hand and foot protection. Note that the PPE will not protect against irradiation. If levels of irradiation are unavoidably high, it may be necessary to rotate decontamination personnel. Individual dosimeters would be useful for decontamination staff and health-care staff in contact with contaminated patients.

Staff working in the clean area should take standard precautions, i.e. wearing a surgical facemask, apron and gloves.

Radiation monitors will be very useful for checking patients entering and exiting the decontamination point, checking for radiation in the clean part of the facility and monitoring waste for signs of radioactivity.

Disposal of contaminated waste
Contaminated waste should be sealed in plastic bags and disposed of in accordance with local regulations. Incineration of radioactive waste does not reduce its radioactivity. Radioactive waste should only be burned in dedicated facilities – radioactive waste incinerators are equipped with high efficiency filters and must be well maintained.

A radiation monitor is very useful for determining whether waste is radioactive or not.
MANAGEMENT OF RADIATION SICKNESS
The management of radiation sickness is largely supportive, using standard treatment protocols. The damage to the patient occurs at the point of exposure and cannot be mitigated.

Treatment of cutaneous syndrome
The initial illness of intense urticaria and irritation may be alleviated by antihistamines and steroid cream. This phase is usually short-lived and self-limiting. The skin lesions that follow in the manifest stage of illness should be treated with standard emollients and dressings. Various specialized regimens have been suggested that can be useful following consultation with a dermatologist.

Treatment of haemopoietic syndrome
The management of this syndrome is identical to that of bone marrow failure from other causes.

The main cell lines affected are white cells and platelets.

The principles of treatment are:
• regular monitoring of blood count
• infection control
• hormonal bone marrow stimulants
• irradiated blood and blood products, including immunoglobulins
• bone marrow transplantation.

Specialist haematology advice is required.

Treatment of alimentary syndrome
Radiation produces a painful mucositis that may progress to gut necrosis, perforation and death. Fluid and electrolyte loss can be enormous. Sepsis is common, owing to gut translocation of bacteria in an immunocompromised patient.

Treatment is supportive, with analgesia and fluid resuscitation. The surgical and nutritional management of these injuries is extremely complicated and beyond the scope of this handbook.

Treatment of cerebrovascular syndrome
This syndrome suggests fatal radiation exposure. Treatment should be palliative.
MANAGEMENT OF COMBINED CONVENTIONAL AND RADIATION INJURIES

Injuries from radiation exposure can take hours or days to manifest, whereas conventional injuries require immediate intervention. Unless the casualty has received a fatal dose of radiation, management of conventional injuries takes precedence.

When the two are combined, radiation exposure complicates and significantly increases the mortality from conventional injuries.

Surgery should be avoided in patients with manifest radiation illness. Ideally, surgery should be completed before that phase.

Burns
Burns and radiation appear to have a particularly unpleasant synergistic effect. Initial burn assessment may underestimate the final damaged area, as the effects of radiation take days to manifest. Healing is slower, and grafts are not as successful. Infections are more common as well. Overall mortality is probably doubled.

Wounds
Radiation severely impairs wound healing. Surgery should be completed early, ideally within 72 hours of radiation exposure and before radiation sickness manifests. This can complicate the timing of delayed primary closures.

Amputation
Damaged limbs that would normally be salvageable may need to be amputated early to avoid operating on a patient with manifest radiation illness.

Sepsis
Patients who have been exposed to radiation are particularly susceptible to sepsis owing to mucosal damage in the gut and neutropenia.

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3. SAMPLING AND FOLLOW-UP OF RADIATION INJURY

**SAMPLING**
Faeces and urine samples can be used to confirm exposure to radionucleotides.

**LONG-TERM FOLLOW-UP**
Ideally, following an incident, everyone who has been exposed to radiation should be registered to facilitate long-term follow-up.

<table>
<thead>
<tr>
<th>Phase of syndrome</th>
<th>Feature</th>
<th>Prodrome</th>
<th>Dose range (Gy)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1–2</td>
<td>2–6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6–8</td>
<td>8–30</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt; 30</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Incidence of nausea and vomiting</td>
<td>5–50%</td>
<td>50–100%</td>
</tr>
<tr>
<td></td>
<td>Time of onset of nausea and vomiting after exposure</td>
<td>2–6 hours</td>
<td>1–2 hours</td>
</tr>
<tr>
<td></td>
<td>Duration of nausea and vomiting</td>
<td>&lt; 24 hours</td>
<td>24–48 hours</td>
</tr>
<tr>
<td></td>
<td>Severity and incidence of diarrhoea</td>
<td>None</td>
<td>None to mild (&lt; 10%)</td>
</tr>
<tr>
<td></td>
<td>Time of onset of diarrhoea after exposure</td>
<td>NA</td>
<td>3–8 hours</td>
</tr>
<tr>
<td></td>
<td>Severity and incidence of headache</td>
<td>Slight</td>
<td>Mild to moderate (50%)</td>
</tr>
<tr>
<td></td>
<td>Time of onset of headache after exposure</td>
<td>NA</td>
<td>4–24 hours</td>
</tr>
<tr>
<td></td>
<td>Severity of fever</td>
<td>Afebrile</td>
<td>Moderate increase</td>
</tr>
<tr>
<td></td>
<td>Incidence of fever</td>
<td>NA</td>
<td>10–100%</td>
</tr>
<tr>
<td>Phase of syndrome</td>
<td>Feature</td>
<td>Dose range (Gy)(^{*, **})</td>
<td>1–2</td>
</tr>
<tr>
<td>-------------------</td>
<td>-----------------------------------------------</td>
<td>-------------------------------</td>
<td>-----</td>
</tr>
<tr>
<td></td>
<td>Time of onset of fever after exposure</td>
<td></td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td>Central nervous system function</td>
<td></td>
<td>No impairment</td>
</tr>
<tr>
<td>Latent period</td>
<td>No symptoms</td>
<td></td>
<td>28–31 days</td>
</tr>
<tr>
<td>Overt illness</td>
<td>Clinical manifestations</td>
<td></td>
<td>Mild to moderate leukopenia, fatigue, weakness</td>
</tr>
<tr>
<td></td>
<td>Dominant organ system syndrome</td>
<td></td>
<td>Haematopoietic</td>
</tr>
<tr>
<td></td>
<td>Hospitalization</td>
<td></td>
<td>Outpatient observation</td>
</tr>
<tr>
<td></td>
<td>Acute mortality without medical care</td>
<td></td>
<td>0–5%</td>
</tr>
<tr>
<td></td>
<td>Acute mortality with medical care</td>
<td></td>
<td>0–5%</td>
</tr>
<tr>
<td></td>
<td>Death</td>
<td></td>
<td>6–8 weeks</td>
</tr>
</tbody>
</table>

Table 4: Effects of whole-body irradiation from external radiation or internal absorption

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1 rad = 1 cGy; 100 rad = 1 Gy.

**Whole-body irradiation of up to ~1 Gy is unlikely to cause any symptoms.**

‡ Although time to emesis is a rapid and inexpensive method for estimating radiation dose, it should be used with caution because it is imprecise and has a high false-positive rate. Additional information, such as lymphocyte counts and details of the potential for exposure, improve accuracy.


**CLASSIFICATION**

**Nerve agents**

Share their mechanism of action with carbamates and *organophosphate* insecticides ([insecticide factsheet](#), p. 175). Both block the enzyme *acetyl-cholinesterase* and disrupt nerve transmission. See [nerve agent factsheet](#) (p. 190).

**Choking agents**

Highly volatile compounds usually presenting as a vapour or gas. Attack moist membranes such as conjunctiva and respiratory mucosa. Caustic to the skin in liquid form.

**Blister agents**

Otherwise known as *vesicants* (p. 224). Primarily liquids that evaporate in warm temperatures. The liquid and vapour are both caustic to skin. Vapour also highly toxic to conjunctiva and respiratory mucosa. Absorption of these agents through lungs or skin causes systemic toxicity and organ failure.

**Blood agents**

Gases typified by *cyanide* (p. 212) and *carbon monoxide* (p. 210). Cause oxidative failure by inhibiting cytochromes.

**Riot-control agents**

Collection of less-lethal irritant or psychoactive compounds for police use.
**Toxins**
A wide range of chemicals that can be of natural origin (e.g. botulinum toxin) or synthetic origin (e.g. certain opioids, p. 194) and can cause death, injury, temporary incapacitation or sensory irritation through their chemical action.

**DEFINITIONS**

**Volutility**
How easily a substance vapourizes at a given temperature and pressure. A substance with high volatility will most often exist as a vapour, and a substance with low volatility will more often exist as a liquid or solid.

**Persistence**
The length of time an agent remains in the environment following delivery. Agents that are purely gaseous or very highly volatile will disperse rapidly. Non-volatile liquid agents will linger for much longer periods of time.

**Thickening**
Dissolving volatile agents into an oily or adhesive carrier medium to prolong their persistence. The carrier reduces the rate at which the agent evaporates and gives the agent the ability to coat structures and surfaces.
AUTONOMIC NERVOUS SYSTEM

This is the part of the nervous system that controls the involuntary functions of the body and internal organs, such as blood pressure, body temperature and gut motility. The best-known receptors in the autonomic nerves are the muscarinic and nicotinic receptors.

Figure 30: The peripheral autonomic nervous system
NERVE AGENTS AND ORGANOPHOSPHATE INSECTICIDES

Nerve agents are odourless liquids at room temperature. They persist for a period of hours to days depending on their volatility, environmental conditions and whether they have been thickened.

The traditional nerve agents are divided into G agents (e.g. tabun, sarin, cyclosarin and soman), V agents (e.g. VX) and Novichok agents.\(^{22}\) G agents are primarily designed to act via respiratory absorption. Insecticides, V agents and Novichok agents act primarily by passage through the skin or ingestion.

The symptoms and treatment of nerve agents and organophosphate insecticides are very similar, barring a few important differences that are discussed at the end of this annex.

Both act by inhibiting the enzyme acetylcholinesterase. The inhibition of the enzyme is a two-stage process: The initial bond between the agent and enzyme inactivates the active site. This stage is potentially reversible by a group of drugs known as oximes, which remove the agent from the enzyme. The second stage results from a chemical change in the agent/enzyme complex, which renders the attachment permanent. The transition from first to second stage is known as “ageing”. The ageing half-life is two minutes for soman, five hours for sarin, 14 hours for tabun and 48 hours or more for VX and insecticides.\(^{23}\)

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\(^{22}\) Novichok agents are a fourth generation of chemical warfare agents developed in the 1970s; they have higher toxicities and persistence than V agents. Very little information on these compounds is available.

It is very likely that all these compounds also have other mechanisms of toxicity which are, as yet, poorly understood.

For more information, see the factsheets on nerve agents (p. 190) and organo-phosphate insecticides (p. 175).

**SYMPTOMS OF POISONING**

Inhalation of agent can produce symptoms in seconds to minutes, transcutaneous absorption in minutes to hours. The first manifestation of poisoning is autonomic hyperactivity. The symptoms of parasympathetic overactivation appear first – thus the term “cholinergic syndrome”. This description is an oversimplification, though. Stimulation of autonomic nicotinic ganglia can also produce features of sympathetic activation (listed in the Figure 30 on p. 85).

As toxicity progresses, the casualty will become confused, convulse and lose consciousness. Death is usually due to hypoxia from disruption of central respiratory drive, muscle paralysis and secretions.

A variety of other syndromes have been described following exposure to these toxins. These syndromes are more common with organophosphates than with nerve agents and are discussed in more detail at the end of this section.

**TREATMENT OF POISONING**

Treatment can be broken down into three categories:

- prevention of ongoing toxic absorption by decontamination
- specific antidotes
- general supportive care.
AUTO-INJECTORS – FOR EMERGENCY FIELD THERAPY

Types of auto-injector and medication depend on the country in which they are used

- Pralidoxime chloride (600 mg) and atropine (2 mg)
- Pralidoxime methylsulfate (350 mg), atropine (2 mg) and avizafone chlorhydrate (20 mg)
- Obidoxime chloride (220 mg) and atropine (2 mg)
- TMB-4 (80 mg) and atropine (2 mg)
- HI-6 dimethanesulfonate (750 mg), atropine (2 mg) and diazepam (10 mg)

SPECIFIC ANTIDOTES

Atropine

For the last century, the foundation for the treatment of nerve agent poisoning has been atropine. Atropine works mainly at muscarinic receptors and has much less effect at nicotinic receptors. It can only partially protect against the effects of nerve agents but is the mainstay of treatment.

The dose of atropine required to treat nerve agent poisoning is less than the dose required to treat organophosphate poisoning. It depends on the severity of poisoning but is in the order of:

- 5 – 20 mg in 24 hours for nerve agents (may be much higher)
- 20 – 200 mg in 24 hours for insecticide poisoning.

Atropine dosing is titrated against pulse rate, secretions and subjective feeling of dyspnoea.

Topical atropine is effective in relieving severe eye pain and miosis not responding to intravenous or intramuscular atropine.
Oximes

Oximes are most effective when administered before the agent can “age”; they should be given as early as possible following exposure. The two most commonly available oximes are pralidoxime (Contrathion) and obidoxime. Oximes are ionized and do not cross the blood–brain barrier well. They appear to be more effective at nicotinic receptors than muscarinic receptors and complement the action of atropine.

Oximes are toxic, causing hypertension and cardiovascular instability. The maximum recommended pralidoxime dose for an adult is 2 g in any 60-minute period. A 24-hour infusion of pralidoxime at 0.5 g/hour may be given after an initial loading dose of 2 g. Infusions are most useful for agents which age slowly and to prevent the need for mechanical ventilation. A patient who still requires invasive ventilation after a 2 g loading dose of pralidoxime is unlikely to benefit from an infusion.

Benzodiazepines

Early use of benzodiazepines appears to significantly reduce convulsions and the morbidity associated with these agents. The choice of benzodiazepine is probably unimportant. A suggested dosing regimen for diazepam (one of the more widely available benzodiazepines), can be seen in Figure 31, on p. 88.

GENERAL SUPPORTIVE CARE

Mechanical ventilation may be required for patients with severe poisoning who fail to respond to antidote administration. The duration of ventilation is difficult to predict but will be much longer for insecticides than for nerve agents. Recovery following ventilation appears to be good, with no long-term health problems.
POISONING BY NERVE AGENTS VERSUS BY ORGANOPHOSPHATE INSECTICIDES

Many of the predictions made for nerve agent have been drawn from insecticide poisoning.

Although the two groups share a common mechanism of action, their biological effects differ. Extrapolating from one group to another has caused misconceptions around antidote dosing and likely duration of symptoms.

Absorbed dose

Nerve agent is far more potent than insecticides and is used in much smaller quantities. The doses of toxin ingested by patients poisoned with insecticide may be ten to 1,000 times higher than the amount encountered in nerve agent poisoning.

Cholinergic syndrome

The initial symptoms of poisoning (cholinergic syndrome) produced by both agents are very similar. The duration, however, is very different – hours to days for nerve agents versus days to weeks for insecticides. Insecticide poisoning is more likely to produce features of sympathetic stimulation after the initial cholinergic syndrome has settled.

Antidote dose

Compared to treatment of nerve agent poisoning, treatment of insecticide poisoning is likely to require significantly bigger doses of atropine for longer periods of time.

Intermediate syndrome

This syndrome is only seen in insecticide poisoning. Twenty-four to 96 hours following the cholinergic crisis, patients develop muscle weakness and nerve palsies, which may require ventilatory support.

Polyneuropathy: Organophosphorus-ester-induced delayed neurotoxicity (OPIDN)

OPIDN is a well-recognized syndrome involving flaccid paralysis of limbs presenting two to three weeks after exposure to some insecticides. There are no reports in humans following exposure to nerve agents. OPIDN is only seen in nerve agent animal models if 50 to 100 times the lethal dose is administered.
**Muscle necrosis**
Areas of muscle necrosis around motor end-plates can be produced by insecticides and nerve agents. Their clinical significance unclear.

**Long-term neuropsychiatric effects**
The evidence is unclear for both insecticides and nerve agents.
RIOT-CONTROL AGENTS

Riot-control agents are a highly diverse group of compounds whose function is to disable or disorientate without causing actual harm.

The Chemical Weapons Convention limits their use to training and law enforcement. Their use in war is prohibited.

The best-known agents are lacrimators, adamsite (a vomiting agent) and BZ (an incapacitating agent).

LACRIMATORS

Figure 34: Emergency first aid after tear gas exposure

- Tear gas (CN, CS and CR)
- Pepper spray (OC)

These agents cause pain without any tissue damage by stimulating specific chemical receptors on pain fibres.

All these agents are solid at room temperature and delivered as an aerosol of fine particles. Symptoms start seconds after contact and last about 20 minutes. Washing with water, soap or milk has no real effect on the symptoms of CS, CN or pepper spray but magnifies and prolongs the irritation caused by CR.
There have been reports of deaths from the use of these agents. The causes have been attributed to:

- injuries from the projectiles used to deliver the agents
- underlying health problems
- acute lung injury from inhalation of excessively high concentrations of the agent in a closed space.

**VOMITING AGENT: ADAMSITE (DM)**

DM is a yellow arsenic-containing organic compound. It is released as an odourless smoke of fine particles. It is soluble in organic solvents but not water.

Absorption is by inhalation. Symptoms start right after exposure with irritation of the eyes, lungs and mucous membranes. Malaise and persistent vomiting soon follow and persist for 12 hours.

**INCAPACITATING AGENT: BZ**

BZ is an odourless white powder with a bitter taste. It is soluble in water, acids and organic solvents. Following release, it persists in the environment for several days.

It is absorbed by inhalation and is a powerful centrally and peripherally acting muscarinic antagonist. Symptoms of exposure include confusion, dilated pupils, hyperthermia, tachycardia and dry mouth and can persist for 12 to 24 hours.

Physostigmine has been tested as a useful antidote.

**VESICANTS**

Vesicants are compounds that cause cutaneous chemical burns (vesicles). They are oily liquids which can produce a significant vapour threat, especially in warm climates.

Three main agents make up this group:

- mustard agents (in particular, sulfur mustard; nitrogen mustard has never been deployed)
- lewisite
- phosgene oxime.
All these compounds are highly reactive and cause damage to intracellular proteins and DNA.

For more information, see the factsheet on p. 224.

Vesicants are rapidly inactivated by contact with tissue.

Fluid in blisters and contaminated skin do not pose a risk to health workers.

Sulfur mustard has been used extensively since 1915, and its effects have been well described. Information on the other two agents is far more limited.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Skin lesions</th>
<th>Onset of lesions</th>
<th>Pain</th>
<th>Smell</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfur mustard</td>
<td>Thin-walled blisters</td>
<td>2–4 hours</td>
<td>Develops as blisters develop</td>
<td>Garlic</td>
</tr>
<tr>
<td>Lewisite</td>
<td>Thin-walled blisters</td>
<td>1–4 hours</td>
<td>Immediate pain that improves as blisters develop</td>
<td>Geraniums</td>
</tr>
<tr>
<td>Phosgene oxime</td>
<td>Solid urticarial wheels</td>
<td>1 hour or less</td>
<td>Immediate severe pain</td>
<td>Bitter or burning</td>
</tr>
</tbody>
</table>

Table 5: Comparison of vesicants

**SULFUR MUSTARD**

Sulfur mustard is an oily liquid that vapourizes quickly at warm temperatures. In colder climates, it is, however, very persistent. Its melting point is 14.45°C, meaning it is solid below that temperature. At 38°C, its persistence is only seven hours. Vapour, rather than liquid, causes 80% of reported casualties. For more information, see factsheet on p. 224.

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Toxicity
Symptoms develop two to four hours following exposure. 80 to 90% of patients develop conjunctivitis, 80 to 90% develop skin lesions and 75% have some sort of respiratory involvement. Systemic absorption can affect the gut, haemopoiesis and the central nervous system. Overall mortality is 2 to 3%, usually from respiratory failure.

Eyes

Vapour produces mild or moderate conjunctivitis that resolves over one to five weeks. Liquid contamination can produce extensive scarring and loss of vision but is much less common. Initial treatment of both consists of:

- daily irrigation
- topical antibiotics
- topical mydriatics to prevent formation of synechiae
- topical steroids
- lubricating eyedrops to prevent eyelids from adhering to each other.

Skin
Vapour exposure produces lesions that resemble superficial or partial-thickness burns. Moist areas of skin and mucosa surfaces are affected first. Mild cases resemble sunburn. Vesicles appear one to two hours after the onset of symptoms. Liquid contamination produces deeper lesions that resemble full-thickness burns. Blister fluid and contaminated skin do not retain any active agent and are not a threat to health workers. Transient hyperpigmentation of affected areas is often observed as the lesions start to heal.
Treatment is supportive and modelled on burns management, with a few differences:

- Care should be taken with initial fluid resuscitation, as fluid loss in patients exposed to vesicants is lower than in patients with thermal burns. Care should be taken with initial fluid resuscitation, as fluid loss in patients exposed to vesicants is lower than in patients with thermal burns. Standard regimes such as the Parkland Formula tend to overestimate fluid requirements and have been associated with volume overload, especially in the context of respiratory injury.
- The optimal surgical management of these lesions is very slightly different to thermal burns. Detailed description is beyond the scope of this handbook.

**Respiratory**

Inhalation of vapour can affect the entire respiratory tree:

- Oedema and spasm of the larynx have been associated with acute airway obstruction.
- Acute lung injury can develop into acute respiratory distress syndrome (ARDS).
- Damage to the bronchial tree can cause mucosal sloughing, progressing to strictures three to 12 months after the initial exposure.

---


Standard intensive care management is recommended for these injuries. Discussion of the specific protocols is beyond the scope of this handbook.

**Systemic absorption**
Systemic absorption of mustard agent results in:
- **bone marrow failure** not dissimilar to that seen in radiation poisoning and **lymphopaenia**. Lymphopaenia develops 24 hours after exposure, with bone marrow failure developing one to three weeks later. Treatment is highly specialized and may require antibiotics, transfusions, infection control and hormonal marrow stimulation.
- **haemorrhagic diarrhoea** caused by mucosal damage. Treatment is supportive with fluid and analgesics.
- **central nervous system toxicity**. Confusion and fits have been described following acute exposure to mustard agent. A variety of neurological syndromes and peripheral neuropathy may follow.

**Long-term damage**
The following long-term complications have been described:
- increased risk of cancer later on
- chronic pulmonary disease
- bronchial strictures
- conjunctival scarring and blindness
- chronic non-healing skin lesions
- peripheral neuropathy.

Patients exposed to mustard agent should be subject to some form of long-term surveillance.

**LEWISITE**
Lewisite is an arsenic-based compound whose effects are very similar to that of sulfur mustard.

Differences between mustard and lewisite:
- Lewisite vapour or liquid produces pain or irritation on contact. The pain decreases as blisters form. The pain associated with mustard burns, by contrast, develops with the blisters.
- Blisters from lewisite develop faster.
• Large amounts of lewisite produce a syndrome of generalized increased endothelial permeability, resulting in so-called “lewisite shock”.
• Skin pigmentation changes are much less likely with lewisite.
• Lewisite has a specific antidote, British anti-lewisite.
• No data exist on long-term complications from lewisite.

For more information, see factsheet on p. 224.

**British anti-lewisite**
British anti-lewisite, or BAL, is a binding agent that has a higher attraction for arsenic than human tissue. It captures and deactivates the agent. It is available as eye drops, skin cream and intravenous preparations. Early application of topical BAL prevents or reduces blister formation. Intravenous BAL may be useful for lewisite shock and can also be used for various metal poisonings, but it produces a variety of unpleasant side effects.

**PHOSGENE OXIME**
Phosgene oxime is also described as an “urticariant” or “nettle agent” owing to the skin lesions it produces.

Phosgene oxime is synthesized from phosgene (p. 196), which is a readily available industrial chemical. It has never been deployed as a chemical weapon and it is the least studied of all these agents.

It has no specific antidote.

The effects of phosgene oxime are different from mustard in that:
• exposure instantly produces severe pain
• it produces solid urticarial wheals rather than fluid-filled blisters
• skin lesions occur within an hour
• systemic absorption or inhalation of phosgene oxime rapidly induces pulmonary oedema
• phosgene oxime exposure is associated with pulmonary embolism.

For more information, see factsheet on p. 196.
CYANIDE POISONING – BLOOD AGENT

Cyanide (HCN) is a commonly produced industrial chemical and blood agent that smells of bitter almonds. It is less toxic than most of the other agents used in chemical warfare and highly volatile (boiling point: 25.7°C). This limits its use as a chemical warfare agents, as large amounts of agent need to be delivered to achieve a significant affect. Cyanogen chloride is a derivative of cyanide that is less volatile and more persistent than pure cyanide. It shares the toxic features of cyanide but is also similar to chlorine (p. 173) in the irritation it produces to the upper airway. For more information, see the factsheet on p. 212.

SYMPTOMS OF TOXICITY
Cyanides/cyanogens inhibit multiple enzyme systems, the most important being cytochromes. This impairs oxidative respiration and cell function, which means that no cells get energy to conduct their basic processes.

Symptoms and signs of acute poisoning include:
• rapid onset of coma with no focal neurological signs
• mydriasis
• tachypnoea
• tachycardia with signs of the heart not having enough oxygen
• severe metabolic acidosis
• death by cardiac failure.

Figure 38: Chemical treatment field kit
<table>
<thead>
<tr>
<th>Agent</th>
<th>Description</th>
</tr>
</thead>
</table>
| Nitrites                     | • Nitrites convert some haemoglobin into methaemoglobin, which binds cyanide and can remove it from other enzymes  
• Amyl nitrite pearl dosage: one pearl per min. via inhalation  
• Sodium nitrite dosage: 4 mg/kg 10 ml (30 mg/ml) intravenously  
• Side effects: Methaemoglobin cannot carry oxygen nitrite poisoning may need treatment with methylthioninium chloride (methylene blue) |
| Thiosulfate                  | • Thiosulfate acts as a substrate for rhodanase, a naturally occurring enzyme that inactivates cyanide; usually used in conjunction with nitrites  
• Dosage: 100 mg/kg  
• Side effects: concentrations above 10 mg/dl may lead to vomiting, psychosis, arthralgia and myalgia |
| 4-dimethylaminophenol (4-DMAP) | • 4-DMAP is perhaps a safer and more predictable way of producing methaemoglobin  
• Dosage: 3–4 mg/kg 5 ml (50 mg/ml) intravenously (only one ampoule)  
• Side effects: reduction of oxygen carrying capacity, overdose, haemolysis |
| Dicobalt edetate (EDTA)      | • The cobalt in EDTA chelates cyanide  
• Sold commercially as KELOCYANOR  
• Dosage: 4 mg/kg 20 ml (15 mg/ml) intravenously  
• Side effects: EDTA is toxic in its own right; can cause severe hypotension, cardiac arrhythmias, convulsions |
| Hydroxocobalamin             | • The cobalt atom in hydroxocobalamin binds cyanide in a form that can be excreted in urine  
• Sold commercially as CYANOKIT  
• Dosage: 5 g initially; additional – 10 g intravenously  
• Side effects: transient discoloration (skin, mucous membranes, urine), allergic reactions |
| Oxygen                       | Administered as part of treatment                                                                                                                                                                           |
| Glucose                      | Binds cyanide weakly and may mitigate some of the toxicity of EDTA                                                                                                                                              |

Table 6: Treatment of cyanide poisoning
INTRODUCTION

Toxic industrial chemicals (also referred to as “TICs”) are chemicals that are developed or manufactured for use in industrial operations, or industrial, government, or academic research and pose a health hazard.

There are a vast number of chemicals that fulfil these criteria. The most dangerous are those which can spread as gas or vapour or by aerosol or droplet. Localized chemical spills of non-volatile compounds will only affect the individuals caught in the immediate vicinity. Discharge of toxic gas into the atmosphere can contaminate many more people. The best example of this is the 1984 Bhopal disaster, where toxic gases were accidentally released into the atmosphere from a chemical plant. Half a million people were poisoned and 40,000 injured. Chemicals can also pollute water supplies and food sources, but the consequences are not usually as severe as atmospheric release.

The likelihood that a chemical may be involved in a chemical incident (an act that results in the release of chemicals potentially dangerous to health into the atmosphere) is higher when the chemical is:

- produced on multiple continents
- produced by multiple manufactures
- toxic in small quantities
- a gas or highly volatile liquid.
<table>
<thead>
<tr>
<th>Risk rating</th>
<th>Chemical</th>
<th>Appearance</th>
<th>Odour</th>
<th>IDLH</th>
<th>Smell</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Chlorine ((\text{Cl}_2))</td>
<td>greenish-yellow gas; non-flammable</td>
<td>strong swimming pool</td>
<td>10</td>
<td>no</td>
</tr>
<tr>
<td>2</td>
<td>Ammonia ((\text{NH}_3))</td>
<td>colourless gas; non-flammable</td>
<td>pungent</td>
<td>300</td>
<td>no</td>
</tr>
<tr>
<td>3</td>
<td>Formaldehyde ((\text{CH}_2\text{O}))</td>
<td>colourless gas; combustible</td>
<td>pungent, suffocating</td>
<td>20</td>
<td>no</td>
</tr>
<tr>
<td>4</td>
<td>Ethylene oxide ((\text{C}_2\text{H}_4\text{O}))</td>
<td>colourless gas; flammable</td>
<td>ether-like</td>
<td>800</td>
<td>no</td>
</tr>
<tr>
<td>5</td>
<td>Sulfur dioxide ((\text{SO}_2))</td>
<td>colourless gas; non-flammable</td>
<td>irritating, pungent</td>
<td>100</td>
<td>no</td>
</tr>
<tr>
<td>6</td>
<td>Phosgene ((\text{COCl}_2))</td>
<td>colourless gas; non-combustible</td>
<td>suffocating, musty hay</td>
<td>2</td>
<td>no</td>
</tr>
<tr>
<td>7</td>
<td>Hydrogen fluoride ((\text{HF}))</td>
<td>colourless gas or fuming liquid</td>
<td>strong irritating</td>
<td>30</td>
<td>no</td>
</tr>
<tr>
<td>8</td>
<td>Arsine ((\text{AsH}_3))</td>
<td>colourless gas; flammable</td>
<td>mild, garlic</td>
<td>3</td>
<td>no</td>
</tr>
<tr>
<td>9</td>
<td>Nitric acid ((\text{HNO}_3))</td>
<td>colourless, yellow or red fuming liquid</td>
<td>acrid, suffocating</td>
<td>25</td>
<td>no</td>
</tr>
<tr>
<td>10</td>
<td>Boron trichloride ((\text{BCl}_3))</td>
<td>colourless gas</td>
<td>hydrochloric acid-like</td>
<td>unknown</td>
<td>no</td>
</tr>
<tr>
<td>11</td>
<td>Phosphorus trichloride ((\text{PCl}_3))</td>
<td>colourless to yellow fuming liquid</td>
<td>hydrochloric acid-like</td>
<td>25</td>
<td>no</td>
</tr>
<tr>
<td>12</td>
<td>Hydrogen cyanide ((\text{HCN}))</td>
<td>colourless-pale blue gas or liquid; flammable</td>
<td>bitter, almond</td>
<td>50</td>
<td>yes</td>
</tr>
<tr>
<td>13</td>
<td>Fluorine ((\text{F}_2))</td>
<td>pale yellow-greenish gas; non-flammable</td>
<td>pungent, irritating</td>
<td>25</td>
<td>no</td>
</tr>
<tr>
<td>14</td>
<td>Hydrogen sulfide ((\text{H}_2\text{S}))</td>
<td>colourless gas; flammable</td>
<td>rotten eggs</td>
<td>100</td>
<td>no</td>
</tr>
<tr>
<td>Risk rating</td>
<td>Chemical</td>
<td>Appearance</td>
<td>Odour</td>
<td>IDLH</td>
<td>Smell</td>
</tr>
<tr>
<td>------------</td>
<td>-----------------------------------------------</td>
<td>---------------------------------</td>
<td>--------------------------</td>
<td>------</td>
<td>-------</td>
</tr>
<tr>
<td>15</td>
<td>Sulfuric acid $(\text{H}_2\text{SO}_4)$</td>
<td>colourless to dark brown oily liquid</td>
<td>odourless</td>
<td>4</td>
<td>no</td>
</tr>
<tr>
<td>16</td>
<td>Boron trifluoride $(\text{BF}_3)$</td>
<td>colourless gas; non-flammable</td>
<td>pungent, suffocating</td>
<td>25</td>
<td>no</td>
</tr>
<tr>
<td>17</td>
<td>Diborane $(\text{B}_2\text{H}_6)$</td>
<td>colourless gas; flammable</td>
<td>repulsive, sweet</td>
<td>15</td>
<td>no</td>
</tr>
<tr>
<td>18</td>
<td>Hydrogen bromide $(\text{HBr})$</td>
<td>colourless gas; non-flammable</td>
<td>sharp, irritating</td>
<td>30</td>
<td>no</td>
</tr>
<tr>
<td>19</td>
<td>Hydrogen chloride $(\text{HCl})$</td>
<td>colourless-light yellow gas; non-flammable</td>
<td>pungent, irritating</td>
<td>50</td>
<td>no</td>
</tr>
</tbody>
</table>

Table 7: Chemicals most likely to be involved in a chemical incident (IDLH: concentration at which the chemical is immediately dangerous to life and health)\textsuperscript{27}

CHEMICAL WARNING LABELS EXPLAINED

NFPA 704
NFPA 704 is known as the “fire diamond”. The classification system was designed by the US National Fire Protection Agency in 1960. Each quadrant of the diamond represents a different potential hazard, and the contents of the quadrant specify the danger posed by the chemical in that respect.

![Diagram of NFPA 704]

**Health hazard**
- 4 Deadly
- 3 Extreme Danger
- 2 Hazardous
- 1 Slightly hazardous
- 0 Normal material

**Fire hazard**
- 4 Below 25°C
- 3 Below 37°C
- 2 Below 93°C
- 1 Above 93°C
- 0 Not Flammable

**Specific hazard**
- OXY Oxidizer
- ACID Acid
- ALK Alkali
- CORR Corrosive
- 🥇 Radioactive
- 🔴 Use no water

**Reactivity**
- 4 May Detonate
- 3 Shock + Heat
- 2 Violent Reaction
- 1 Unstable If heated
- 0 Stable

Figure 39: Warning labels for phosgene

ADR
“ADR” stands for the European Agreement concerning the International Carriage of Dangerous Goods by Road. The orange ADR label is used to label hazardous materials for transport. The ADR labels provided in this manual are comprised of two numbers: a hazard identification number (HIN) and a United Nations (UN) number.
• **HIN:** Also known as a Kemler code, a HIN consists of two to three digits, each of which represents a particular hazard. Examples:
  - 268 Toxic gas, corrosive
  - 85 Corrosive or slightly corrosive substance, oxidizing (fire-intensifying)
  - 265 Toxic gas, oxidizing (fire-intensifying)
  - 886 Highly corrosive substance, toxic

• **UN:** A UN number is a unique identifier assigned by the United Nations Committee of Experts on the Transport of Dangerous Goods to a chemical or group of chemicals with similar properties.

**IDLH**
“IDLH” stands for the concentration at which an agent is immediately dangerous to life or health.

**AEGL**
“AEGL” stands for “acute exposure guideline level”. An AEGL represents the concentration at which a chemical poses a defined level of risk for humans exposed to it.

• **AEGL-1:** Level of the chemical in air at or above which the general population could experience notable discomfort
• **AEGL-2:** Level of the chemical in air at or above which there may be irreversible or other serious long-lasting effects or impaired ability to escape
• **AEGL-3:** Level of the chemical in air at or above which the general population could experience life-threatening health effects or death
HAZARD PICTOGRAMS

Explosive (symbol: exploding bomb)

Corrosive (symbol: dripping liquid)

Flammable (symbol: flame)

Acute toxicity (symbol: skull and crossbones)

Oxidizing (symbol: flame over circle)

Hazardous to the environment (symbol: environment)

Health hazard/hazardous to the ozone layer (symbol: exclamation mark)

Gas under pressure (symbol: gas cylinder)

Serious health hazard (symbol: person with white mark on chest)
DOWNWIND HAZARD DISTANCES

With a chemical incident, a toxic cloud will concentrate downwind from the source, especially in low-lying areas. The term "downwind hazard distance" refers to the distance from the source of hazardous material within which people may be at risk of injury.

<table>
<thead>
<tr>
<th>Chemicals</th>
<th>Quantity</th>
<th>Distance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorine</td>
<td>Up to 100 tonnes</td>
<td></td>
</tr>
<tr>
<td>Phosgene</td>
<td>Up to 50 tonnes</td>
<td></td>
</tr>
<tr>
<td>Ammonia</td>
<td>Up to 500 tonnes</td>
<td>5 km</td>
</tr>
<tr>
<td>Hydrogen cyanide in hot climates</td>
<td>Up to 50 tonnes</td>
<td></td>
</tr>
<tr>
<td>Hydrogen sulfide</td>
<td>Up to 50 tonnes</td>
<td></td>
</tr>
<tr>
<td>Hydrogen cyanide in cold climates</td>
<td>Up to 50 tonnes</td>
<td></td>
</tr>
<tr>
<td>Hydrogen fluoride</td>
<td>Up to 100 tonnes</td>
<td>2.5 km</td>
</tr>
<tr>
<td>Hydrogen chloride</td>
<td>Up to 50 tonnes</td>
<td></td>
</tr>
<tr>
<td>Ammonia</td>
<td>Up to 100 tonnes</td>
<td></td>
</tr>
</tbody>
</table>

Table 8: Hazard distances following the release of chemical agents

ADAPTING TO THE TYPE OF INCIDENT

PLANNING

There is a vast body of literature on the management of HAZMAT incidents worldwide. Most of it originates from countries with well-equipped emergency services responding to a single, known toxin. Management plans are often in place and stockpiles of monitoring equipment and antidotes available. The principles and challenges described in that literature are useful and valid. Unfortunately, the solutions proposed often require more resources than can be found in a conflict zone.

Adapted from: idem.
**PPE**

The selection of PPE must be based on a careful assessment of the tasks, hazards and risks involved, as discussed previously in the handbook. The quantity of the toxic agent is often higher in a HAZMAT incident, and it is often located in a more concentrated area, meaning that a higher level of PPE may be necessary.

**CHOKING AGENTS AND TOXIC INDUSTRIAL GAS AND VAPOURS**

There are a large number of caustic industrial agents which are gas or highly volatile liquids that, if released accidentally, can be blown over a wide area and create large numbers of casualties. Gases can attack moist mucosal surfaces, such as the eyes and the respiratory tract. Splashes of liquid can cause damage to skin.

**DAMAGE TO THE AIRWAYS**

Descriptions of damage by caustic smokes and gases classify the airways into three zones:

- larynx and pharynx
- central compartment (main airways)
- peripheral compartment (small airways and lung parenchyma).

The part of the airways that is affected can to some extent be predicted by:

- the size of the inhaled particle
- the water solubility of the agent
- irritation and laryngospasm produced by the agent.
PARTICLE SIZE
Particles greater than 100 µ in size are filtered out by the nose/pharynx and do not enter the airways. Particles greater than 5 µ do not progress beyond the central compartment.

WATER SOLUBILITY
Agents that are water soluble appear to cause more irritation to the upper airway than insoluble agents. This may be protective, alerting people to an environmental hazard. The irritation can also cause laryngospasm/bronchospasm, swelling and severe airway compromise requiring intubation.

<table>
<thead>
<tr>
<th>Particle description</th>
<th>Particle size</th>
<th>Effect site</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coarse</td>
<td>1–10 µ</td>
<td>Nasopharynx</td>
</tr>
<tr>
<td>Fine</td>
<td>0.1–1 µ</td>
<td>Tracheobronchial tree</td>
</tr>
<tr>
<td>Ultrafine</td>
<td>&lt; 0.1 µ</td>
<td>Terminal bronchioles and alveoli</td>
</tr>
</tbody>
</table>

Table 9: Inhaled particles and the airways – effect site depending on particle size

<table>
<thead>
<tr>
<th>Agent</th>
<th>Water solubility</th>
<th>Principal site of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ammonia</td>
<td>High</td>
<td>More central than peripheral</td>
</tr>
<tr>
<td>Chlorine</td>
<td>Intermediate</td>
<td>Central and peripheral</td>
</tr>
<tr>
<td>Nitrogen dioxide</td>
<td>Low</td>
<td>More peripheral than central</td>
</tr>
<tr>
<td>Phosgene</td>
<td>Low</td>
<td>Peripheral</td>
</tr>
<tr>
<td>Sulfur dioxide</td>
<td>High</td>
<td>Central and peripheral</td>
</tr>
<tr>
<td>Sulfur mustard</td>
<td>Low</td>
<td>Central and peripheral</td>
</tr>
</tbody>
</table>

Table 10: Effect of solubility on site of respiratory injury
SYMPTOMS AND TREATMENT

Initial symptoms consist of:
• watering painful eyes
• blepharospasm
• coughing
• tight chest, chest pain
• wheezing, shortness of breath
• laryngospasm and acute respiratory failure
• nausea and vomiting
• contact burns or eye damage if exposed to liquid.

This is followed over the next few hours by:
• progressive lung injury, possibly requiring intubation and ventilation
• acute respiratory distress syndrome
• ulcerative tracheobronchitis
• sepsis and organ failure.

Long-term effects include pulmonary fibrosis and chronic pulmonary disease.

Treatment is purely supportive and should follow standard medical and intensive care protocols. No specific antidotes are available. Skin lesions produced by splashes of liquid will require surgical review.

Phosgene and chlorine are examples of industrial chemicals that have been used as chemical warfare agents. Phosgene (p. 196) is particularly effective, as the threshold for olfactory detection by humans is four times the concentration at which tissue damage can occur. With chlorine (p. 173), the detection limit is a tenth of the concentration at which damage begins.
Suggested reading


INTRODUCTION

All health-care organizations are familiar with outbreaks of pathogenic organisms. Reports of so-called pestilence and plagues can be found in chronicles dating back to 430 BCE. The essentials of management described in those texts share many similarities with modern practices. The outbreaks usually occur naturally but can also be released deliberately. A number of agents have been purposefully researched as weapons of war. Possible routes of spread are by contamination of food or water, person-to-person transmission, or aerosol and droplet sprays.

Figure 42: After the first World War baths were established for disinfection to fight epidemics
DETECTING PATHOGENS

Disease outbreaks are best detected by a functioning public health system and mechanisms for reporting infectious diseases, as mentioned in Section 3.7 (p. 35). The unrest and restrictions that result from ongoing conflict challenge health-care systems’ surveillance capacities. The International Red Cross and Red Crescent Movement has a system for community-based surveillance called Nyss. It can act as an early warning system at the community level to limit spread of disease. The WHO can also support all member states in developing their surveillance system, contingency plans and the collaboration between law enforcement and health-care sector that is necessary when the deliberate spread of disease is a concern.

Clinical warning signs of an outbreak include:

- multiple patients presenting with the same symptoms
- geographical clustering of cases
- unusual locations, e.g. plague in Switzerland
- unusual age group, e.g. pneumonia outbreak in young patients
- unusual patient group, e.g. animal disease in non-farm workers
- unusual clinical signs or progression, e.g. antibiotic resistance
- unusual organism, e.g. a single case of smallpox, anywhere in the world.

Organizations such as the UN, the WHO and the CDC can all provide assistance and advice if an outbreak is suspected.

MANAGING OUTBREAKS

All health-care facilities should have a minimum standard for infection prevention and control practices. Health-care workers should take those standard precautions whenever caring for patients to prevent the transmission of microorganisms. Managing infectious disease is a core health-care function, and a detailed description is beyond the scope of this handbook.

The principles of outbreak management are:

- seek expert advice
- vaccinate patients and give pre-exposure antimicrobial prophylaxis (if indicated)

For more about Nyss, see: https://www.cbsrc.org.
- use appropriate PPE and isolation measures — including packaging of laboratory samples
- treat symptomatic patients
- carry out testing
- perform contact tracing
- collect data and report outbreaks.

**SOME PATHOGENS OF PARTICULAR CONCERN**

**Anthrax**
Caused by the bacterium *Bacillus anthracis*. Forms spores that are extremely persistent. Found naturally in cattle. Can have cutaneous, pulmonary, gastrointestinal or central nervous system presentations depending on the mode of exposure.

**Botulism**
Produced by neurotoxins released by spore-forming bacterium *Clostridium botulinum*. Found naturally in soil and anaerobic environments. Infection usually from contaminated food or wounds. Classically presents with dysphonia, dysarthria, dysphagia and diplopia, progressing to descending paralysis and death from respiratory failure.

**Brucellosis**
Caused by a variety of *Brucella* bacteria commonly found in pigs and cattle. Usually presents insidiously as a fever of unknown origin, weight loss, endocarditis and hepatitis.

**Coronavirus**
Group of viruses responsible for COVID-19, severe acute respiratory syndrome (SARS) and Middle Eastern Respiratory Syndrome (MERS). All present with fever and respiratory failure and progress to multi-organ failure with early renal involvement. Treatment is supportive. MERS and SARS have especially high mortality rates.
Coccidioi-do-mycosis  
*Coccidioides immitis* is a spore-forming saprophytic fungus found in soil in the Americas. Presents as an acute fever and pneumonia from inhalation exposure, which resolves in a few weeks. One in 20 patients progresses to a chronic or disseminated form, with skin ulcers, osteomyelitis, meningitis or severe lung disease. Immuno-suppressed patients are at particular risk.

Glanders  
Principally a disease of cattle produced by *Pseudomonas mallei*. Can have cutaneous, pulmonary or systemic (septicaemia) presentations, depending on the mode of exposure.

Meliodosis  
Produced by *Burkholderia pseudomallei*, a bacterium found in contaminated soil and water in tropical climates. Very variable onset and presentation, starting with fever and weight loss and progressing to involve other organs.

Plague  
Caused by *Yersinia pestis* spread by fleas and rodents. Classically has pneumonic, bubonic or septicaemic presentations. Central nervous system involvement is common and treated with chloramphenicol.

Q fever  
Produced by *Coxiella burnetii*. Very hardy bacteria found in a wide range of cattle and mammals. Infection usually by inhalation or ingestion causing fever, weight loss, meningitis, endocarditis, hepatitis or other organ involvement.

Smallpox  
DNA virus that has been eradicated from the general human population; does not exist outside laboratories. Initial presentation very similar to chicken pox but with progression to coagulopathy, encephalitis and multi-organ failure.
**Tularaemia**  Caused by the bacteria *Francisella tularensis*, which is commonly found in a variety of small mammals in Europe, Asia, the Americas and Australia. Presentation can be cutaneous, pulmonary or pharyngeal, depending on the route of exposure.

**Typhus**  Flea- or mite–borne infection caused by a variety of *Rickettsia*. Presents with fever, malaise and a typical rash. Progresses to encephalitis. Usually occurs sporadically, but catastrophic outbreaks associated with conflict and population migrations occur. Significant mortality without antibiotic treatment.

**VEE**  Venezuelan equine encephalitis (VEE) is produced by a virus found in bats, birds, horses, mules and donkeys and spread by mosquitos. Presents with fever, malaise and myalgia and may progress to fatal encephalitis.

**VHF**  Viral haemorrhagic fevers (VHFs) are produced by a wide range of viruses from a variety of geographical locations and animal reservoirs. Present with fever, disseminated intravascular coagulopathy and organ failure.

Ebola, Marburg virus disease, Lassa fever and Congo-Crimean haemorrhagic fever are the most concerning. They have a high mortality rate and a high rate of person–to–person spread, potentially to medical staff.

Other examples with a lower mortality rate include dengue fever, yellow fever and Rift Valley fever.
<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Incubation period</th>
<th>Strict isolation</th>
<th>Organism type</th>
<th>Contact tracing</th>
<th>PPE precautions required</th>
<th>Vaccine available</th>
<th>Example of first-line therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthrax</td>
<td>1–7 days</td>
<td>x</td>
<td>Bacteria</td>
<td>x</td>
<td>Standard</td>
<td>✓</td>
<td>Ciprofloxacin or doxycycline; antitoxins available</td>
</tr>
<tr>
<td>Botulism</td>
<td>12–72 hours</td>
<td>x</td>
<td>Bacteria</td>
<td>x</td>
<td>Standard</td>
<td>Phase II trials</td>
<td>Antitoxin available</td>
</tr>
<tr>
<td>Brucellosis</td>
<td>1–3 weeks</td>
<td>x</td>
<td>Bacteria</td>
<td>x</td>
<td>Standard</td>
<td>x</td>
<td>Doxycycline (or oral tetracycline) combined with rifampicin</td>
</tr>
<tr>
<td>Glanders</td>
<td>10–14 days</td>
<td>x</td>
<td>Bacteria</td>
<td>x</td>
<td>Standard</td>
<td>x</td>
<td>Meropenem + trimethoprim/ sulfamethoxazole</td>
</tr>
<tr>
<td>Melioidosis</td>
<td>2–4 weeks? Years</td>
<td>x</td>
<td>Bacteria</td>
<td>x</td>
<td>Standard</td>
<td>x</td>
<td>Meropenem</td>
</tr>
<tr>
<td>Plague</td>
<td>2–8 days</td>
<td>✓</td>
<td>Bacteria</td>
<td>x</td>
<td>Droplet</td>
<td>✓</td>
<td>Ciprofloxacin +/- chloramphenicol</td>
</tr>
<tr>
<td>Q fever</td>
<td>1–5 weeks</td>
<td>x</td>
<td>Bacteria</td>
<td>x</td>
<td>Standard</td>
<td>✓</td>
<td>Cotrimoxazole or doxycycline</td>
</tr>
<tr>
<td>Tularaeemia</td>
<td>Up to 2 weeks</td>
<td>x</td>
<td>Bacteria</td>
<td>x</td>
<td>Standard</td>
<td>Not generally available</td>
<td>Ciprofloxacin or gentamicin</td>
</tr>
<tr>
<td>Typhus</td>
<td>1–2 weeks</td>
<td>x</td>
<td>Bacteria</td>
<td>x</td>
<td>Standard</td>
<td>x</td>
<td>Doxycycline or chloramphenicol</td>
</tr>
</tbody>
</table>

31 Examples of appropriate antibiotics are included to show whether therapies are available, but the choice must take into account local resistance, the patient’s condition and severity of illness. Local guidelines should be consulted or, if none is available, the WHO’s classification.
<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Incubation period</th>
<th>Strict isolation</th>
<th>Organism type</th>
<th>Contact tracing</th>
<th>PPE precautions required</th>
<th>Vaccine availability</th>
<th>Example of first-line therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronavirus</td>
<td>1–2 weeks</td>
<td>✓</td>
<td>Virus</td>
<td>✓</td>
<td>Aerosol</td>
<td>SARS – No</td>
<td>MERS – No</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>COVID-19 – Yes</td>
<td></td>
</tr>
<tr>
<td>Smallpox</td>
<td>1–2 weeks</td>
<td>✓</td>
<td>Virus</td>
<td>✓</td>
<td>Aerosol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Viral haemorrhagic fever</td>
<td>Up to 3 weeks</td>
<td>✓</td>
<td>Virus</td>
<td>✓</td>
<td>Aerosol</td>
<td>Depends(^{33})</td>
<td>Ribavirin(^{34})</td>
</tr>
<tr>
<td>Venezuelan equine encephalitis</td>
<td>1–5 days</td>
<td>×</td>
<td>Virus</td>
<td>×</td>
<td>Standard</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coccidioido-mycosis</td>
<td>1–3 weeks? Months</td>
<td>×</td>
<td>Fungus</td>
<td>×</td>
<td>Standard</td>
<td></td>
<td>Amphotericin</td>
</tr>
</tbody>
</table>

Table 11: Organism summary\(^{35}\)

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\(^{32}\) Includes severe acute respiratory syndrome (SARS) and Middle Eastern Respiratory Syndrome (MERS).

\(^{33}\) Marburg virus disease: no vaccine. Lassa fever vaccine in Phase I trials as of Aug. 2022. A vaccine is available against the strain of Ebola responsible for 60 to 70% of outbreaks. Less effective against the three other strains in circulation.

\(^{34}\) Ribavirin may be effective for Lassa fever and Congo–Crimean haemorrhagic fever only.

INFECTION CONTROL AND PPE

See also Annex F (p. 143) for more information.

Patients infected with any of the agents previously discussed present a significant health hazard to medical and nursing staff. Appropriate precautions must be taken to minimize the risk of cross-contamination. Standard precautions may vary nationally, but the guidelines provided in this handbook are common practices.

Infection control precautions are categorized here as standard, droplet or aerosol.

Note: PPE selection must be based on a careful assessment of the tasks, hazards and risks involved, not just general advice. Avoid reducing it to a checklist.

STANDARD PRECAUTIONS

Figure 43: Medical staff following standard precautions
• Practice good basic hygiene with regular hand cleaning.
• Staff with wounds or skin lesions should cover them with waterproof dressings.
• Wear disposable gloves (sterile or non-sterile depending on the task).
• Avoid touching eyes, nose, mouth or face or adjusting PPE with contaminated hands or gloves.
• Limit contact with items in the patient’s immediate environment.
• Work from clean to dirty. Change gloves during a procedure when moving from a dirty body site to a clean one. Torn or heavily soiled gloves should be discarded and replaced after handwashing.
• Wear a single-use plastic apron as a standard precaution, but use a full-body, fluid-impermeable gown for tasks with a risk of splashing by contaminated fluids.
• Consider wearing eye protection or a surgical facemask, especially for tasks where there is a risk of splashes or spray to the face or eyes.

Figure 44: Eye protection shields against splashes of contaminated fluids

Take precautions to minimize the risk of sharps injury – if necessary, modify or change equipment.

Visitors are permitted.

Contaminated waste must be disposed of appropriately and biohazard spills rapidly cleaned up.

Keep in mind that, if biohazardous waste cannot be managed on site, it may be subject to regulatory transport restrictions. Do not reuse single-use items, and ensure all equipment is correctly decontaminated.
DROPLET PRECAUTIONS

- Practice good basic hygiene with regular hand cleaning.
- Use a full-body, fluid-impermeable gown and/or a disposable plastic apron on top that can be changed between tasks.
- Wear a facemask, ideally FFP3.
- Wear a face shield.
- Staff with wounds or skin lesions should cover them with waterproof dressings.
- Wear disposable gloves – one or two pairs, sterile or non-sterile, depending on the task. Nitrile gloves are preferred to latex.
- Develop a donning and doffing protocol.

Place patients in single rooms or cubicles, and limit movement as much as possible. Patients should wear face masks when leaving the room or cubicle.

Relatives may visit the patient but should wear facemasks and avoid contact with the patient.

Maintain droplet precautions until infection has been excluded or the patient is no longer contagious.
AEROSOL PRECAUTIONS


During the 2014 to 2016 Ebola outbreak in West Africa, estimated overall infection rates among health-care workers were 30 to 44 per 1,000. Many of them died. Well run facilities with proper training and equipment were able to reduce their infection rate to 4.3 per 1,000, or even lower. The infection rate in the general population was estimated at 1.4 per 1,000.

Figure 46: PPE worn at an Ebola treatment unit in Liberia – the staff are wearing different gloves because PPE is not just dependent on the pathogen but also the activity being performed

These are precautions for some of the most transmissible pathogens on earth:

- scrupulous hand hygiene
- scrubs/uniform (this PPE is very hot, so undergarments should be thin)
- waterproof gown or coveralls that extend beyond the top of the boots
- rubber boots or plastic boot covers

• FFP3 mask or full-face respirator
• facial protection with goggles and/or face shield and hood
• two pairs of gloves for standard clinical tasks (nitrile gloves are preferred to latex).

This level of PPE is difficult to work in and physically demanding. Staff should be fitted with equipment of the correct size and must be trained in correct use. Donning and doffing is best performed under direct supervision – especially when PPE is being removed. Shifts and work patterns must be carefully planned.

Suspected patients should be isolated in separate spaces and wear respiratory protection when leaving the area. Confirmed patients can be isolated in cohorts. All staff in the vicinity of the patient must wear appropriate PPE.

Movement of patients must be minimized and visits conducted at a safe distance with appropriate precautions.

Maintain aerosol spread precautions until infection has been excluded or the patient is no longer contagious.
DU is an extremely dense and heavy material. It is used in civilian contexts for ballast and radioactive shielding. In military contexts, it is used for armour-piercing projectiles and tank armour. It emits about 60% of the radioactivity of natural uranium.
INTRODUCTION

Most DU is formed from the processing of natural uranium. A small amount has been produced from the reprocessing of spent nuclear fuel. DU produced in this way contains traces of radionucleotides such as U–232, U–236, plutonium, americium, neptunium and technetium–99. There is no evidence that DU from reprocessed nuclear fuel has been used in the manufacture of munitions.

Figure 48: Barrels similar to those used for storing uranium hexafluoride (left) and a uranium hexafluoride molecule

About one-and-a-half million tonnes of DU has been produced worldwide in the manufacture of enriched uranium for fuel and weapons. Some of the storage techniques have significant potential for environmental contamination. Uranium hexafluoride stored in metal barrels has been a particular cause for concern. Other methods of containment have been developed, but safe storage of low-grade radioactive waste continues to be a problem.

There is no specific rule in international humanitarian law prohibiting the use of DU munitions as a category of weapon. DU munitions are conventional weapons and are not considered nuclear, radiological or chemical weapons. As with all conventional weapons, the use of DU munitions is subject to the general rules governing the conduct of hostilities. DU ammunition has been used in a number of locations around the world. Most of the published studies which investigate the potential for human and environmental contamination by these projectiles originate from the Balkans and the Middle East.
**DU RADIOACTIVITY**

DU emits alpha, beta and gamma radiation at 60% of the rate of natural uranium. Areas where depleted uranium munitions have been used do not appear to have increased levels of background radiation.

**WEAPON CONTAMINATION FROM DU**

A projectile striking a hard surface produces vapourized particles of metal, dust and fragments. Anyone within a 50-metre radius of the impact point of a projectile containing DU can inhale, ingest or be penetrated by the uranium. This produces the highest level of contamination, and follow-up of casualties injured in this way is extremely valuable in determining the long-term health risks of DU.

![Figure 49: A 30mm shell tipped with depleted uranium](image)

DU involved in a fire will oxidize and spread as smoke/particulates over a wide area.

Discarded rounds containing DU can contaminate the environment and present a long-term but low-grade chemical and radiation hazard. Contamination of water sources appears to be more of a threat than uptake by plants and the rest of the food chain.

Soldiers and industrial workers may work in environments where they are in close proximity to DU munitions for long periods of time.
URANIUM METABOLISM AND TOXICITY

Uranium is toxic in two ways:

• As a heavy metal, it is biochemically toxic to humans and presents about the same level of hazard as mercury or lead.
• DU is a low-level radiation source. Fragments of uranium that are incorporated following ingestion, inhalation or penetration may pose a long-term cancer risk.

The chemical properties of DU are probably more significant than its radioactivity. 37

URANIUM METABOLISM

Uranium usually enters the body by ingestion or inhalation – a trivial amount can be absorbed through the skin. Ingested uranium is absorbed through the small intestine via a transcellular pathway. Particles of uranium dust entering the lungs are cleared into the bloodstream surprisingly quickly. Respiratory absorption of uranium is probably more significant than intestinal absorption. Of the uranium that enters the bloodstream, 70% is eliminated by the kidneys, and 20% is retained in bone. It is also found in the brain, liver and spleen.

Data for uranium toxicity come from animal experiments, industrial accidents, exposure to weapon contamination and historical treatments for diabetes.

Kidneys: Uranium–induced renal failure

Acute uranium salts injected into rats can produce a 50% reduction in renal function 48 hours after exposure. This pattern also has been observed in survivors of industrial accidents. Rats with fragments of uranium metal implanted into muscle have shown build-up of uranium in their kidneys along with histological changes suggesting damage.

Follow-up of Gulf War veterans with known retained fragments of uranium have shown increased levels of urinary uranium but no evidence of renal damage.

**Other organs**

Uranium toxicity has been postulated to cause, myocarditis, cognitive impairment, immune disruption, teratogenicity and carcinogenesis – especially leukaemia.

Evidence for damage to organs other than the kidney is weak. No deaths have ever been definitively attributed to acute uranium poisoning.

**DU AND HEALTH**

Laboratory evidence shows that uranium is a heavy metal and has the potential to cause harm to humans. Demonstrating an irrefutable impact to health or the environmental has not, as yet, been possible. Research in this area is hotly debated; data collection and comparative studies are plagued with confounding factors.

Most published data are from uranium miners, industrial uranium workers, veterans and the civilian populations of the Balkans and the Middle East.

**TERATOGENICITY**

Hundreds of tonnes of DU were used in the First Gulf War, in particular by the US and UK militaries. Data from Gulf War veterans and the Iraqi civilian population suggest that these two groups may well have a significant risk of producing children with birth defects:

- An American study in 2001 compared 15,000 Gulf War veterans to 15,000 non-deployed soldiers. The incidence of birth defects in children of veterans is two to three times that of the control group.
- Rates of congenital malformations in children of British servicemen who deployed to the Gulf in 1991 may be up to 50% higher than expected.

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A 2010 study of children born in Fallujah, Iraq, reported the rate of congenital malformation to be almost 15%.\(^{41}\)

All the studies listed above have been criticized for flaws in data collection, case selection, confounding factors and methodology.

It is possible that there is an elevated rate of congenital malformations in the populations described. Animal models show that uranium may be teratogenic in rats. However, at this time, the available evidence fails to prove a definite link between depleted uranium and teratogenicity. Research is ongoing.

**CARCINOGENESIS**

Uranium miners have a high rate of respiratory disease and lung cancer. This has mostly been attributed to dust, cigarette-smoking and radon gas inhalation. Uranium miners in the US are entitled to compensation for occupation related lung disease.

Soldiers deployed to the Gulf and the Balkans (even those suffering from Gulf War Syndrome) do not appear to have an increased rate of cancer.\(^{42}\) Tank crews and ammunition workers who operate in close proximity to DU munitions do not appear to have an increased rate of cancer. Additionally, veterans with retained fragments of DU from penetrating fragments do not appear to have an increased incidence of cancer despite elevated levels of uranium in urine and tissues. While Italian soldiers who deployed to the Balkans in 2001 and 2002 reported a spike in lymphoma rates, following further analysis and data collection this spike was attributed to natural fluctuations in case incidence and not related to deployment.\(^{43}\)


Anecdotal reports from Fallujah, Mosul and Basra have suggested high rates of death from leukaemia. Unfortunately, all these studies are plagued with confounding factors, and proving a causal link to DU has not been possible.

In summary, a link between DU contamination and carcinogenesis has been postulated, but as yet no human studies have been able to show a definite association.

**GULF WAR SYNDROME**

Veterans of multiple nationalities from the 1991 Gulf War have complained of wide-ranging chronic symptoms that have collectively been described as Gulf War Syndrome. A huge amount of research has been published on the syndrome. Summarizing the research published on Gulf War Syndrome is beyond the scope of this document. DU has been investigated as a possible cause, but no evidence to support an association was uncovered. It is probable that other factors are responsible.

**REVIEWS BY INTERNATIONAL BODIES**

Detailed reviews trying to establish a link between DU and human health have been produced by the IAEA, the WHO, the UN Environment Programme, the North Atlantic Treaty Organization and the European Commission. The summary of all these documents is the following:

- experimental evidence suggests DU is potentially toxic
- no actual link between ammunition containing DU and damage to human health has been established
- more research is required.


ANNEX E
DETECTION EQUIPMENT

INTRODUCTION

Detector systems are available for detecting chemical, radioactive, radionucleotide and biological contamination of the environment.

Figure 50: ICRC staff performs detection of unknown chemical substances at a training course

CHEMICAL AGENT DETECTORS

UNDERLYING TECHNOLOGIES

Detectors have been designed that work in several different ways.

Chemical

These are some of the oldest and crudest methods of detecting chemical agents.

Strips of paper impregnated with a detector medium change colour when coming into contact with liquid G-type and V-type nerve agent as well as blister agent (H).
Vapours can be detected by bubbling samples of air through a solution of detector medium.

**Figure 51: Chemical detector paper**

**Ion mobility spectroscopy**
Gas is introduced into a detector and ionized. An electric field moves the ions towards a detector through a gas medium that slows down the movement of the ions.

**Flame photometry**
Sample gas is drawn into a monitor and burned. The wavelengths of light given off by the burning gas help to identify it.

**Infrared spectroscopy**
Infrared spectroscopy identifies an agent by its absorption of infrared light.

**RAMAN spectroscopy**
A sample of gas is illuminated by a laser of a set frequency. The atoms irradiated by this laser emit light at a variety of different frequencies, which can be used to identify the sample gas.

**Surface acoustic waves**
A sample gas can be identified by how it alters the vibration of a piezoelectric crystal.
DETECTORS USED BY THE ICRC

AP4C

Figure 52: AP4C

**Introduction to flame spectrometry**

The AP4C detects chemicals using flame spectrometry. Usually, chemical emissions are analysed to detect the presence and quantity of metallic elements, but elements from organic molecules can be detected too. Flame spectrometry is one of the most sensitive analytical methods: A few milligrams of a sample are usually enough to detect elements present to the extent of a few parts per million or less. Additionally, the method is able to detect several atomic species simultaneously, without having to separate them.

The AP4C detects chemicals using flame spectrometry. A flame is used to heat the substance being analysed, and the energy from the flame excites the electrons in the substance into a higher energy state. When the excited electrons return to their normal energy state, they emit a photon (light). Each chemical element emits light at a specific wavelength; by measuring the wavelength of the light emitted, the AP4C identifies the elements present in the substance being analysed.
Quantitative analysis

With flame spectrometry, it is possible to detect not just what elements are present, but in what quantity. The quantity of light (i.e. the intensity) emitted at a given wavelength is proportional to the number of atoms excited. The quantity of given element is usually determined by, in essence, comparing the intensity of the light emitted to that of a known composition.

In comparison to other technologies, flame spectrometry presents some distinct advantages:

- The measurement is instantaneous: each time molecules are sampled in the flame, they react immediately.
- The burner can be moved to adjust the relative height of the flame, which allows for optimum sensitivity for all detectable elements.
- There’s no memory effect. Even if you sample a high concentration of chemicals, the sensitivity for all detectable elements will be recovered after a few seconds.
- Hydrogen is a low-noise flame. Hydrogen combustion with air produces very few emission lines in the visible spectrum. There is no interference between them.
- All elements can be detected simultaneously because they emit photons of different energy.
**Application to detecting toxic chemicals**

The AP4C uses the combustion of hydrogen in air to excite the atoms that are to be sampled. The reaction between hydrogen and oxygen (which makes up approximately 20% of air) produces enough energy to split up organic molecules into their component parts and to excite the electrons in sulfur, phosphorus, arsenic and nitrogen. These atoms can be found in most chemical warfare agents.

The atmosphere is continuously sampled, and some of the sample is introduced into the flame. The flame is maintained in a chimney to optimize the output of heat, stabilize its position and induce the recombination of emissive species.

A sensor device measures the light emitted through an optical filter or with small, height-sensitive spectrometers.
Dräger X-am 5000 – multi-gas monitor

*Intended use*

The Dräger X-am 5000 is a portable gas-detection instrument for continuous monitoring of the concentration of several gases in the ambient air within the working area and in explosion-hazard areas. Depending on the device type and the sensor configuration, it is possible to carry out independent measurement of up to five gases.

![Figure 54: Dräger X-am 5000](image)

**Informational video (total time: 14:49)**

Part 1/5: Overview
https://www.youtube.com/watch?v=TqSbhMR8RVM

Part 2/5: Operation
https://www.youtube.com/watch?v=otiX6rw39Fs

Part 3/5: Alarms
https://www.youtube.com/watch?v=o2DqMONAffM

Part 4/5: Operation with Dräger X-am pump
https://www.youtube.com/watch?v=Ya3N7hNwCBU

Part 5/5: Dräger X-am 5000/X-am 5600 – bumptest and maintenance
https://www.youtube.com/watch?v=dmh3cF6wUFg
RADIATION AND RADIONUCLEOTIDE DETECTORS

TYPES OF DETECTORS
Detectors fall into one of three main types: survey meters, dosimeters and radiation isotope identification devices.

Survey meter
This device measures the amount of radiation in the environment at the moment of measurement. Not all devices measure all types of radiation.

Dosimeter
Dosimeters monitor accumulated radiation exposure; they are usually radiation-type specific. Some dosimeters have a display for real-time readings.

Radiation isotope identification device (RIID)
Different radioactive materials produce unique fingerprints of radiation. RIIDs identify the radioactive materials from the radiation they emit. Identifying which radioactive materials are present help to target the response accordingly.
UNDERLYING TECHNOLOGIES

Gas-filled
Gas in a tube becomes ionized when encountering radiation. The rate and quantity of ion formation reflects the amount of radiation present. Subtle differences in technology subdivide these devices into ion chambers, proportional detectors and Geiger–Müller tubes.

This technology is used for dosimeters and survey meters.

Scintillators
These detectors contain a compound that emits photons when hit by radiation. The photons can be detected by a receiver that produces a current when hit by photons. The number of photons emitted depends on the energy of the radiation, which is specific to the radionucleotide emitting it.

This technology is used for RIIDs.

Figure 56: Example of an RIID
**Solid-state**

These devices contain semiconductors that release electrons when hit by radiation. They are particularly useful for devices that need to measure very high levels of radioactivity.

![Figure 57: Sputum samples ready for analysis at a maximum-security prison in Muntinlupa city, something that requires time and training to do](image)

**BIOLOGICAL DETECTORS**

Biological detectors continually sample the environment (usually the air) to identify traces of biological pathogens.

![Figure 58: Microscope analysis at an ICRC and Syrian Arab Red Crescent hospital in Al Hol](image)
Detection techniques:
• tests for total protein that may suggest the presence of organisms
• tests for specific antigens
• tests for specific enzymes that pathogens may contain
• spore detection
• DNA analysis
• enzyme–linked immunosorbent assay (ELISA)
• rapid polymerase chain reaction (PCR) capability
• pH analysis
• count of particles of a specific size
• culture mediums into which pathogens may fall and grow.

These early–warning biological detectors are very crude, and microbiological support must be available to confirm their findings.

Figure 59: An example of a rapid diagnostic test
PPE must be selected based on a careful assessment of the tasks, hazards and risks involved, as discussed previously in the handbook. The US Occupational Safety and Health Administration outlines four levels of PPE, as depicted in the table 12 (p. 144).

The various types of PPE mentioned in the table 12 (p. 144) may be combined depending on the threat. An example is shown in Figure 61, where an air-purifying respirator is worn without a splash suit. The combination is less physically stressful and is suited for such operations as clinical tasks with minimal contamination exposure.

If no PPE is available, you may be able to instruct people at a distance to undress and decontaminate themselves.

If you expose yourself to the hazard without appropriate PPE, you may end up becoming one more patient on the scene.
<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Completely encapsulated suit and self-contained breathing apparatus</td>
<td>Highest level of protection available for both contact and inhaled threats or oxygen-deficient environments</td>
<td>Expense and training requirements restrict use to HAZMAT response teams</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lack of mobility; heat and other physical stresses; limited air supply/difficulty in re-supplying air and maintaining equipment in certain environments</td>
</tr>
<tr>
<td>B</td>
<td>Encapsulating suit or junction seams sealed, supplied air respirator or self-contained breathing apparatus</td>
<td>High level of protection suitable for unknown environment entry; supplied air ensemble with increased mobility and dexterity</td>
<td>Dependence on air hose or limited air supply; heat and physical stresses; significant expense and training; fit testing required</td>
</tr>
<tr>
<td>C</td>
<td>Splash suit or CBRN breathable suit and air-purifying respirator (for more details: please refer to the ICRC’s Guidelines for the Pre-Hospital Management of Persons Contaminated with Chemical Agent during Armed Conflict and Other Violence (2019))</td>
<td>Significantly increased mobility; decreased physical stress; extended operation time with high levels of protection against most agents in limited concentrations; no fit testing required for hood type</td>
<td>Insufficient for some high-concentration environments or less than atmospheric oxygen environments (unless appropriate detection equipment and training in place) or high levels of splash contamination; filter specificity; moderate expense and training</td>
</tr>
<tr>
<td>D</td>
<td>Work clothes, including standard precautions for health-care workers (e.g. gloves, splash protection)</td>
<td>Increased mobility; decreased physical stresses; extended operation time; cheap and minimal training required</td>
<td>Offers no protection against chemical or other agents</td>
</tr>
</tbody>
</table>

Table 12: Advantages and disadvantages of the different levels of PPE

---

FACIAL HAIR

It is very difficult, if not impossible, to keep a good seal between the respirator and the skin with facial hair. It is advisable that anyone wearing level C PPE is clean-shaven, as even stubble can hinder an effective seal. If you do have facial hair, it must not disrupt the functioning of your respirator, and careful fit testing is recommended.

Using a hood with a large visor can eliminate the need to test the seal between a mask and the face, and thus to shave facial hair. It also enables the user to wear normal glasses or a scarf. This eliminates any limitations on use for cultural or religious reasons or for those wearing glasses.
PPE WORN IN A DECONTAMINATION AREA

Figure 62: Splash suits, protective gloves, boots and air-purifying respirators used at an ICRC decontamination training course conducted in Iraq in 2016

AVON C50 RESPIRATOR

- Depending on the filter used, the respirator is effective against chemical, biological agents and provides limited HAZMAT protection (see p. 148, below, for the difference between CBRN and HAZMAT incidents).
- The respirator is available in small, medium and large.
- An internal lens can be fitted for glasses-wearers.
- The cannister can be fitted to the left or right.
- The respirator is compatible with CamelBak hydration systems, for drinking water.
- The respirator has communication capability.

Respirators must be correctly fit-tested, maintained and cleaned

Figure 63: AVON C50 respirator
CBRN COVERALL

- There are multiple manufactures of coveralls with a variety of materials of varying permeability.
- Coveralls are usually worn over a layer of clothing.
- Their shelf life and resistance to agents vary by manufacturer.
- Change coveralls if torn, soaked, covered in oil/petrol or contaminated with liquid agent.
- They must be sized to fit.
- Dehydration and overheating are a major problem.

DOUBLE GLOVES

- The outer glove should be butyl rubber; a variety of thickness are available:
  - 7mil (not millimetres) is the minimum safe thickness for staff involved in delicate work
  - 25mil is for heavy manual labour
  - intermediate thicknesses are available.
- The inner glove is usually of cotton and is to protect the skin from sweat.
- The outer glove is worn over the sleeve, and a secondary seal with tape is often made at the junction of the sleeve and glove.
BOOTS

Figure 66: Protective boots

A variety of boots and over boots are available. Overboots are designed to fit over other shoes.

Depending on the design, overalls may fit inside or over the boot and a secondary seal may be made with tape or a gaiter.

DIFFERENCE BETWEEN CBRN AND HAZMAT INCIDENTS

Chemical warfare agents are some of the most dangerous chemicals on earth. They are usually delivered at a low concentration. Level C PPE, which is worn by the military and recommended by the ICRC for decontamination areas, is designed to deal with the secondary contamination staff may be exposed to while taking care of victims as they exit the hot zone.

Industrial HAZMAT incidents usually result in the release of large amounts of chemical – often over a small area. Depending on the chemical, level A or B PPE is likely required.

If using level C PPE, it is important to be sure that:
- the agent in question is removed by the filter of the respirator
- the atmosphere is not oxygen-deficient
- the suits, gloves and boots will be able to resist the chemical (i.e. not be dissolved by it).

In general, level A or B PPE is recommended for chemical spills. Level C respirators cannister have a finite filtering ability. The filter becomes saturated as it
absorbs chemicals and will fail with time. In areas of relatively low contamination, such as a casualty decontamination point, it will function effectively for several hours. In an area of high contamination, such as the scene of a chemical spill, it may be overwhelmed by the quantity of vapour that needs to be removed and fail faster.

RESPIRATOR CANNISTERS

SHELF LIFE
A variety of manufactures and cannister sizes are available. In general, cannisters have a shelf life of:

• five years (occasionally ten) if kept in the original packaging
• six months once the packaging has been opened.

USE AND MAINTENANCE

Figure 67: Respirator cannister, Avon Protection

The number of hours of use must be recorded for each cannister. Cannisters should be inspected regularly. Test cannisters with a strongly smelling agent – the smell should be filtered out. If not, the cannister must be changed.

As well, cannisters should be changed out if:

• exposed to oils
• dented or damaged
• soaked in water
• excessive breathing resistance is perceived
• the predicted maximum operational life (in hours) is reached.
DIFFERENTIATING FILTER TYPES
Each gas filter has a specific colour code (see Table 13, below). The colour code tells you which harmful gases and vapours the filter protects against.

Filters are split into different classes according to their capacity (gas filters) or their efficiency (particle filters); see Table 14, below. Class 2 gas filters may be used at higher concentrations or for a longer time than Class 1 filters. The class of a particle filter indicates how efficient the filter is in filtering out particles (Class 1: 80%, Class 2: 94%, Class 3: 99.95%).

<table>
<thead>
<tr>
<th>Colour code</th>
<th>Filter type</th>
<th>Contaminants present</th>
</tr>
</thead>
<tbody>
<tr>
<td>AX¹</td>
<td>Gases and vapours of organic compounds with boiling point ≤ 65°C</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>Gases and vapours of organic compounds with boiling point &gt; 65°C</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>Inorganic gases and vapours, e.g. chlorine, hydrogen sulfide, hydrogen cyanide</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>Sulfur dioxide, hydrogen chloride</td>
<td></td>
</tr>
<tr>
<td>K</td>
<td>Ammonia and organic ammonia derivatives</td>
<td></td>
</tr>
<tr>
<td>CO²</td>
<td>Carbon monoxide</td>
<td></td>
</tr>
<tr>
<td>Hg³</td>
<td>Mercury vapour</td>
<td></td>
</tr>
<tr>
<td>NO⁴</td>
<td>Nitrous gases, including nitrogen monoxide</td>
<td></td>
</tr>
<tr>
<td>Reactor⁵</td>
<td>Radioactive iodine, including radioactive methyl iodide</td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>Pesticides</td>
<td></td>
</tr>
</tbody>
</table>

1 AX filters may only be used as supplied from the factory. Reuse, and use against gas compounds, is absolutely impermissible.
2 CO filters are single-use only and must be disposed of after use. Special guidelines according to local regulations apply.
3 Hg filters can only be used for a maximum of 50 hours, according to European Standard EN14387.
4 NO filters are single-use only and must be disposed of after use.
5 For reactor filters, special guidelines according to local regulations apply.

Table 13: Filter colour codes

<table>
<thead>
<tr>
<th>Filter type</th>
<th>Filter class</th>
<th>Protection against</th>
<th>Maximum permissible concentration of toxic substance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gas filter¹</td>
<td></td>
<td>Gases and vapours</td>
<td>50x the OEL with half mask / 2,000x the OEL with full-face mask, but maximum:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Capacity:</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Small</td>
<td>0.1 vol% (1,000 ppm)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Medium</td>
<td>0.5 vol% (5,000 ppm)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Large</td>
<td>1.0 vol% (10,000 ppm)¹</td>
<td></td>
</tr>
<tr>
<td>Particle filter²</td>
<td></td>
<td>Particles efficiency</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Separation ability:</td>
<td></td>
</tr>
<tr>
<td>P1</td>
<td>Small</td>
<td>4x the OEL with half mask / 5x the OEL with full-face masks</td>
<td></td>
</tr>
<tr>
<td>P2</td>
<td>Medium</td>
<td>12x the OEL with half mask / 16x the OEL with full-face mask</td>
<td></td>
</tr>
<tr>
<td>P3</td>
<td>Large</td>
<td>48x the OEL with half mask / 1,000x the OEL with full-face mask</td>
<td></td>
</tr>
</tbody>
</table>

Example: Lead dust OEL = 0.1 mg/m³; 4 x 0.1 mg/m³ = 0.4 mg/m³, i.e. maximum permissible concentration of lead dust for P1-filter with half mask.

<table>
<thead>
<tr>
<th>Combined filter</th>
<th>Gases, vapours and particles</th>
<th>Appropriate combined gases and particulate filters</th>
<th>Appropriate combined levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-P2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-P2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-P3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-P3</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

¹ Values taken from European Standard EN14387
² Values taken from CEN Report 529

**Table 14:** Classes of filters (OEL: occupational exposure limit)
An example is the following filter type:

![Filter Types (A2 B2 P3)](image)

This filter would be suitable for the following contaminants:

**A2:** Gases and vapours of organic compounds with boiling point above 65°C up to concentrations covered by filter class 2.

**B2:** Inorganic gases and vapours, e.g. chlorine, hydrogen sulfide and hydrogen cyanide, up to concentrations covered by filter class 2.

**P3:** Particles up to concentrations covered by filter class 3.

**OPERATIONAL LIFE**

Operational cannister life is the length of time a cannister has been used in a polluted environment. The maximum operational life of a canister is very difficult to predict and depends on the level of environmental contamination. In a casualty decontamination area, where levels of pollution will be fairly low, it will be hours (a figure of eight hours is often quoted anecdotally). That number will be much lower in areas of heavy contamination, such as the scene of a chemical spill.

*Staff should not be working in heavily contaminated areas with this type of respirator.*

As an example, the table below breaks down the typical performance of the Avon CBRNCF50 cannister against the gaseous agents specified in the NIOSH CBRN Air-Purifying Respirator standard, chemical warfare agents and industrial agents (toxic industrial chemicals). A variety of other cannisters and manufactures with different characteristics are available.
<table>
<thead>
<tr>
<th>Gaseous agents</th>
<th>Challenge concentration (ppm)</th>
<th>IDLH (ppm)</th>
<th>Protection time (min.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nerve agent</td>
<td>87</td>
<td>0.02</td>
<td>&gt;180</td>
</tr>
<tr>
<td>Hydrogen cyanide</td>
<td>940</td>
<td>50</td>
<td>&gt;100</td>
</tr>
<tr>
<td>Cyanogen chloride</td>
<td>300</td>
<td>Unknown</td>
<td>&gt;80</td>
</tr>
<tr>
<td>Ammonia</td>
<td>2,500</td>
<td>300</td>
<td>&gt;20</td>
</tr>
<tr>
<td>Cyclohexane</td>
<td>2,600</td>
<td>1,300</td>
<td>&gt;30</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>500</td>
<td>20</td>
<td>&gt;200</td>
</tr>
<tr>
<td>Hydrogen sulfide</td>
<td>1,000</td>
<td>100</td>
<td>&gt;100</td>
</tr>
<tr>
<td>Nitrogen dioxide</td>
<td>200</td>
<td>20</td>
<td>&gt;20</td>
</tr>
<tr>
<td>Phosgene</td>
<td>250</td>
<td>2</td>
<td>&gt;400</td>
</tr>
<tr>
<td>Phosphine</td>
<td>300</td>
<td>50</td>
<td>&gt;375</td>
</tr>
<tr>
<td>Sulfur dioxide</td>
<td>1,500</td>
<td>100</td>
<td>&gt;20</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Industrial agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloroacetophenone (CN)</td>
</tr>
<tr>
<td>Chlorobenzylidene (CS)</td>
</tr>
<tr>
<td>Hydrogen sulfide</td>
</tr>
<tr>
<td>Hydrogen fluoride</td>
</tr>
<tr>
<td>Hydrogen chloride</td>
</tr>
<tr>
<td>Methylamine</td>
</tr>
<tr>
<td>Chlorine</td>
</tr>
</tbody>
</table>

Table 15: Performance of the Avon CBRNCF50 cannister against chemical and biological agents (IDLH: concentration immediately dangerous to life or health).

Note that the protection time is indicated for standard laboratory test conditions. **These do not necessarily relate to actual use times.** Actual use times must be verified on the basis of a risk assessment of the likely hazards in the intended area of use.

PPE WORN IN CLINICAL AREAS

The standards for PPE worn in the clinical areas beyond the decontamination point are split into standard, droplet and aerosol precautions. See Annex C, p. 113.

CHEMICAL, RADIOLOGICAL AND NUCLEAR CASUALTIES

Chemical, radiological and nuclear casualties present a relatively small risk to staff working in the clinical areas. Standard or level D precautions are adequate.

PATIENTS WITH INFECTIOUS DISEASES

Patients with infectious diseases pose more of a threat to staff. Standard precautions alone may be inadequate. Extra care must be taken with organisms that spread by droplet or aerosol or that have a high mortality. As with all PPE selection, the choice is dependent both on the pathogen and on the activity to be undertaken. See the examples in the table below.

<table>
<thead>
<tr>
<th>Activity</th>
<th>Risk of exposure</th>
<th>PPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any activity with a safety distance from potentially contaminated people, e.g. triage centre</td>
<td>No direct exposure to potentially contaminated bodily fluids but presence of potentially contaminated people</td>
<td>![Image](M. Saeed/ICRC)</td>
</tr>
<tr>
<td>Contact with potentially contaminated people, e.g. medical examination</td>
<td>Risk of exposure to saliva and other potentially contaminated bodily fluids</td>
<td>![Image](J. Nehme/ICR)</td>
</tr>
<tr>
<td>Surgical procedure, e.g. caesarean section</td>
<td>Risk of exposure to large amounts of potentially contaminated bodily fluids</td>
<td><img src="Shutterstock" alt="Image" /></td>
</tr>
</tbody>
</table>

Table 16: Examples of PPE for protection against Ebola virus, selected according to activity
TRIAGE OF CASUALTIES

TRIAGE DEFINITION

Triage is the preliminary assessment of patients or casualties to determine the urgency of their need for treatment and the nature of treatment required. The purpose is to try to save as many as possible with the limited resources available. This annex will not discuss triage in detail, but it does raise a few points specific to CBRN/HAZMAT situations.

TRIAGE SYSTEMS

A triage system is a method of prioritizing patients based on the severity of their medical condition and the urgency of their need for medical attention. A triage sieve is a subset of a triage system that is used in mass casualty incidents or disasters. It is a rapid screening tool that can quickly identify patients who require immediate life-saving interventions and those who can be managed with delayed or minimal interventions. The goal of a triage sieve is to rapidly sort large numbers of patients into categories based on their need for medical attention so that limited medical resources can be allocated efficiently and effectively. There are many different triage sieves available, for both conventional and CBRN/HAZMAT incidents. For CBRN/HAZMAT incidents, triage sieves fall into two broad categories: physiological and functional.
**PHYSIOLOGICAL TRIAGE**

Physiology-based systems use parameters such as pulse rate, capillary refill, etc., to prioritize patients and assess their severity of injury. A wide range of systems have been described and they vary enormously in purpose and complexity. Most Trauma modern triage systems measure some physiological variables. An example is shown below in Table 5.

Physiology-based triage usually requires a brief review of injuries and measurement of whatever parameters the specific triage system requires. This may be difficult while wearing PPE.

<table>
<thead>
<tr>
<th>Category</th>
<th>Priority</th>
<th>Injuries / symptoms by agent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Chemical</td>
</tr>
<tr>
<td>1. Red</td>
<td>Immediate</td>
<td>Severe</td>
</tr>
<tr>
<td>2. Yellow</td>
<td>Delayed</td>
<td>Moderate</td>
</tr>
<tr>
<td>3. Green</td>
<td>Minimal</td>
<td>Mild</td>
</tr>
<tr>
<td>4. Blue or black (varies)</td>
<td>Expectant</td>
<td>Expectant</td>
</tr>
</tbody>
</table>

Table 17: Triage categories

---

FUNCTIONAL TRIAGE

Another way of assessing patients is by their level of function, i.e. what they can do. An example of this is the categorization system recommended by *PRISM: Primary Response Incident Scene Management, Vol. 2: Tactical Guidance.*

<table>
<thead>
<tr>
<th>Category</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>3. Green</strong></td>
<td>Patients who can understand instructions and perform activities without assistance</td>
</tr>
<tr>
<td><strong>2. Yellow</strong></td>
<td>Patients who are either unable to understand instructions or unable to perform activities without accommodations or assistance</td>
</tr>
<tr>
<td><strong>1. Red</strong></td>
<td>Patients who are unresponsive, have life-threatening injuries or require extensive accommodations or assistance</td>
</tr>
</tbody>
</table>

Table 18: Functional triage for decontamination

Functional triage is quick and easy but crude compared to physiological triage. It is not a good tool to grade the severity of injuries and is not as accurate as physiological triage at prioritizing patients for treatment or predicting outcomes.
TRIAGE POINTS

Following a CBRN/HAZMAT incident, casualties need to be triaged at two stages at least:
• prior to decontamination (dirty triage)
• prior to entry into the facility (clean triage).

Figure 69: Triage after a CBRN/HAZMAT incident

DIRTY TRIAGE

A casualty decontamination area is made up of a series of lanes; each lane is designated for patients with different levels of functioning (e.g. walking or on a stretcher). Dirty triage consists of directing patients to the appropriate lane.

Dirty triage will need to be carried out while wearing PPE. Working in this environment is difficult. Taking a pulse in butyl rubber gloves is very hard, stethoscopes cannot be used, and communication is difficult. Staff who can work in this kind of PPE are also limited. If available, cheap electrical devices such as pulse oximeters are extremely useful for rapid physiological assessment.
There is very little published data available about triage for decontamination. Which triage system is chosen will depend on local resources and systems.

**CLEAN TRIAGE**

Once patients are decontaminated, they are moved into the clean zone, where they are re-triaged. Patients arriving from clean areas enter the triage system at this point. Clean triage consists of assessing the severity of injuries and prioritizing patients for treatment.

Those performing the triage should ideally be experienced health-care providers with knowledge in CBRN/HAZMAT exposure; in their absence, the second-best option is staff trained to triage for priority of treatment and decontamination.

**EQUIPMENT**

In the decontamination area, it is recommended to limit medical interventions to a minimum during dirty triage, as outlined in Section 4.4 (p. 42). It is preferable to replace any equipment that cannot be easily decontaminated, although it may not be possible to change life-saving equipment such as tourniquets (see Section 4.4 for more information).

The same applies to triage tags. If possible, one tag should be applied at dirty triage, removed during decontamination and replaced with a new one during clean triage. Any treatment provided should be documented and transferred to the new triage tag. In case of a shortage of triage tags, patients can still be identified using alternative methods, such as tape, as shown below.

![Figure 70: Triage tags can be simply improvised (left) or more advanced (right)](image)

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ANNEX G: TRIAGE OF CASUALTIES

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K. Finsnes/ICRC
TRIAGE STAFF

Triage is typically performed by experienced health-care workers who have been trained in the process of triage. On the dirty side, it is essential to possess sound knowledge of how to use level C PPE. In some cases, there might not be enough adequately trained health-care workers to staff both clean and contaminated triage areas. In emergency situations, triage may be carried out by others who have been trained in the triage process. It may also be necessary to perform pure functional triage on the dirty side, followed by physiological triage on the clean side.

CHOOSING A TRIAGE SYSTEM

Specific triage systems have been developed to address certain toxic industrial chemicals and CBRN agents. Examples are advice for triaging those exposed to chlorine, hydrogen cyanide, mustard agents, nerve agents and phosgene published on Chemical Hazards Emergency Medical Management (CHEMM) website. Such specific advice presumes the agent is known and will mostly be useful for well-resourced and trained personnel or those based next to specific known, stationary hazards.

Triage is already chaotic and stressful, and the process should also account for the possibility of trauma in addition to signs or degree of poisoning. Standard national triage tools with familiar categories should be used for physiological triage and can, if needed, be adapted to include other indicators for contamination and poisoning. In general though, chemical agents affect the body in ways that conform to all-hazard triage approaches. Nerve agent exposure can lead to breathing problems, resulting in casualties being triaged as red. Blister agent exposure may cause less mobility and manifest symptoms, leading to most exposed individuals being triaged as yellow and some as red. For radiological exposures, the radiation triage category can be assigned after initial trauma triage and stabilization. The Radiation Emergency Medical Management (REMM) system can help determine the severity of radiation sickness.
If no national triage sieve is available, the following sieve is effective and easy both for trauma and HAZMAT/CBRN exposure.53

Modified CBRN triage sieve

**ANNEX G: TRIAGE OF CASUALTIES**

1. Where resources permit, resuscitation may be attempted on cases of witnessed respiratory arrest with early use of antidotes (example: antidote autoinjector or atropine for nerve agent toxicity).
2. The application of a tourniquet mandates T1 category remains in place.

**Figure 71: Adapted triage sieve**


54 Triage sieve used with permission from Surgeon Captain Steven Bland, Defence Medical Services (UK), Chair, CBRN Medical Working Group (North Atlantic Treaty Organization).
ANNEX H

FACTSHEETS

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• Hydrazines (p. 178)
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• Hydrochloric acid (p. 184)
• Hydrogen fluoride/hydrofluoric acid (p. 186)
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For an explanation of the chemical warning labels contained in these factsheets, see Chemical warning labels explained (p. 104).

**Warning:** All approaches listed in the factsheets presume first responders are wearing appropriate PPE. Please see Annex F (p. 143) for more details. PPE protects rescuers from exposure to the toxicant and from secondary contamination. The level of PPE should always correspond to the risk of exposure; the highest level of PPE (level A*) should be used for unknown hazards and for airborne toxicants with the potential for skin exposure to vapour.

* Level A PPE includes a self-contained breathing
AMMONIA

OVERVIEW
Ammonia is a clear, colourless gas and a clear, colourless liquid under pressure. Although ammonia gas is lighter than air, released compressed ammonia gas can react with moisture and form a visible ammonia fog, which is likely to remain low to the ground. Sudden release could be cold (icing), and hence heavier than when at room temperature. Ammonia undergoes an exothermic reaction with water, forming ammonium hydroxide, a toxic and corrosive alkaline solution. Concentrated ammonia is highly toxic owing to its corrosive effect on tissue.

DETECTING AND RECOGNIZING EXPOSURE
Ammonia has a characteristic smell and an olfactory threshold of 1 to 50 ppm, well below a hazardous concentration. The gas itself is colourless, but a fog would be visible.

Time to symptoms is short, within seconds or minutes, but respiratory symptoms can worsen over time.

Because the gas is water-soluble, symptoms involving the upper airways, nose and eyes occur fast and might prevent people from entering higher-concentration areas.

Ammonia is detected by all common toxic industrial chemical detectors.

CLINICAL SIGNS AND SYMPTOMS
Ammonia causes local irritation in low concentrations, and tissue damage and corrosive burn lesions in higher concentrations.

The main toxic symptoms involve the airways and breathing: cough, chest tightness, dyspnoea, sore throat, mucus oedema in airways and respiratory collapse.
Contact with the eyes may cause severe corrosive injury and/or significant spasmodic blinking (blepharospasm).

Contact with liquid ammonia could cause frostbite from vapourization.

**DECONTAMINATION**

Undress the patient, as their clothes will smell and be a source of exposure.

If they have only been exposed to gas: Decontamination is not necessary before treatment, and there is a low risk of cross-contamination. However, rinse irritated skin with water.

If the patient has been exposed to liquid ammonia: Decontaminate with copious amounts of lukewarm water.

**FIRST AID**

Remove the patient from the source of exposure and remove contaminated clothing – decontaminate the patient if necessary (see above).

For respiratory symptoms: Avoid strenuous activity, ensure unobstructed airway, give oxygen and support ventilation if needed (bag valve mask).

For eye symptoms: Rinse with water or saline (preferred) for at least 15 minutes.

For burns/ corrosive lesions: Rinse with water. Treat as burn lesions.

Get immediate medical attention!

**TREATMENT**

There are no specific antidotes.

Treatment is focused on respiratory symptoms: give inhaled bronchodilators and inhaled or IV steroids. Consider need for intubation (in cases of laryngeal oedema or respiratory collapse); consider positive pressure (via continuous positive airway pressure (CPAP), non-invasive ventilation (NIV) or mechanical ventilation).

Rinse and treat corrosive eye lesions.

Rinse and treat corrosive skin lesions. Look for frostbite.
ARSENIC

Excluding arsine gas (p. 170)

OVERVIEW
Arsenic compounds are found in certain industrial, commercial and pharmaceutical products (e.g. wood preservatives, herbicides, anticancer drugs). Inorganic arsenic is used in the production of nonferrous alloys, semiconductors and certain types of glass. As inorganic arsenic is abundant in the Earth’s crust, artesian water wells can be contaminated with arsenic.

Arsenic compounds are either organic or inorganic and contain arsenic in pentavalent (arsenate) or trivalent (arsenite) form.

There are various mechanisms of toxicity, including inhibition of enzymatic reactions, induction of oxidative stress, and alteration of gene expression and signal transduction.

Ingestion and inhalation of soluble arsenic compounds pose the greatest risk of acute poisoning. Inorganic arsenic dusts are irritant to the skin and mucous membranes. The toxicity of arsenic compounds depends on their valence state, chemical composition and solubility. Tri- and pentavalent arsenic are known human carcinogens.

DETECTING AND RECOGNIZING EXPOSURE
The diagnosis is mostly based on a history of exposure with typical multisystemic signs and symptoms (the arsenic toxidrome). Acute arsenic poisoning should be suspected in patients with abrupt onset of abdominal pain, nausea, vomiting, watery diarrhoea and hypotension, followed by delayed cardiac dysfunction, pancytopenia and peripheral neuropathy. Some arsenic compounds are radiopaque.

Analytical detection is done in urine: In the first two to three days after acute symptomatic exposure, urine concentrations are several thousand µg/24h (over 1,000 µg/L) and may take several weeks to return to normal (background) levels (under 70 µg/24h and under 50 µg/L).
CLINICAL SIGNS AND SYMPTOMS
The most common route of exposure is oral ingestion. A single massive dose produces multisystemic signs and symptoms over hours to weeks. Gastrointestinal symptoms occur minutes to hours after ingestion (haemorrhagic gastroenteritis with nausea, vomiting, abdominal pain and watery diarrhoea). Fluid losses may lead to hypovolemic shock. After a delay of one to six days, a secondary phase may ensue with congestive cardiomyopathy, pulmonary oedema (cardiogenic or non-cardiogenic), QT-prolongation and cardiac arrhythmias. Mental status may be normal, or there may be lethargy, agitation or delirium. Generalized seizures are rare. After one to five weeks, a sensorimotor axonal peripheral neuropathy may evolve, beginning with painful distal dysesthesias (in the feet), ascending weakness and paralysis. Pancytopenia (particularly leukopenia and anaemia) typically develops after one to two weeks. Dermatologic effects after one to six weeks include desquamation (of the palms and soles), diffuse maculopapular rash, periorbital oedema, herpes zoster and herpes simplex lesions, and Mees’ lines (transverse white striae in the nails).

DECONTAMINATION
Skin decontamination
Remove all clothing, jewellery, shoes, etc. Blot away any adherent liquid, and gently remove any solid particles with gauze or a microfibre towel. Then wash all body surfaces from head to toe with mild (liquid) detergent or soap and lukewarm water, protecting the patient’s airway. For water-soluble toxicants, water alone is sufficient. Pay close attention to exposed skin folds, axillae, genitals, feet and eyes (see below). Then dry the entire body surface with towels in order to remove desorbed toxicants and prevent hypothermia.

Eye decontamination
Flush the eyes with plenty of water, Ringer’s solution or isotonic saline. Do not forget to remove contact lenses! For continuous rinsing during patient transport, eye irrigation lenses are most efficient.

Gastrointestinal decontamination
For organic arsenic compounds, administer oral activated charcoal (1 g/kg) within one to two hours after ingestion; do not give activated charcoal for inorganic arsenic salts, which are poorly adsorbed on activated charcoal. Consider gastric lavage or whole-bowel irrigation when large quantities of an arsenic compound have been ingested.
FIRST AID (ABCs)
Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss. Treat coma, shock and arrhythmias. Avoid drugs that contribute to QT-prolongation. Continuous cardiac monitoring beyond 48 hours is warranted in patients with persistent symptoms.

TREATMENT
Enhanced elimination techniques are not effective. Haemodialysis is only of benefit in patients with concomitant renal failure.

Given sufficient evidence of arsenic exposure, promptly treat seriously symptomatic patients with chelating agents; do not wait for analytical laboratory confirmation.

Chelation therapy can be stopped when urinary arsenic levels are less than 50 µg/L over 24h.

Unithiol (Sodium 2,3-dimercapto-1-propanesulfonate, also known as DMPS)
- 3–5 mg/kg every 4 hr by slow IV (or IO) infusion (over 20 min.)

When the patient is haemodynamically stable and gastrointestinal symptoms have subsided, unithiol can be continued orally (4–8 mg/kg every 6 hr) or be replaced by oral succimer.

IO administration is equivalent to IV administration.

Dimercaprol (2,3-dimercaptopropanol, also known as British anti-lewisite or BAL)
- 3–5 mg/kg by deep intramuscular injection every 4–6 hr

Dimercaprol is the second-choice agent if unithiol is not immediately available.

Succimer (2,3-dimercaptosuccinic acid, also known as DMSA)
- 7.5 mg/kg every 6 hr orally, or 10 mg/kg every 8 hr
ARSINE GAS

OVERVIEW
Arsine gas is a colourless gas with a slight garlic odour. It forms when arsenic comes in contact with hydrogen or reducing agents. Exposure mostly occurs in industrial settings (smelter operations, semiconductor industry and recycling).

Arsine is a potent haemolytic agent. Massive haemolysis causes renal failure and decreases systemic oxygen delivery. Arsine is the most toxic form of arsenic.

DETECTING AND RECOGNIZING EXPOSURE
The diagnosis is mostly based on the arsine toxidrome: arsine poisoning should be suspected in patients with abrupt onset of haemolysis, haemoglobinuria and progressive oliguria.

Urine and whole-blood concentrations of arsenic may be elevated. In severe arsine poisoning, whole-blood levels may range from several hundred to several thousand µg/L.

Haematocrit will progressively decline 12 to 36 hours after exposure, with erythrocyte haemoglobin levels decrease to 5 to 10 g/dL. Free plasma haemoglobin and serum bilirubin is elevated. Urinalysis is haem-positive, with only very few erythrocytes on microscopy. With progressing oliguria, an active urinary sediment emerges, and serum creatinine will rise.

CLINICAL SIGNS AND SYMPTOMS
Arsine gas is not acutely irritating, and thus does not produce immediate symptoms; this increases the risk of prolonged exposure. After a latent period of two to 24 hours, massive haemolysis occurs along with early symptoms including malaise, headache, fever, chills and numbness or coldness of the extremities. There may be concomitant gastrointestinal complaints. With severe exposure, abrupt cardiovascular collapse and death may ensue within one to two hours. Haemoglobinuria can be detected, along with acute renal failure, one to three days after exposure. Some patients develop agitation and delirium.
PERSONAL PROTECTIVE EQUIPMENT (PPE) AND DECONTAMINATION

PPE
PPE protects rescuers from exposure to the toxicant and from secondary contamination. It should always be worn according to the risk of exposure; the highest level of PPE (level A*) should be used for unknown hazards and for airborne toxicants with the potential for skin exposure to vapour.

* Level A PPE includes a self-contained breathing apparatus with a vapour-protective, fully encapsulated chemical-resistant suit.

Decontamination
For exposure to gas, no decontamination of the skin or gastrointestinal tract is necessary. Removing the patient from the source of exposure is sufficient.

FIRST AID (ABCs)
Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss. Treat coma, shock and arrhythmias. Provide vigorous hydration and, if needed, osmotic diuresis with mannitol. Prompt exchange transfusion with whole blood is a key therapeutic intervention in patients with a free serum haemoglobin level over 1.5 g/dL and signs of renal insufficiency or early acute tubular necrosis. Haemodialysis may be needed for progressive renal failure.

TREATMENT
Enhanced elimination techniques are not effective. Haemodialysis is only of benefit in patients with concomitant renal failure.

Given sufficient evidence of arsine exposure, promptly treat seriously symptomatic patients with chelating agents; do not wait for analytical laboratory confirmation.

Dimercaprol (2,3-dimercaptopropanol, also known as British antilewisite or BAL)
- 3–5 mg/kg by deep intramuscular injection every 4–6 hr

Dimercaprol is the primary choice agent during the first 24 hours. After 24 hours, treatment can be switched to oral unithiol or succimer.
**Unithiol (2,3-dimercaptopropanesulfonic acid, also known as DMPS)**

- 3–5 mg/kg every 4 hr by slow IV (or IO) infusion (over 20 min.)

When the patient is haemodynamically stable and gastrointestinal symptoms have subsided, unithiol can be continued orally (4–8 mg/kg every 6 hr) or be replaced by oral succimer.

IO administration is equivalent to IV administration.

**Succimer (2,3-dimercaptosuccinic acid, also known as DMSA)**

- 7.5 mg/kg orally every 6 hr, or 10 mg/kg every 8 hr
CHLORINE

OVERVIEW
Chlorine is a highly corrosive gas with intermediate water solubility. It is a pulmonary irritant, causing acute damage in the upper and lower respiratory tract. At room temperature, it is a yellow–greenish gas that is denser than air (relative vapour density: 2.5) and has a characteristic bleach or swimming-pool smell. It is widely used as an industrial chemical, but it has also been used as a chemical warfare choking agent. Chlorine is transported as a pressurized or cold liquid (boiling point: –34°C).

DETECTING AND RECOGNIZING EXPOSURE
Chlorine has a characteristic bleach/swimming-pool smell, and its olfactory threshold is 0.02 to 0.5 ppm, much lower than any hazardous concentration. The gas is greenish-yellow and tend to remain low to the ground.

Time to symptoms is short, within seconds or minutes, but symptoms can worsen over time. Chlorine is detected by all common toxic industrial chemical detector.

CLINICAL SIGNS AND SYMPTOMS
Chlorine causes local irritation in low concentrations, and tissue damage and corrosive burn lesions in higher concentrations.

The main toxic symptoms involve the airways and breathing: cough, chest tightness, dyspnoea, sore throat, mucus oedema in airways and respiratory collapse.

Contact with the eyes may cause severe corrosive injury and/or significant spasmodic blinking (blepharospasm).

Contact with liquid chlorine could cause frostbite from vapourization.
**DECONTAMINATION**
Undress the patient, as their clothes will smell and be a source of exposure.

If they have only been exposed to gas: Decontamination is **not necessary** before treatment. The risk of cross-contamination is low. However, rinse irritated skin with water.

If they have been exposed liquid chlorine: Decontaminate with copious amounts of lukewarm water.

**FIRST AID**
Remove the patient from the source of exposure and remove contaminated clothing – decontaminate the patient if necessary (see above).

*For respiratory symptoms:* Avoid strenuous activity, ensure unobstructed airway, give oxygen and support ventilation if needed (with a bag valve mask).

*For eye symptoms:* Rinse with water or saline (preferable) for at least 15 minutes.

For burns/corrosive lesions: Rinse with water. Treat as burn lesions.

Get immediate medical attention!

**TREATMENT**
There are no specific antidotes.

Treatment is focused on respiratory symptoms: give inhaled bronchodilators and inhaled or IV steroids. Consider need for intubation (in cases of laryngeal oedema or respiratory collapse); consider positive pressure (via continuous positive airway pressure (CPAP), non-invasive ventilation (NIV) or mechanical ventilation). Any pulmonary oedema is primarily toxic, not cardiogenic, and must be treated accordingly.

Rinse and treat corrosive eye lesions and skin lesions. Look for frostbite.
CHOLINESTERASE-INHIBITOR-TYPE INSECTICIDES
EXAMPLE: ORGANOPHOSPHATES AND CARBAMATES

OVERVIEW
Cholinesterase-inhibitor-type insecticides typically belong to the chemical groups of organophosphorus or carbamate compounds. Exposure occurs mainly in the occupational setting (agriculture), although domestic exposure occurs. The route of exposure is either dermal (typical in accidental poisoning) or oral ingestion (typical in suicide attempts).

Pure organophosphates or carbamates are solids or liquids at room temperature and are either insoluble or only slightly soluble in water. Therefore, commercial preparations contain organic solvents. Most compounds have low vapour pressure, excluding significant inhalation effects.

The mechanism of toxicity is via inhibition of the enzyme acetylcholinesterase (EC 3.1.1.7) in neuronal synapses, which leads to local accumulation of the neurotransmitter acetylcholine with continuous overshooting cholinergic neurotransmission in the central and peripheral (autonomic) nervous systems.

Nerve agents used as warfare agents belong to the same chemical group and share the mechanism of toxicity (thus producing the same cholinergic toxidrome), but inhalation is a more likely route of exposure because of their higher vapour pressure.

DETECTING AND RECOGNIZING EXPOSURE
The pure substances have only poor to fair warning properties (unless dissolved in organic solvents with a typical smell), and skin irritation is minimal after dermal exposure, so diagnosis is based on the appearance of the typical cholinergic toxidrome.

Analytical detection of the toxicants is not widely available, so laboratory diagnosis is based on low acetylcholinesterase activity (red blood cells) or butyrylcholinesterase activity (serum).
CLINICAL SIGNS AND SYMPTOMS
The characteristic cholinergic toxidrome includes muscarinic and nicotinic symptoms (at the muscarinic and nicotinic receptors, respectively). Muscarinic symptoms are diarrhoea, urination, miosis, bronchospasm, bradycardia, emesis and hypersecretion (bronchorrhoea, lacrimation, salivation, sweating), while nicotinic symptoms are mydriasis, tachycardia, muscular weakness, hypertension and fasciculations. The central nervous system symptoms are confusion, convulsions and coma. Time to onset of symptoms depends on dose and route of exposure (shortest to longest time of onset: inhalative < oral < dermal).

DECONTAMINATION
Skin decontamination
Remove all clothing, jewellery, shoes, etc. Blot away any adherent liquid and gently remove any solid particles with gauze or a microfibre towel. Then wash all body surfaces from head to toe with mild (liquid) detergent or soap and lukewarm water, protecting the patient’s airway. For water-soluble toxicants, water alone is sufficient. Pay close attention to exposed skin folds, axillae, genitals, feet and eyes (see below). Then dry the entire body surface with towels in order to remove desorbed toxicants and prevent hypothermia.

Eye decontamination
Flush the eyes with plenty of water, Ringer’s solution or isotonic saline. Do not forget to remove contact lenses! For continuous rinsing during patient transport, eye irrigation lenses are most efficient.

Gastrointestinal decontamination
Administer oral activated charcoal (1 g/kg) within one to two hours after ingestion. Consider gastric lavage or whole-bowel irrigation when large quantities have been ingested.

FIRST AID (ABCs)
Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss. Treat coma, shock, seizures and arrhythmias. Begin with antidotal treatment (see below) if cholinergic signs are present.
**TREATMENT**

In addition to continued supportive treatment, antidotal treatment is the mainstay of therapy. Treat seizures with **IV** or **IO** benzodiazepines (diazepam, lorazepam or midazolam). Atropine is the symptomatic antidote for organophosphate and carbamate poisoning. Oximes are the curative antidotes for organophosphate poisoning but are typically not needed for carbamate poisoning because cholinesterase enzymes that have been inhibited by car bamates tend to recover their function naturally (known as spontaneous reactivation). Oximes may nevertheless reduce the severity of symptoms in carbamate poisoning. Poisoning may result from a mix of insecticides.

**Atropine**

Atropine is the symptomatic antidote for the muscarinic signs and symptoms of cholinergic poisoning with cholinesterase inhibitors. It blocks cholinergic neurotransmission at muscarinic (but not nicotinic!) receptors.

Initial dose is 0.5 to 2.0 mg IV or IO*, doubling this dose every five minutes until bronchorrhoea, bronchospasm and bradycardia resolve (referred to as “atropinization”). Follow this with a continuous infusion of 20% of the total atropinization dose per hour. Large amounts of atropine may be needed in severe poisoning.

**Oximes (obidoxime, pralidoxime)**

Oximes reactivate acetylcholinesterase (which is blocked by organophosphates but not car bamates), thus restoring normal neurotransmission. However, reactivation is only feasible as long as ageing of the enzyme–organophosphate complex has not occurred yet (i.e., as long as the toxicant has not bonded irreversibly with the enzyme – this does not occur with car bamates). The half-life of ageing is substance–specific (hours to days).

Obidoxime dosage: loading dose is 250 mg IV or IO* with slow injection, followed by 750 mg/24h as a continuous infusion, so long as reactivation is achievable (no ageing has occurred yet), indicated by an improvement in symptoms after administration.

Pralidoxime dosage: give 1 to 2 g IV or IO* over 15 to 30 minutes; this dose can be repeated if weakness or fasciculations have not resolved after one hour.

* IO administration is equivalent to IV administration.
HYDRAZINES

OVERVIEW
Hydrazines are polar and strongly alkali liquids that are clear, colourless, fuming and hygroscopic, with a characteristic ammonia-like odour (odour threshold of 1.7 to 8.0 ppm). They are used as potent rocket and military jet fuel and can be found in a wide variety of industrial manufacturing.

Hydrazines include the parent compound, hydrazine, and its methylated derivatives, such as monomethyl hydrazine (MMH) and 1,1-dimethyl hydrazine (also referred to as “heptyl” by Russian aerospace scientists). MMH is the toxic principal of the Gyromitra mushroom genus.

Mechanisms of toxicity include the corrosive effect, hepatotoxicity and neurotoxicity via inhibition of the enzymes glutamic acid decarboxylase and pyridoxal kinase, which results in generalized seizures owing to a lack of the inhibitory neurotransmitter gamma amino butyric acid (GABA). Moreover, hydrazines are potential human carcinogens.

Exposure is most likely to occur in the occupational setting by skin contact or inhalation; ingestion is unusual but possible.

DETECTING AND RECOGNIZING EXPOSURE
The diagnosis is circumstantial and based on the characteristic combination of caustic and neurotoxic effects, liquefactive necrosis of the skin and mucous membranes, and intractable seizures that are refractory to standard treatment (i.e. benzodiazepines and antiepileptic drugs). Analytical laboratory tests for confirmation of exposure are not routinely available.

CLINICAL SIGNS AND SYMPTOMS
Hydrazines are acutely corrosive, producing immediate signs of tissue damage and leading to reddening (erythema), burning pain, swelling and liquefactive necrosis at the site of contact. Neurotoxic manifestations are agitation, seizures, coma and death.
PERSONAL PROTECTIVE EQUIPMENT (PPE) AND DECONTAMINATION

After contact, the toxic effects occur within minutes. As the extent of damage depends on concentration and the duration of exposure, decontamination is of paramount importance.

PPE
PPE protects rescuers from exposure to the toxicant and from secondary contamination. It should always be worn according to the risk of exposure; the highest level of PPE (level A*) should be used for unknown hazards and for airborne toxicants with the potential for skin exposure to vapour.

* Level A PPE includes a self-contained breathing apparatus with a vapour-protective, fully encapsulated chemical-resistant suit.

Skin decontamination
Remove all clothing, jewellery, shoes, etc. Blot away any adherent liquid, and gently remove any solid particles with gauze or a microfibre towel. Then wash affected body surfaces from head to toe with lukewarm water, protecting the patient’s airway. Pay close attention to exposed skin folds, axillae, genitals, feet and eyes (see below). Dry the entire body surface with towels in order to remove desorbed toxicants and prevent hypothermia.

Eye decontamination
Flush the eyes with plenty of water, Ringer’s solution or isotonic saline. Do not forget to remove contact lenses! For continuous rinsing during patient transport, eye irrigation lenses are most efficient.

Gastrointestinal decontamination
Although it is not known if hydrazines are adsorbed to activated charcoal, administration is recommended. Cleanse the oesophagus by giving a few sips of water, but only immediately after exposure. Avoid large quantities of water in order to prevent vomiting, which would repeatedly expose the oesophageal mucosa to the caustic effect. If the patient has ingested large quantities, consider removal from the stomach by endoscopy or a soft nasogastric tube.
FIRST AID (ABCs)
After decontamination, management is analogous to that of burn lesions.

Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss. Treat coma, shock and arrhythmias.

The antidote for seizures not responding to standard anticonvulsive treatment (i.e. benzodiazepines and antiepileptic drugs) is pyridoxine (Vitamin B6): give 25 to 70 mg/kg IV (or IO) over five minutes, up to a maximum of 5 g per dose.

IO administration is equivalent to IV administration.

TREATMENT
Enhanced elimination techniques are not effective.

After decontamination, management of caustic lesions is analogous to that of burn lesions.
HYDROCARBONS

OVERVIEW
Hydrocarbons are a heterogeneous group of chemicals. According to their chemical structure, they are classified as aliphatic, aromatic or substituted. Within each group, they can be gaseous, liquid or solid at normal temperature and pressure. Accordingly, the most likely route of exposure is inhalation, followed by dermal contact and, less frequently, oral ingestion. Most hydrocarbons are lipophilic and are not miscible with water. They are typically mixtures, not pure chemical substances, which is reflected in their boiling-point ranges.

DETECTING AND RECOGNIZING EXPOSURE
Given the high number of possible substances, recognition of exposure is circumstantial.

Most hydrocarbons have a characteristic odour and an oily appearance.

Time to symptoms varies widely for different substances.

Analytical laboratory tests to confirm exposure are not routinely available.

For aspiration pneumonitis, arterial blood gas analysis and chest X-ray are helpful.

For suspected systemic toxicity, electrolytes, glucose, blood urea nitrogen, creatinine and liver transaminases should be obtained, and electrocardiogram monitoring performed.

CLINICAL SIGNS AND SYMPTOMS
Hydrocarbon gases and vapours are simple asphyxiants leading to hypoxia followed by tachypnoea and central nervous system (CNS) excitation with agitation, followed by CNS depression and possibly respiratory arrest. Inhalation of longer-chain hydrocarbons (containing more than four carbon molecules) or tracheobronchial aspiration of low-viscosity hydrocarbons can lead to chemical pneumonitis.

Hypoxia can cause headache, dizziness, weakness, confusion, agitation, convulsions and coma, eventually leading to death.
More generally, hydrocarbons lower the heart’s threshold for ventricular dysrhythmias due to catecholamines.

They are general anaesthetics causing sedation and coma.

They are also irritants to skin and mucous membranes and can cause defatting dermatitis and chemical burns.

Some hydrocarbons cause specific toxic effects:
- aniline – hepatotoxicity, methaemoglobinaemia
- phenol – painless, depigmented (pale) chemical burns to the skin
- dinitrophenol, pentachlorophenol – uncoupling oxidative phosphorylation
- carbon tetrachloride (with chronic exposure) – hepatotoxicity
- dichloromethane (i.e. methylene chloride) – carbon monoxide poisoning.

**DECONTAMINATION**
Remove the patient from the source of exposure.

For exposure to gaseous hydrocarbons, no decontamination is necessary.

For exposure to liquid or solid hydrocarbons, remove clothing and wash the skin with lukewarm water and soap or a mild liquid detergent. Pay close attention to skin folds, axillae, genitals and feet. After ocular exposure, rinse the eyes continuously (at least 20 to 30 minutes) with copious amounts of water, Ringer’s solution or normal saline. Irrigations lenses are helpful for this purpose.

For oral ingestion of hydrocarbons, consider aspirating the liquid from the stomach via a nasogastric tube and administering activated charcoal.

**FIRST AID (ABCs)**
Ensure open airway, adequate ventilation and oxygenation. Monitor for dysrhythmias.

**TREATMENT**
There are no specific antidotes for hydrocarbon toxicity.

Give high-flow oxygen (at least 15 litres per minute via a non-rebreather reservoir mask).
For inadequate spontaneous ventilation or uncontrolled airway, intubate and ventilate.

For significant ventricular irritability, consider IV or IO* betablockers.

Treat seizures with parenteral benzodiazepines (diazepam, lorazepam, midazolam).

For aniline-induced methaemoglobinaemia, give methylene blue: 1 to 2 mg/kg IV or IO* slowly over five minutes; can be repeated after 30 to 60 minutes. If no response after two doses, do not repeat again.

* IO administration is equivalent to IV administration.
HYDROCHLORIC ACID

OVERVIEW
Hydrochloric acid (also known as muriatic acid) is the aqueous solution of hydrogen chloride gas. Hydrochloric acid is a strong irritant and corrosive agent, depending on its concentration. Concentrated hydrochloric acid has a pungent smell.

It is used as scale remover and for metal cleaning, ore reduction, rubber production and chemical synthesis, among many others. Exposure occurs mainly in industrial or commercial activities.

The anion (chloride) is not toxic.

DETECTING AND RECOGNIZING EXPOSURE
The diagnosis is based on the rapid caustic effect at the contact site (skin, mucous membranes, airways, eyes). As with all strong acids, it causes a coagulation-type necrosis of organic tissues by protein denaturation, thus preventing to some extent a rapid and deep penetration.

After ingestion, diagnostic/prognostic endoscopy (to determine the damage) should be performed within 12 hours but not later than 24 hours afterwards – during this time interval, the risk of iatrogenic perforation is minimal.

CLINICAL SIGNS AND SYMPTOMS
Hydrochloric acid is acutely irritating, producing immediate signs of tissue damage and leading to reddening (erythema), burning pain, swelling and necrosis at the site of contact.

DECONTAMINATION
After contact, the damage occurs within minutes. As the extent of damage depends on the concentration and the duration of exposure, immediate decontamination is of paramount importance.

Skin decontamination
Remove all clothing, jewellery, shoes, etc. Wash affected body surfaces from head to toe with copious amounts of lukewarm water, protecting the patient’s airway. Pay close attention to exposed skin folds, axillae, genitals, feet and eyes (see below). Then dry the entire body surface with towels in order to remove desorbed toxicants and prevent hypothermia.
Eye decontamination
Flush the eyes with plenty of water, Ringer’s solution or isotonic saline. Do not forget to remove contact lenses! For continuous rinsing during patient transport, eye irrigation lenses are most efficient.

Gastrointestinal decontamination
Hydrochloric acid is not adsorbed to activated charcoal, so the latter is not therapeutically effective. Cleanse the oesophagus by giving a few sips of water, but do so only immediately after exposure. Avoid large quantities of water in order to prevent vomiting, which would repeatedly expose the oesophageal mucosa to the caustic effect. After ingestion of large quantities of hydrochloric acid, consider removal from the stomach by endoscopy or a soft nasogastric tube.

FIRST AID (ABCs)
After decontamination, management is analogous to that of burn lesions.

TREATMENT
Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss.

Treat coma, shock and arrhythmias.

There are no antidotes, and enhanced elimination techniques are not effective.
HYDROGEN FLUORIDE AND HYDROFLUORIC ACID

OVERVIEW
Hydrogen fluoride is a colourless fuming liquid below 19.4°C, or a colourless gas. Hydrogen fluoride combined with water becomes hydrofluoric acid. Hydrofluoric acid that is more than 40% hydrogen fluoride fumes in air. It is considered a weak acid but is still extremely harmful owing to its ability to penetrate tissue. Both the gas and acid pose significant toxicity with deep corrosive lesions and systemic toxicity.

DETECTING AND RECOGNIZING EXPOSURE
Hydrofluoric acid is a colourless liquid that in low concentrations is visually indistinguishable from water. Vapours from hydrogen fluoride and hydrofluoric acid may collect and stay in poorly ventilated, low-lying or confined areas (e.g. sewers, basements and tanks). Odour does not provide sufficient warning of exposure.

CLINICAL SIGNS AND SYMPTOMS
Time to symptoms can be delayed by hours – do not underestimate severity because symptoms immediately after exposure are scarce! The primary corrosive effect on skin, eyes and mucus membranes is caused by the acid. The secondary effect, with deep tissue damage, is caused by dissociated fluoride ions forming salts with tissue cations (calcium and magnesium). Skin exposure causes painful erythema (redness) that may evolve to blisters; deeper damage results in white or pale skin. Respiratory symptoms include cough, dyspnoea, pulmonary oedema and haemoptysis. Eye symptoms include painful corrosive injuries. Systemic toxicity includes electrolyte disturbances, cardiac dysrhythmias, central nervous system depression and convulsions.

DECONTAMINATION
Remove clothes immediately. Decontaminate the patient with copious amounts of lukewarm water.
**FIRST AID**

Remove the patient from the source of exposure, and remove contaminated clothing. Decontaminate the patient with water.

*For eye symptoms:* Rinse eyes with water or 0.9% saline (preferable) for at least 15 minutes.

*For corrosive lesions:* Immerse in or soak with compounds of quaternary ammonium (e.g. 0.13% benzalkonium chloride) if available. If quaternary ammonium is not available, use calcium gluconate in 2.5 to 33% concentrations.

*For respiratory symptoms:* Avoid strenuous activity. Ensure airway is unobstructed. Give oxygen, and support ventilation if needed with a bag valve mask.

*If ingested:* Give a glass milk or water to drink.

Get immediate medical attention!

**TREATMENT**

*For skin and deep tissue damage:* Use a topical gel or mixture with calcium gluconate. Treatment for severe local lesions includes subcutaneous injections with 10% calcium gluconate or intraarterial injection with 2% calcium gluconate – seek advice on dosing and treatment options.

*For inhalation injury:* Treatment includes positive end-expiratory pressure (PEEP), intubation and mechanical ventilation, bronchodilation therapy and steroids. Nebulization of 4 ml of 2.5% calcium gluconate can be repeated.

*If ingested, and to prevent systemic effects:* Give up to 6 g of calcium gluconate tablets.

*For systemic effects:* Correct hypocalcaemia and hypomagnesaemia by IV. Dialysis can eliminate fluorides from circulation. Seek advice for advanced medical treatment.
LUNG-DAMAGING AGENTS

OVERVIEW
In contrast to highly water-soluble irritant gases (such as hydrochloric acid or ammonia), lung-damaging agents are inhaled toxins of low water solubility that readily enter the lower airways without many acute effects, producing delayed alveolar damage. Typical agents include phosgene (carbonyl dichloride, see p. 196) or nitrogen oxides (NOx, see p. 192). They cause injury through free radical generation and acid formation (hydrochloric acid, nitric acid), leading to toxic alveolitis and acute respiratory distress syndrome.

Phosgene was originally developed as a chemical weapon. It is now used in the manufacture of dyes, resins and pesticides. It is also produced by incinerating chlorinated organic compounds or when cleaning welded metal with chlorinated solvents.

Nitrogen oxides (e.g. nitric oxide and nitrogen dioxide) are gases released from nitrous or nitric acid, from reactions between nitric acid and organic materials, from incineration of, for example, nitrocellulose, as a by-product of detonations, and as a breakdown product of rocket fuels. It is an air pollutant from engine exhaust. Occupational exposure occurs in electric arc welding, electroplating, engraving and silo storage of high-nitrite grain.

DETECTING AND RECOGNIZING EXPOSURE
Recognition of exposure is circumstantial. There are no useful analytical tests. Concomitant methaemoglobinaemia may be a consequence of nitrogen oxide inhalation.

CLINICAL SIGNS AND SYMPTOMS
Lung-damaging agents cause delayed-onset alveolar damage, with dyspnoea and hypoxaemia*: the delay is inversely proportional to the agent’s concentration in inhaled air and the duration of exposure.

*Hypoxia can cause headache, dizziness, weakness, confusion, agitation, convulsions and coma, eventually leading to death.
**DECONTAMINATION**
Remove the patient from the source of exposure as quickly as possible. External or internal decontamination do not apply.

**FIRST AID (ABCs)**
Remove the patient from the source of exposure as quickly as possible.

**TREATMENT**
Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss. Treat coma, shock and arrhythmias. Treatment is for non-cardiogenic pulmonary oedema and acute respiratory distress syndrome.

Enhanced elimination techniques are not effective.

There are no antidotes (except methylene blue for methaemoglobinaemia). Inhalation of corticosteroids is controversial.
NERVE AGENTS

Muscarinic effect
*Counteracted by oximes

M Mydriasis
T Tachycardia
W Weakness
H Hypertension
F Fasciculations

Nicotinic effect
*Counteracted by atropine

D Diarrhoea
U Urination
M Miosis
B Bronchospasm
E Emesis
L Lacrimation
S Salivation
Secretions
Sweating

Convulsion, confusion, coma

Treatment for nerve agents is antidotes and:

A: Oxygenation, continual suctioning, endotracheal intubation if necessary
B: 15 L/min O2 via non-rebreather reservoir mask
C: IV isotonic, blood sample, cardiac monitor, when necessary control haemorrhage
D: Glasgow Coma Scale
E: Proper decontamination, hypothermia prevention

Mild/moderate symptoms include localized sweating, muscle fasciculation, nausea/vomiting, weakness and shortness of breath

Severe symptoms include unconsciousness and shortness of breath or apnoea
<table>
<thead>
<tr>
<th>Age</th>
<th>Mild/moderate symptoms</th>
<th>Severe symptoms</th>
<th>Other treatment</th>
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| Infant (> 2 yr) | **Atropine** 0.05 mg/kg IM  
**Pralidoxime** 20–40 mg/kg IV | **Atropine** 0.1 mg/kg IM  
**Pralidoxime** 25–50 mg/kg IV | Assisted ventilation should be started after administration of antidotes where needed for severe exposure.  
**Repeat atropine** (2 mg IM/IV) at five 10-min. intervals until secretions have diminished and breathing is comfortable or airway resistance has returned to near-normal.  
**Repeat pralidoxime** for severe poisoning: second dose is equivalent to first dose after 1 hr if muscle weakness is still present; can be repeated again later if needed.  
**Diazepam**  
Adult: 5–10 mg IV  
Paediatric: 0.1–0.2 mg/kg IV |
| Child (2–10 yr) | **Atropine** 1 mg IM/IV  
**Pralidoxime** 20–40 mg/kg IV | **Atropine** 2 mg IM/IV  
**Pralidoxime** 25–50 mg/kg IV | |
| Adult | **Atropine** 2–4 mg IM/IV  
**Pralidoxime** 1–2 g IV over 30 min. | **Atropine** 6 mg IM/IV  
**Pralidoxime** 1–2 g IV over 30 min. | |
| Elderly, frail | **Atropine** 1 mg IM/IV  
**Pralidoxime** 1–2 g IV over 30 min. | **Atropine** 2–4 mg IM  
**Pralidoxime** 1–2 g IV over 30 min. | |

NITRIC ACID

OVERVIEW
Nitric acid is a strong, corrosive acid and a powerful oxidizer. It dissolves in water. Its boiling point is 83 to 122°C. The vapour may be invisible and is heavier than air. It spreads along the ground and may enter sewers and basements. When nitric acid boils in light, nitrogen dioxide (NO2), a yellow-red gas, and other nitrogen oxides are formed (see factsheet on lung-damaging agents, p. 188). Nitrogen oxides (NOx gases) produce reactive nitrogen and reactive oxygen species, which cause bronchopulmonary toxicity. Hence, nitric acid can be toxic to the skin, eyes and airway both as a direct liquid corrosive and from vapour and NOx gases.

DETECTING AND RECOGNIZING EXPOSURE
The liquid is colourless. Pure vapour may be invisible but can turn yellow-red-brownish in reaction. The vapour is heavier than air. Nitric acid’s olfactory threshold is 0.3 to 1 ppm, but the odour is unspecific. NO2 gas has a chlorine-like odour. As a powerful corrosive, time to symptom is short, but respiratory toxicity mediated by NOx gases might be delayed. NOx gases are detected by all common toxic industrial chemical detectors.

CLINICAL SIGNS AND SYMPTOMS
Signs and symptoms related to the airways and breathing are cough, chest tightness, dyspnoea, sore throat, mucus oedema in airways and respiratory collapse. Be aware that respiratory symptoms also can occur late.

Nitric acid causes corrosive lesions of the skin.

Contact with the eyes may cause severe corrosive injury and/or significant spasmodic blinking (blepharospasm).
DECONTAMINATION
If the patient has been exposed liquid nitric acid: Remove clothes immediately. Decontaminate with copious amounts of lukewarm water.

If they have only been exposed to gas (dry clothes, skin unaffected): Decontamination is not necessary before treatment. The risk of cross-contamination is low. Rinse irritated skin with water. Rinse irritated eyes (see below).

FIRST AID
Remove the patient from the source of exposure. Remove contaminated clothing immediately and douse affected skin with plenty of water.

For respiratory toxicity: Avoid strenuous activity — even if no symptoms are present. Ensure unobstructed airway, give oxygen and support ventilation if needed (bag valve mask).

For eye symptoms: Rinse eyes with water or 0.9% saline (preferred) for at least 15 minutes.

For corrosive lesions: Rinse with water. Treat as burn lesions.

Get immediate medical attention!

TREATMENT
There are no specific antidotes.

Treatment is focused on respiratory symptoms: give inhaled bronchodilators and inhaled or IV steroids. Consider need for intubation (in cases of laryngeal oedema or respiratory collapse); consider positive pressure (via continuous positive airway pressure (CPAP), non-invasive ventilation (NIV) or mechanical ventilation). Any pulmonary oedema is primarily toxic, not cardiogenic, and must be treated accordingly.

Rinse and treat corrosive eye lesions.

Rinse and treat burns and corrosive skin lesions.
**OPIOIDS**

**OVERVIEW**

Opiates are natural opium-derived substances. Opioids are all natural, synthetic or semi-synthetic chemicals that activate the opiate receptors, of which μ- and κ-receptors in the central nervous system (CNS) are the most important ones.

Medically, the most significant effects of opioids are CNS depression (coma, respiratory depression and analgesia). Their clinical effect is determined by their pharmacokinetic properties, receptor affinities and potency.

Common routes of exposure include oral ingestion and parenteral injection. Under certain circumstances and for certain substances, inhalation or dermal absorption may occur.

**DETECTING AND RECOGNIZING EXPOSURE**

The diagnosis is mainly based on the opioid toxidrome (see below). Most, but not all, opioids are readily detectable by urine enzyme-linked immunosorbent assay (ELISA).

**CLINICAL SIGNS AND SYMPTOMS**

The opioid toxidrome consists of impaired consciousness (from somnolence to coma), respiratory depression (hypoventilation, hypercapnia, hypoxia,* respiratory arrest), miosis (pinpoint pupils) and bowel hypomotility. Some agents (e.g. tramadol) may cause seizures.

*Although hypoxia can cause headache, dizziness, weakness, confusion, agitation and convulsions, the CNS depression induced by opiates/opioids suppresses most of these symptoms.

**DECONTAMINATION**

In case of gaseous or oral exposure, no skin decontamination is necessary. In case of skin exposure to liquid agents, skin decontamination is required, to both stop absorption and prevent secondary contamination of rescuers and caregivers.
FIRST AID (ABCs)
Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss. Treat coma, shock and arrhythmias. In patients with life-threatening respiratory depression, naloxone (IV, intraosseous or intranasal) is the antidote of choice.

TREATMENT
The focus of treatment is respiratory depression. The patient can be mechanically ventilated or treated with the antidote naloxone.

Enhanced elimination techniques are not effective. Haemodialysis is only of benefit in patients with concomitant renal failure.

Treat patients with serious opioid-induced respiratory depression with naloxone if mechanical ventilation is not feasible. Do not wait for analytical laboratory confirmation.

Naloxone is a parenteral opiate-receptor antagonist without intrinsic effect. Its main adverse effect is withdrawal in opioid-dependent individuals who have tolerance from chronic use. How much is needed depends on the differences of potency and receptor affinity between naloxone and the opioid in their system. Naloxone must be repeatedly administered until opioid effects are no longer detectable.

Administer 0.4 to 2.0 mg IV/IN; repeat every 2 to 3 min. until the desired response is achieved. If no response is achieved by a total dose of 10 to 15 mg, the diagnosis of opioid poisoning should be questioned. Sedation can recur when the naloxone wears off, in 0.5 to 2 hr. Repeated doses may be required to continue counteracting the opioid’s effects. In these cases, a continuous infusion of naloxone may be recommended; give approximately two-thirds of the initial dose needed to awaken the patient per hour.
PHOSGENE

OVERVIEW
Phosgene is a highly reactive gas and pulmonary irritant that causes acute damage to the upper and lower respiratory tract. It is sparingly soluble in water, in which it decomposes to hydrochloric acid and carbon dioxide. Phosgene is a clear gas at room temperature, with a higher density than air (relative vapour density of 3.3), and a characteristic smell of freshly cut grass or hay. It is widely used as an industrial chemical, but it has also been used as a chemical warfare choking agent. Phosgene is transported as a pressurized gas or cold liquid (boiling point of −8.3°C).

DETECTING AND RECOGNIZING EXPOSURE
Phosgene has a characteristic smell of cut grass; its olfactory threshold is 0.4 ppm, which is four times higher than its reported hazardous concentration. The gas is colourless and tends to remain low to the ground.

Time to symptoms is short, within seconds or minutes, but symptoms can worsen over time. Phosgene is detected by all common toxic industrial chemical detectors.

CLINICAL SIGNS AND SYMPTOMS
Phosgene causes local irritation in low concentrations, and tissue damage and corrosive lesions in higher concentrations of liquid phosgene.

The main toxic symptoms relate to airways and breathing: cough, chest tightness, dyspnoea, sore throat, mucus oedema in airways, respiratory collapse.

Contact with the eyes may cause severe corrosive injury and/or significant spasmodic blinking (blepharospasm).

Contact with liquid could cause frostbite from vapourization.
DECONTAMINATION
Undress the patient.

If they have only been exposed to gas: Decontamination is not necessary before treatment, and the risk of cross-contamination is low. However, rinse irritated skin with water.
If they have been exposed to liquid phosgene: Decontaminate with copious amounts of lukewarm water.

FIRST AID
Remove the patient from the source of exposure and remove contaminated clothing – decontaminate the patient if necessary (see above).

For respiratory symptoms: avoid strenuous activity, ensure unobstructed airway, give oxygen and support ventilation if needed (bag valve mask).

For eye symptoms: rinse with water or saline (preferred) for at least 15 minutes.

For burns or corrosive lesions: rinse with water and treat as burn lesions.

Get immediate medical attention!

TREATMENT
There are no specific antidotes.

Treatment is focused on respiratory symptoms: give inhaled bronchodilators and inhaled or IV steroids. Consider need for intubation (in cases of laryngeal oedema or respiratory collapse); consider positive pressure (via continuous positive airway pressure (CPAP), non-invasive ventilation (NIV) or mechanical ventilation). Any pulmonary oedema is primarily toxic, not cardiogenic, and must be treated accordingly.

Rinse and treat corrosive eye lesions.

Rinse and treat corrosive skin lesions. Look for frostbite.
POTASSIUM IODIDE TO COUNTER EFFECTS OF RADIOACTIVE IODINE-131

Radioactive iodine (I-131) is a by–product of nuclear fission processes in nuclear reactors. In a nuclear accident, I-131 and other radioactive elements are released into the environment. People may then inhale the I-131 as the radioactive cloud passes or ingest products (mainly milk) that have been contaminated by radioactive fallout in the soil.

I-131 is absorbed by the thyroid gland, which leads to an increased risk of thyroid cancer in later life (which is otherwise rare). The increased risk is especially significant in cases of childhood exposure to I-131.

Taking potassium iodide decreases the probability of developing thyroid cancer from exposure to I-131.

HOW DOES POTASSIUM IODIDE WORK?
Potassium iodide is a compound that contains potassium and stable (non-radioactive) iodine. The thyroid gland uses stable iodine to produce thyroid hormones.

When a person takes potassium iodide, the thyroid gland is saturated with non-radioactive iodine; if they are then exposed to I-131, the latter will not be absorbed by the gland.

DOES POTASSIUM IODIDE PROTECT AGAINST RADIATION?
Potassium iodide only protects the thyroid gland from I-131: by preventing the absorption of I-131, it reduces the risk of thyroid cancer. Potassium iodide does not protect against the effects of I-131 on the rest of the body.

Potassium iodide also does not protect against the effects of radiation from other radioactive elements.

Potassium iodide should be used as an adjunct to evacuation, sheltering and control of foodstuffs.
WHEN SHOULD POTASSIUM IODIDE BE TAKEN?
A person should take potassium iodide when there is a high probability of exposure to a significant amount of I-131. It should be taken before or shortly after exposure to I-131 to be effective — optimally before or immediately coincident with passage of the radioactive cloud. It may still have a substantial protective effect even if taken three to four hours after exposure. Furthermore, if the release of I-131 into the atmosphere is protracted, then even delayed administration may have benefits by partially reducing the total radiation dose to the thyroid.

To know when to start taking potassium iodide, listen to experts such as local health authorities. At the same time, take the preventive measures of sheltering and ensuring access to safe food and water.

WHO SHOULD TAKE POTASSIUM IODIDE?
Adults under 40, children and pregnant and breastfeeding women should take potassium iodide.

Adults over 40 should not take potassium iodide unless local health authorities say so, e.g. because a very high degree of contamination of I-131 is expected. Adults over 40 are at low risk for developing thyroid cancer after exposure to I-131, and they have a greater chance of having allergic reactions to potassium iodide.

Breastfeeding women should stop breastfeeding because I-131 quickly gets into breast milk; however, if breast milk is the only food available, nursing should continue.

HOW IS POTASSIUM IODIDE SUPPLIED?
A liquid formulation in a dropper bottle is available, but packages of potassium iodide tablets are more common, with packages of 12 tablets being most common. Each white, round, cross-scored tablet contains 65 mg of potassium iodide. Store at 15 to 25°C. Keep the package dry and foil intact.
HOW MUCH POTASSIUM IODIDE SHOULD A PERSON TAKE?

<table>
<thead>
<tr>
<th>Age/weight</th>
<th>Dose (per day)</th>
<th>No. of 65 mg tablets</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults and children over 68 kg</td>
<td>130 mg</td>
<td>2</td>
</tr>
<tr>
<td>Children, 3–18 yr</td>
<td>65 mg</td>
<td>1</td>
</tr>
<tr>
<td>Children, 1 mo.–3 yr</td>
<td>32.5 mg</td>
<td>1/2</td>
</tr>
<tr>
<td>Infants (&lt; 1 mo.)</td>
<td>16 mg</td>
<td>1/4</td>
</tr>
</tbody>
</table>

HOW LONG SHOULD POTASSIUM IODIDE BE TAKEN?

Potassium iodide should be taken as long as the exposure to I-131 persists; follow the advice of the local health authorities.

Infants younger than one month and pregnant and breastfeeding women should take only one dose, as they have a higher risk of developing a thyroid illness after repeated doses. In this group, additional doses should be taken only after a medical consultation.

WHO SHOULD NOT TAKE POTASSIUM IODIDE?

- People with a history of iodine allergy or skin diseases such as dermatitis herpetiformis.
- People with thyroid disease should consult a doctor before taking potassium iodide.

ACCESS TO POTASSIUM IODIDE

In case of significant exposure to I-131, the most important issue is to have access to authoritative information in real time and access to potassium iodide at all times.
RICIN

OVERVIEW
Ricin is a toxin derived from the castor bean, *Ricinus communis*. Castor beans are grown in high quantities for castor-oil production; ricin is found in the bean pulp after the oil is separated from the bean. Poisoning can occur via ingestion, inhalation or injection. Parenteral exposure is more lethal than oral ingestion. Ricin is a heat-labile glycoprotein lectin composed of two chains: the B-chain facilitates entry into cells, where the A-chain inhibits protein synthesis by ribosome inactivation, resulting in cell death. Ricin is a potent toxin and easy to obtain, thus making it suitable as a biological weapon.

DETECTING AND RECOGNIZING EXPOSURE
Ricin poisoning presents similarly to gastroenteritis or respiratory illnesses. Detection is mainly circumstantial (e.g. an increased number of patients seeking care, unexpected progression of symptoms, credible threats in the community). Tests to detect ricin in body fluids or environmental samples are not routinely available.

CLINICAL SIGNS AND SYMPTOMS
Ingestion
Mild poisoning can result in nausea, vomiting, diarrhoea and abdominal pain. In moderate to severe poisoning, gastrointestinal tract symptoms can progress (over four to 36 hours) to hypotension, shock, liver and renal dysfunction, and possibly death.

Inhalation
Illness can occur within eight hours and includes cough, dyspnoea, arthralgias and fever, which may progress to respiratory distress and death.

Injection
Initial symptoms (within six hours) can include generalized weakness and myalgias. Progression (24 to 36 hours) may include vomiting, fever, hypotension, multi-organ failure and death.
PERSONAL PROTECTIVE EQUIPMENT (PPE) AND DECONTAMINATION

PPE
For aerosolized ricin, level B PPE is appropriate. The toxicant is not transmitted person-to-person, nor is it absorbed through intact skin. Prevent secondary contamination.

Decontamination
Administer single-dose oral activated charcoal 1 g/kg as a slurry within one to two hours of ingestion. Consider gastric lavage or endoscopic removal for large, recently (within one hour) ingested quantities.

FIRST AID (ABCs)
Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss. Treat coma, shock, seizures and arrhythmias.

TREATMENT
Treatment is mainly supportive. First-aid therapies should be continued.

Extracorporeal elimination is not effective.

No antidotes are available.
SIMPLE ASPHYXIANTS

OVERVIEW
Simple asphyxiants do not damage the mucous membranes of the respiratory tract. They simply displace the oxygen from the air, thus reducing the usual 21% oxygen available in ambient air.

The most relevant compounds in this group include nitrogen (N2), carbon dioxide (CO2), methane (CH4), propane (C3H8) and butane (C4H10). Some of these are shorter-chain hydrocarbons; at high doses of exposure, effects can be similar to those of longer-chain hydrocarbons (for more information on hydrocarbons, see the factsheet on p. 181).

DETECTING AND RECOGNIZING EXPOSURE
All the gases mentioned above are colourless. While nitrogen and carbon dioxide are odourless, methane, propane and butane have a characteristic odour. Recognition of exposure is circumstantial.

Time to symptoms is short but dependent on the grade of oxygen deficiency. While mild hypoxia causes symptoms as described below, absolute lack of oxygen in breathed air causes loss of consciousness within one to two minutes. Increased concentrations of carbon dioxide in inhaled ambient air leads to hypercapnia.

The laboratory test for confirmation of hypoxia is arterial blood gas analysis.

CLINICAL SIGNS AND SYMPTOMS
Hypoxia can cause headache, dizziness, weakness, confusion, agitation, convulsions and coma, eventually leading to death. Sudden loss of consciousness is typical in an oxygen-deficient environment.

Hypercapnia leads to an increased respiratory rate (tachypnoea), air hunger and central nervous system depression in a dose-dependent way.
PERSONAL PROTECTIVE EQUIPMENT (PPE) AND DECONTAMINATION
Remove the patient from the oxygen–deficient environment. Emergency responders need a self-contained breathing apparatus.

Decontamination is not necessary.

FIRST AID (ABCs)
Ensure open airway, adequate ventilation and oxygenation.

TREATMENT
Give high-flow oxygen (>15 litres per minute via non-rebreather reservoir mask).

Intubate and ventilate for inadequate spontaneous ventilation or uncontrolled airway.

Check for hypoxia–related complications and sequelae.
SODIUM HYDROXIDE (STRONG ALKALIS)

OVERVIEW
Sodium hydroxide is found as a hygroscopic solid (as pellets) or, usually, as an aqueous solution. Sodium hydroxide can be a strong irritant and corrosive agent, depending on its concentration. Concentrated sodium hydroxide has a pungent smell.

It is used as a detergent, paint remover, drain cleaner and oven cleaner and in chemical industry. Exposure occurs in the home as well as in industrial or commercial activities.

The cation (sodium) is not toxic.

DETECTING AND RECOGNIZING EXPOSURE
The diagnosis is based on the rapid caustic effect at the contact site (skin, mucous membranes, airways, eyes). As all strong alkalis, it causes a liquefaction–type necrosis of organic tissues by saponification of fats, thus promoting rapid and deep penetration.

After ingestion, diagnostic/prognostic endoscopy (to determine the damage) should be performed within 12 hours, but not later than 24 hours – during this time interval, the risk of iatrogenic perforation is minimal.

CLINICAL SIGNS AND SYMPTOMS
Sodium hydroxide is acutely irritating, producing immediate signs of tissue damage, leading to reddening (erythema), burning pain, swelling and necrosis at the site of contact.

DECONTAMINATION
After contact, the damage occurs within minutes. As the extent of damage depends on the concentration and the duration of exposure, immediate decontamination is of paramount importance.

Skin decontamination
Remove all clothing, jewellery, shoes, etc. Wash affected body surfaces from head to toe with copious amounts of lukewarm water, protecting the patient’s airway. Pay close attention to exposed skin folds, axillae, genitals, feet and eyes
(see below). Then dry the entire body surface with towels in order to remove desorbed toxicants and prevent hypothermia.

**Eye decontamination**
Flush the eyes with plenty of water, Ringer’s solution or isotonic saline. Do not forget to remove contact lenses! For continuous rinsing during patient transport, eye irrigation lenses are most efficient.

**Gastrointestinal decontamination**
Sodium hydroxide is not adsorbed to activated charcoal, so the latter is not therapeutically effective. Cleanse the oesophagus by giving a few sips of water, but only immediately after exposure. Avoid large quantities of water in order to prevent vomiting, which would repeatedly expose the oesophageal mucosa to the caustic effect. After ingestion of large quantities, consider removal from the stomach by endoscopy or a soft nasogastric tube.

**FIRST AID (ABCs)**
After decontamination, management is analogous to that of burn lesions.

Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss. Treat coma, shock and arrhythmias.

**TREATMENT**
Enhanced elimination techniques are not effective.

After decontamination, management is analogous to that of burn lesions.

There are no antidotes.
SYSTEMIC ASPHYXIANTS

OVERVIEW
While simple asphyxiants decrease the amount of oxygen inhaled, systemic asphyxiants interfere with the body’s ability to transport to or use oxygen within the tissues.

Systemic asphyxiants include azides (p. 208), carbon monoxide (p. 210), cyanides (p. 212), hydrogen sulfide (p. 215) and methaemoglobin-forming compounds (p. 217) such as nitrites or aniline. Carbon monoxide and methae- moglobin-forming compounds decrease the transport capacity of haemoglobin for oxygen, while cyanides and hydrogen sulfide impair oxygen consumption in the mitochondria by blocking cytochrome c oxidase in the electron transport chain.
AZIDES

OVERVIEW
While simple asphyxiants decrease the amount of oxygen inhaled (fraction of inspired O2, FiO2), systemic asphyxiants interfere with the body’s ability to transport to or use oxygen within the tissues.

Systemic asphyxiants include carbon monoxide, cyanides, azides, hydrogen sulfide and methaemoglobin-forming compounds such as nitrites or aniline. Azides, like cyanides and hydrogen sulfide, impair oxygen consumption in the mitochondria by blocking cytochrome c oxidase in the electron transport chain.

DETECTING AND RECOGNIZING EXPOSURE
Recognition of exposure is circumstantial and time to symptoms is short (minutes). For hydrogen azide (also known as hydrazoic acid) the route of exposure is inhalation, whereas it is cutaneous or oral for azide salts and other azide compounds.

Analytical laboratory tests for confirmation of exposure are not routinely available. Marked lactic acidosis is invariably present in severe cases of azide poisoning.

CLINICAL SIGNS AND SYMPTOMS
Exposure to azides results in inhibited aerobic glycolysis and oxidative phosphorylation as a consequence of decreased ATP production due to inhibition of mitochondrial cytochrome c oxidase. This causes loss of consciousness and cardiovascular collapse. Marked lactic acidosis is invariably present in relevant exposures. Hydrogen azide is an irritant gas to skin and mucous membranes. Aqueous azide solutions are alkaline and corrosive to the eyes. Azides are strong direct vasodilators.
**DECONTAMINATION**

Remove the patient from the source of exposure. First responders and health-care workers need to protect themselves from secondary exposure (e.g. via vomit after oral exposure).

After a patient has been exposed to liquid or solid azides, remove their clothing and wash the skin with lukewarm water and soap or a mild liquid detergent. For solid azide particles adhered to the skin, use dry decontamination as much as possible because soluble azide salts form hydrogen azide on contact with water. Pay close attention to skin folds, axillae, genitals and feet. After ocular exposure, rinse the eyes continuously for at least 20 to 30 minutes with copious amounts of water, Ringer’s solution or normal saline. Irrigation lenses are helpful for this purpose. Health-care workers must avoid secondary contamination and should therefore protect themselves from the patient’s respiration, eructation and emesis. Quickly isolate all vomitus and gastric washings, and keep patients in a well-ventilated area.

**FIRST AID (ABCs)**

Ensure open airway, adequate ventilation and oxygenation. Give intravenous crystalloid fluids for moderate to severe symptoms.

For symptomatic patients with adequate spontaneous ventilation, give high-flow oxygen (more than 15 L/min.) via a non-rebreather reservoir mask. For patients with inadequate respiration, ventilate with 100% oxygen via a bag valve mask, followed by endotracheal intubation and ventilation with 100% oxygen. Cardiac monitor. Treat distributive shock due to excessive vasodilation with large quantities of isotonic crystalloid fluids.

**TREATMENT**

After thorough decontamination, treatment is symptomatic. No antidote is available.
CARBON MONOXIDE

OVERVIEW
While simple asphyxiants decrease the amount of oxygen inhaled (fraction of inspired O2, FiO2), systemic asphyxiants interfere with the body’s ability to transport to or use oxygen within the tissues.

Systemic asphyxiants include carbon monoxide, cyanides, azides, hydrogen sulfide and methaemoglobin–forming compounds such as nitrates or aniline. Carbon monoxide and methaemoglobin–forming compounds decrease the transport capacity of haemoglobin for oxygen.

DETECTING AND RECOGNIZING EXPOSURE
Recognition of exposure is circumstantial. Smoke gas inhalation may lead to combined toxicity with carbon monoxide, hydrogen cyanide, irritant gases and other toxins. The common route of exposure for carbon monoxide is inhalation.

Carbon monoxide is colourless and odourless.

Time to symptoms is short (minutes).

Analytical laboratory tests for confirmation of exposure are not routinely available. Fixed carbon monoxide detectors for buildings are available. Modern pulse oximeters can detect carbon monoxide, while conventional oximeters will report falsely elevated blood oxygen levels in patients with carbon monoxide poisoning! Co-oximeter–type blood gas analysers measure carboxyhaemoglobin (COHb) and methaemoglobin.

CLINICAL SIGNS AND SYMPTOMS
Carbon monoxide causes tissue hypoxia* due to reduced oxygen transport in the blood and diminished oxygen release in the tissues. To a lesser extent it also causes inhibition of mitochondrial cytochrome c oxidase.

*Hypoxia can cause headache, dizziness, weakness, confusion, agitation, convulsions and coma, eventually leading to death.
PERSONAL PROTECTIVE EQUIPMENT (PPE)
AND DECONTAMINATION

Remove the patient from the source of exposure. First responders and healthcare workers need to protect themselves with level B PPE.

No decontamination is necessary.

FIRST AID (ABCs)

Ensure open airway, adequate ventilation and oxygenation. Give intravenous crystalloid fluids for moderate to severe symptoms.

For symptomatic patients with adequate spontaneous ventilation, give high-flow oxygen (more than 15 L/min.) via a non-rebreather reservoir mask. For patients with inadequate respiration, ventilate with 100% oxygen via a bag valve mask, followed by endotracheal intubation and ventilation with 100% oxygen.

TREATMENT

Continue ventilation with 100% oxygen until COHb is under 5%. Consider hyperbaric oxygen for patients with loss of consciousness, persistent neurological signs and symptoms, cardiovascular complications, COHb above 20% or pregnancy with foetal distress. Cardiac monitor for dysrhythmias. Continuously monitor the patient’s level of consciousness.
CYANIDE

OVERVIEW
While simple asphyxiants decrease the amount of oxygen inhaled (fraction of inspired O2, FiO2), systemic asphyxiants interfere with the body’s ability to transport to or use oxygen within the tissues.

Systemic asphyxiants include carbon monoxide, cyanides, azides, hydrogen sulfide and methaemoglobin-forming compounds such as nitrites or aniline. Cyanides and hydrogen sulfide impair oxygen consumption in the mitochondria by blocking cytochrome c oxidase in the electron transport chain.

DETECTING AND RECOGNIZING EXPOSURE
Recognition of exposure is circumstantial. Smoke gas inhalation may lead to combined toxicity with carbon monoxide, hydrogen cyanide, irritant gases and other toxins. The common route of exposure for hydrogen cyanide is inhalation, and cutaneous or oral for cyanide salts and cyanogenic compounds.

Hydrogen cyanide gas has a characteristic odour of bitter almonds. (Note: there is a genetically determined variability in humans to detecting the smell!)

Time to symptoms is short (minutes).

Analytical laboratory tests for confirmation of exposure are not routinely available.

CLINICAL SIGNS AND SYMPTOMS
Exposure to cyanides and cyanogenic compounds results in inhibited aerobic glycolysis and oxidative phosphorylation as a consequence of decreased ATP production due to inhibition of mitochondrial cytochrome c oxidase. This causes loss of consciousness and cardiovascular collapse. Marked lactic acidosis is invariably present in relevant exposures. Hydrogen cyanide is an irritant to mucous membranes.
PERSONAL PROTECTIVE EQUIPMENT (PPE) AND DECONTAMINATION

PPE
PPE protects rescuers from exposure to the toxicant and from secondary contamination, and it should always be worn according to the risk of exposure; the highest level of PPE (level A*) should be used for unknown hazards and for airborne toxicants with the potential for skin exposure to vapour.

* Level A PPE includes a self-contained breathing apparatus with a vapour-protective, fully encapsulated chemical-resistant suit.

Decontamination
Remove the patient from the source of exposure. First responders and healthcare workers need to protect themselves from secondary exposure (e.g. via vomit after oral exposure).

After exposure to gaseous agents, no decontamination is necessary. After exposure to liquid or solid agents, remove clothing and wash the skin with lukewarm water and soap or a mild liquid detergent. Pay close attention to skin folds, axillae, genitals and feet. After ocular exposure, rinse the eyes continuously for at least 20 to 30 minutes with copious amounts of water, Ringer’s solution or normal saline. Irrigations lenses are helpful for this purpose.

FIRST AID (ABCs)
Ensure open airway, adequate ventilation and oxygenation. Give intravenous crystalloid fluids for moderate to severe symptoms.

For symptomatic patients with adequate spontaneous ventilation, give high-flow oxygen (more than 15 L/min.) via a non-rebreather reservoir mask. For patients with inadequate respiration, ventilate with 100% oxygen via a bag valve mask, followed by endotracheal intubation and ventilation with 100% oxygen. For bronchoconstriction, give nebulized beta agonists. Cardiac monitor; treat dysrhythmias following advanced cardiac life support guidelines. Continuously monitor the patient’s level of consciousness. Treat seizures with parenteral benzodiazepines (diazepam, lorazepam or midazolam). The first-aid antidote is amyl nitrite (see below).
TREATMENT
Continue first-aid treatment. Antidotes for cyanide toxicity are hydroxocobalamin and methaemoglobin-forming agents (such as amyl nitrite, sodium nitrite, 4-dimethylaminophenol (4-DMAP) and sodium thiosulfate).

- **Amyl nitrite** (not available in all countries): crush one ampoule (0.3 mL) and let contents inhale via bag valve mask.
- **Sodium nitrite**: 300 mg IV/IO over 5 min.
- **4-DMAP**: 250 mg IV/IO slowly; concomitant sodium thiosulfate and 100% oxygen is advised.
- **Hydroxocobalamin**: 5 g IV/IO over 15 min.; repeat once if necessary.
- **Sodium thiosulfate**: 100–200 mg/kg IV/IO slowly; repeat half dose after 30 to 60 min. if necessary.
SYSTEMIC ASPHYXIANTS

HYDROGEN SULFIDE

OVERVIEW
While simple asphyxiants decrease the amount of oxygen inhaled (fraction of inspired O2, FiO2), systemic asphyxiants interfere with the body’s ability to transport to or use oxygen within the tissues.

Systemic asphyxiants include carbon monoxide, cyanides, azides, hydrogen sulfide and methaemoglobin-forming compounds such as nitrites or aniline. Hydrogen sulfide, like cyanides and azides, impair oxygen consumption in the mitochondria by blocking cytochrome c oxidase in the electron transport chain.

DETECTING AND RECOGNIZING EXPOSURE
Recognition of exposure is circumstantial. The common route of exposure for hydrogen sulfide is inhalation.

Hydrogen sulfide smells like rotten eggs. Because of olfactory nerve paralysis, the absence of this odour does not rule out exposure.

Time to symptoms is short (minutes), and sudden unconsciousness can result.

Analytical laboratory tests for confirmation of exposure are not routinely available.

CLINICAL SIGNS AND SYMPTOMS
Exposure to hydrogen sulfide results in inhibited aerobic glycolysis and oxidative phosphorylation as a consequence of decreased ATP production due to inhibition of mitochondrial cytochrome c oxidase. This causes loss of consciousness and cardiovascular collapse, seizures and respiratory arrest. Marked lactic acidosis is invariably present in relevant exposures. Hydrogen sulfide is an irritant to the skin and mucous membranes. Observe for chemical pneumonia or pulmonary oedema.
DECONTAMINATION
Remove the patient from the source of exposure. First responders and health-care workers need to protect themselves from secondary exposure (e.g. via vomit after oral exposure).

Because hydrogen sulfide is a gas, skin decontamination is theoretically unnecessary, but it may be performed to eliminate the persistent odour. After ocular exposure, rinse the eyes continuously for at least 20 to 30 minutes with copious amounts of water, Ringer’s solution or normal saline. Irrigations lenses are helpful for this purpose.

FIRST AID (ABCs)
Ensure open airway, adequate ventilation and oxygenation. Give intravenous crystalloid fluids for moderate to severe symptoms.

For symptomatic patients with adequate spontaneous ventilation, give high-flow oxygen (more than 15 L/min.) via a non-rebreather reservoir mask. For patients with inadequate respiration, ventilate with 100% oxygen via a bag valve mask, followed by endotracheal intubation and ventilation with 100% oxygen. For bronchoconstriction, give nebulized beta agonists. Cardiac monitor; treat dysrhythmias following advanced cardiac life support guidelines. Continuously monitor the patient’s level of consciousness. Treat seizures with parenteral benzodiazepines (diazepam, lorazepam or midazolam).

TREATMENT
Continue first aid treatment. Antidotes for cyanide toxicity, including hydroxocobalamin and methaemoglobin-forming agents, have been used for hydrogen sulfide poisoning, but evidence for their effectiveness is lacking.
METHAEMOGLOBIN-FORMING COMPOUNDS

OVERVIEW
While simple asphyxiants decrease the amount of oxygen inhaled (fraction of inspired O2, FiO2), systemic asphyxiants interfere with the body’s ability to transport to or use oxygen within the tissues.

Systemic asphyxiants include carbon monoxide, cyanides, azides, hydrogen sulfide and methaemoglobin-forming compounds such as nitrites or aniline. Carbon monoxide and methaemoglobin-forming compounds (such as inorganic nitrate and nitrite salts, organic nitrites, nitric acid, aryl amines including 4-dimethylaminophenol, aniline, and all substances able to oxidize haemoglobin’s ferrous iron Fe2+ to ferric iron Fe3+) decrease the transport capacity of haemoglobin in the blood and the oxygen binding capacity of myoglobin in the muscles.

DETECTING AND RECOGNIZING EXPOSURE
Recognition of exposure is circumstantial. Smoke gas inhalation may lead to combined toxicity with carbon monoxide, hydrogen cyanide, irritant gases and other toxins, including methaemoglobin-forming compounds. Depending on the agent, the route of exposure is inhalation; exposure is cutaneous or oral for methaemoglobin-forming compounds.

Time to symptoms is short (minutes to one hour).

Analytical laboratory tests for confirmation of exposure are not routinely available. Co-oximeter-type blood gas analysers measure carboxyhaemoglobin (COHb) and methaemoglobin.
**CLINICAL SIGNS AND SYMPTOMS**

Exposure to methaemoglobin-forming compounds results in tissue hypoxia due to reduced oxygen transport in the blood, as well as chocolate-brown discoloration of haemoglobin and the blood. Patients have a cyanotic appearance. Additionally, nitrates cause vasodilation with flushing, reflex tachycardia, headache, hypotension, syncope and, in severe cases, cerebral, myocardial or intestinal ischaemia.

* Hypoxia can cause headache, dizziness, weakness, confusion, agitation, convulsions and coma, eventually leading to death.

**DECONTAMINATION**

Remove the patient from the source of exposure. First responders and healthcare workers need to protect themselves from secondary exposure (e.g. via vomit after oral exposure).

After exposure to gaseous agents, no decontamination is necessary. After exposure to liquid or solid agents, remove clothing and wash the skin with lukewarm water and soap or a mild liquid detergent. Pay close attention to skin folds, axillae, genitals and feet. After ocular exposure, rinse the eyes continuously for at least 20 to 30 minutes with copious amounts of water, Ringer’s solution or normal saline. Irrigations lenses are helpful for this purpose.

**FIRST AID (ABCs)**

Ensure open airway, adequate ventilation and oxygenation. Give intravenous crystalloid fluids for moderate to severe symptoms.

For symptomatic patients with adequate spontaneous ventilation, give high-flow oxygen (more than 15 L/min.) via a non-rebreather reservoir mask. For patients with inadequate respiration, ventilate with 100% oxygen via a bag valve mask, followed by endotracheal intubation and ventilation with 100% oxygen. Cardiac monitor. Treat distributive shock due to excessive vasodilation with large quantities of isotonic crystalloid fluids.

**TREATMENT**

Continue first aid treatment. The antidote for methaemoglobinemia above 30% is methylene blue (1–2 mg/kg IV/IO). Watch out for haemolysis in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency.
THALLIUM

OVERVIEW
Thallium is a soft metal that quickly oxidizes upon exposure to air. Thallium salts are used in the manufacture of jewellery, semiconductors and optic devices. Thallium exposure has been reported after ingestion of contaminated herbal preparations and adulteration of illicit drugs.

The mechanism of toxicity, although not fully understood, appears to be related to interaction with a variety of enzyme systems, as well as to the similarity of thallium with potassium (and thus the replacement of potassium with thallium).

Toxicity depends on the thallium compound. The more water-soluble salts (e.g. thallous acetate or thallic chloride) are slightly more toxic than the less soluble forms (e.g. thallic oxide, thallous iodide). Some thallium salts are well absorbed across intact skin. The common route of exposure is oral ingestion.

DETECTING AND RECOGNIZING EXPOSURE
Thallium toxicity should be considered when gastroenteritis and painful lower extremity paraesthesia is followed by alopecia.

Urinary thallium excretion (24-hour) is the standard diagnostic test for thallium exposure (normal urine levels are less than 5 µg/L); spot urine is unreliable. Normal whole-blood levels are less than 2 µg/L. Detection in hair is for forensic purposes (i.e. for past exposures).

Thallium is radiopaque, so it may be visible on abdominal radiographs.

CLINICAL SIGNS AND SYMPTOMS
Symptoms do not appear immediately after exposure but are typically delayed for hours to days.

Acute effects include abdominal pain, nausea, vomiting, diarrhoea. Circulatory shock may result from massive fluid loss. Within two to three days, delirium, seizures, respiratory failure and death may occur.

Chronic effects include painful peripheral neuropathy, myopathy, chorea, stomatitis, ophthalmoplegia, hair loss and nail dystrophy (with Mees’ lines).
DECONTAMINATION
Administer single-dose oral activated charcoal 1 g/kg as a slurry within one to two hours of ingestion. Consider gastric lavage or endoscopic removal for large, recently (within one hour) ingested quantities. Whole-bowel irrigation may be helpful when radiopaque material is still present in the gastrointestinal tract.

Early oral administration of Prussian blue (see below) can prevent enteral absorption of thallium.

FIRST AID (ABCs)
Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids, along with vasopressor agents, for hypotension and fluid loss.

Treat coma, shock, seizures and arrhythmias.

TREATMENT
In addition to supportive therapy, the chelator ferric ferrocyanide (Prussian blue) is the antidote of choice and the mainstay of therapy. It binds thallium ions within the gastrointestinal tract, thus inhibiting enteral absorption, and enhances elimination by interference with the enterohepatic recirculation.

Dosage of Prussian blue is 3 g every eight hours orally (500 mg tablets are available) until the urinary thallium excretion falls below 0.5 mg/day, although this end point is disputed.

Other enhanced elimination techniques are not effective. Haemodialysis is only of benefit in patients with concomitant renal failure.
TOXIC ALCOHOLS

Methanol, ethylene glycol, diethylene glycol

OVERVIEW

Among the various alcohols, methanol, ethylene glycol and diethylene glycol have a particular toxicity which depends on their metabolites (including formic acid and oxalic acid), while the parent compounds (e.g. ethanol, 2-propanol) have toxicities comparable to most other alcohols. Formic acid is neurotoxic, while oxalic acid is nephrotoxic; diethylene glycol is both.

The only route of exposure is oral ingestion; inhalation or dermal absorption are irrelevant.

DETECTING AND RECOGNIZING EXPOSURE

The diagnosis is mostly based on the circumstances of exposure and the typical toxidrome, including inebriation, increasing metabolic acidosis and neurotoxicity with central nervous system depression and visual loss (for methanol) or neurotoxicity and renal failure (for ethylene glycol).

A high index of suspicion should be present when consumption of alcoholic beverages (particularly of clandestine origin or containing antifreeze) is followed by severe metabolic acidosis, high osmolar gap and increasing anion gap.

Blood concentrations of methanol and ethylene glycol are available in specialized analytical laboratories.

CLINICAL SIGNS AND SYMPTOMS

The toxic alcohol toxidrome consists of inebriation with decreased level of consciousness (from somnolence to coma), metabolic acidosis (with marked hyperventilation), high osmolar gap and increasing anion gap. The anion gap increases in parallel to an initially high, decreasing osmolar gap.

In methanol poisoning, after a latent period of up to 30 hours severe metabolic acidosis ensues, with seizures, coma and possibly death. Fundoscopy reveals optic disc changes.
In ethylene glycol poisoning, after a latent period of four to 12 hours, severe metabolic acidosis ensues with seizures, coma, hypocalcaemia with dysrhythmias, and kidney failure with oxalate crystals in the urinary sediment. Kidney failure is usually reversible.

The latent period is prolonged if ethanol has been ingested concurrently.

**DECONTAMINATION**
Aspiration of gastric contents with a small-bore tube may be useful if done early (within 30 to 60 minutes of ingestion). Activated charcoal has no effect.

**FIRST AID (ABCs)**
Ensure open airway, adequate ventilation and oxygenation. Use intravenous crystalloid fluids along with vasopressor agents for hypotension and fluid loss. Treat coma, seizures, shock and arrhythmias. Correct metabolic acidosis with IV sodium bicarbonate. Monitor the patient for several hours with repeated blood gas analyses.

Fomepizole and ethanol are the antidotes of choice:

- **Fomepizole**
  - Loading dose: 15 mg/kg (IV)
  - Maintenance dose: 10 mg/kg every 12 hr for four doses, then increase to 15 mg/kg every 12 hr until methanol/ethylene glycol levels are < 20 mg/dL

- **Ethanol** (target blood alcohol concentration is 100 mg/dL (1‰); obtain serum ethanol levels after loading dose and frequently thereafter)
  - Loading dose (IV): 800 mg/kg (take into account any pre-existing ethanol level)
  - Maintenance dose: approx. 100–150 mg/kg/h; under haemodialysis 175–350 mg/kg/h (higher doses for patients with chronic alcoholism)

In methanol poisoning, leucovorin should be administered as an adjunct antidote. In ethylene glycol poisoning, adjunct antidotes are pyridoxine, thiamine and folate.

Treat hypocalcaemia with intravenous calcium gluconate.
**TREATMENT**

Continue antidotal treatment with fomepizole or ethanol.

Haemodialysis efficiently removes methanol, ethylene glycol, diethylene glycol and their toxic metabolites, and rapidly corrects acidosis. Continuous venovenous haemodiafiltration (CVVHDF) is an acceptable alternative in unstable patients, but the rate of elimination is slower.

**Indications for haemodialysis**

**Ethylene glycol**
- Suspected ethylene glycol poisoning with an osmolar gap greater than 10 mosm/L not accounted for by ethanol or other alcohols, accompanied by metabolic acidosis (pHart < 7.25–7.30) unresponsive to therapy
- Ethylene glycol poisoning accompanied by renal failure
- Ethylene glycol serum concentration greater than 25 to 50 mg/dL, unless the patient is asymptomatic and already receiving fomepizole or ethanol treatment
- Severe metabolic acidosis with a history of ethylene glycol ingestion, even if the ethylene glycol level or osmolar gap is not elevated (in late presentations)

**Methanol**

Methanol exposure with:
- elevated serum methanol level (above 700 mg/dL or 21.8 mmol/L under fomepizole treatment; above 600 mg/dL under ethanol treatment; above 500 mg/dL without fomepizole or ethanol)
- elevated osmolar gap
- serum anion gap above 24 mmol/L
- severe acidosis (pH ≤ 7.15)
- coma or seizures
- new visual deficits
- kidney dysfunction
VESICANTS AND BLISTER AGENTS

OVERVIEW
Vesicants, also referred to as “blister agents”, are chemical warfare agents that causes severe chemical burns, painful blisters and difficulty breathing. Examples include lewisite, mustard and phosgene oxime.

Exposure is rarely deadly to healthy people but can cause long-term effects. If the person is already sick, or if the exposure is significant, it can result in death. No antidote exists for these agents.

DETECTING AND RECOGNIZING EXPOSURE
Physical reactions to vesicants usually manifest within two to 24 hours after exposure, but some people may react sooner (e.g. with lewisite or phosgene oxime, there may be pain at the time of exposure or shortly thereafter). Quick onset of skin changes suggests phosgene oxime, and delayed onset of any symptoms suggests mustard agent.

There are a variety of devices available to detect vesicant vapour and liquid. Clinical diagnosis can be confirmed by laboratory tests (usually measuring either a metabolite or a DNA or protein adduct), but these tests are available only from specialized laboratories.

SIGNS AND SYMPTOMS OF EXPOSURE
Skin
- Severe irritation, including blisters (within one hour for phosgene oxime, delayed for two to 12 hours for lewisite, delayed for two to 24 hours for mustard) and erythema (immediate for lewisite and phosgene oxime, possibly delayed for two to 24 hours with mustard)
- Intense itching and brown-yellow pigmentation at the burn site
- Possible second- and third-degree chemical burns

Eyes
- Severe irritation, including tearing, conjunctivitis and corneal damage

Respiratory
Lesions have decreasing severity down the respiratory tract from the point of entry. In the nose, larynx, and trachea, there may be swelling, erythema and necrosis of the mucosa (which may lead to obstruction), followed by sloughing
and fibrinous exudation. Pulmonary oedema is normally not significant, but secondary pneumonia is common. The damage results in mild (hoarse voice, sore throat) to extreme respiratory distress.

**Gastrointestinal**
Ingestion can lead to wounds to the epithelial cells of the gastrointestinal tract, with eventual necrosis and desquamation with haemorrhage. Nausea may occur early after moderate high doses.

**Cardiovascular**
- Atrioventricular block and cardiac arrest with high-dose exposure
- Hypotension with high-dose exposure to lewisite
- Possible chest tightness and dyspnoea with severe exposure

**DECONTAMINATION**
Proper and quick decontamination is a must. Treatment may be very painful; provide sufficient pain relief, e.g. ketamine.

**Skin decontamination**
Remove all clothing, jewellery, shoes, etc. Wash affected body surfaces from head to toe with copious amounts of lukewarm water, protecting the patient’s airway. Pay close attention to exposed skin folds, axillae, genitals, feet and eyes (see below). Then dry the entire body surface with towels to remove desorbed toxicants and prevent hypothermia.

**Eye decontamination**
Flush the eyes with plenty of water, Ringer’s solution or isotonic saline. Do not forget to remove contact lenses! For continuous rinsing during patient transport, eye irrigation lenses are most efficient.

**FIRST AID AND TREATMENT**
Standard resuscitation and supportive treatment apply. Fluid loss is less than in patients with thermal burns, so less fluid should be used in the initial resuscitation than is standard for burn patients. Ensure that the patient has an unobstructed airway.

Do not induce vomiting (emesis), and keep nil by mouth (NPO). Respiratory complications are the most common reason for death from mustard gas; intensive care may be needed.
Blisters
• Blisters caused by mustard gas are initially fragile but will harden over time.
• Fluid in blisters is not dangerous to rescuers.
• Fluid will reabsorb in one to three weeks, depending on severity, location and the patient’s sensitivity.
• Meticulous hygiene is important to prevent secondary infection.
• Infection is the primary concern when blisters rupture. Deroof ruptured blisters and wash the wound bed well.
  – Cover small open blisters with petrolatum gauze.
  – Leave large open blisters uncovered.
• Inspect wounds and change dressings every three to four days.
• Remove dressings carefully to avoid pulling off the tops of blisters.

Eyes
• Corticosteroids may reduce inflammation and promote healing.
• Apply sterile petroleum jelly to the upper and lower eyelids to prevent the lids from sticking together (do not apply directly on the eyes themselves).
• Serious eye irritation will result in swelling and photophobia, blocking the patient’s vision. With meticulous hygiene, one may gently force the patient’s eyes open to reassure them they are not blind.
• Use sunglasses for photophobia.
• Do not bandage eyes; eyelids can stick together, which can cause corneal damage.

Burns
• Mustard gas burns often look like a sunburn or rash in the area surrounding blisters and will heal in about the same time as a sunburn of equivalent severity.
• Burns may appear without any blisters in cases of mild exposure.
• Burns may be treated with topical ointments to reduce discomfort.

Respiratory distress
• Treatment is seldom required for mild respiratory irritation, such as a hoarse voice and sore throat.
• Provide supportive treatment as needed with humidified oxygen, bronchodilators, inhalation of cooled mist, physiotherapy and respiratory support.
• N-acetylcysteine (NAC) may be of benefit for respiratory injury due to mustard gas.*
• Use targeted antibiotics where appropriate.

**Bone marrow depression**
Bone marrow depression with leukopaenia occurs three to five days post-exposure. Monitor for the first two weeks with full blood counts. Bone marrow depression leading to severe leukopaenia and aplastic anaemia should be treated with colony-stimulating factor, platelet and red cell transfusions.

**LONG-TERM EFFECT**
Skin scarring, and pigment changes may follow a severe skin lesion from sulfur mustard exposure; cancer sometimes develops in scarred skin. Eye exposure may result in permanent eye injury or chronic eye infections. Inhalation may result in chronic respiratory disease, recurrent respiratory infections and possibly respiratory cancers.

WHITE PHOSPHOROUS

OVERVIEW
White phosphorus (WP) is a white, yellow or colourless, waxy combustible solid with a garlic-like odour. It is used in many of the smoke devices that are prevalent in military arsenals. It is a pyrophoric material, meaning it ignites spontaneously when exposed to air.

DETECTING AND RECOGNIZING EXPOSURE
No medical test is available. Since most exposure to WP occurs in the military setting with the use of munitions, a history is often easy to obtain. However, it may be necessary to ask about suicidality or possible accidental ingestion in a patient with signs or symptoms of exposure.

CLINICAL SIGNS AND SYMPTOMS
Exposure to phosphorus-containing compounds causes serious, often fatal, burns (second- to third-degree thermal burns) and can be the source of significant morbidity and lengthy hospital stays.

Release of WP in an enclosed area can cause asphyxiation owing to the decrease in oxygen. Exposure to smoke created by burning WP from military munitions can result in irritation or damage to the eyes, lungs and throat.

WP is highly fat soluble, and absorption results in necrosis of the liver (hepatic necrosis) and/or kidneys (glomerular and tubular damage up to kidney failure). Before the damage to the kidneys and/or liver becomes evident, there may be cardiovascular collapse due to hypovolaemia caused by chemical burns and by cardiotoxicity with pump failure (cardiogenic shock).

WP can produce serious physiological alterations in as little as one hour after the burn, including hypocalcaemia and hyperphosphataemia with calcium-phosphate shifts. The rapid development of hypocalcaemia and hyperphosphataemia post burn is responsible for cardiac arrhythmias with abnormalities, including prolonged QT intervals (with the potential of torsades de pointes), ST-T wave changes and progressive bradycardia.

Furthermore, exposure over a relatively small surface area (10 to 15% of total body surface area) can cause sudden and often unexpected death.*
Breathing in WP for short periods may cause coughing and irritation of the throat and lungs. Breathing in WP for long periods may cause a condition known as “phossy jaw”, which involves poor wound healing of the mouth and breakdown of the jawbone. Ingestion can lead to gastrointestinal symptoms (vomiting, abdominal cramps and pain). Ocular exposure may cause irritation, blepharospasm, photophobia, lacrimation and conjunctivitis, and particles may cause corneal perforation.

**DECONTAMINATION**

**Skin decontamination**
Remove all clothing, jewellery, shoes, etc. Wash affected body surfaces from head to toe with copious amounts of lukewarm water, protecting the patient’s airway. Pay close attention to exposed skin folds, axillae, genitals, feet and eyes (see below). Then dry the entire body surface with towels in order to remove desorbed toxicants and prevent hypothermia. Continuous irrigation can prevent further oxidation and enable removal of particles from the skin surface without reignition. Phosphorus will fluoresce under ultraviolet light (e.g. a Wood’s lamp). This can help to detect loose or embedded phosphorus, including if the suspected areas of contamination are immersed in water.

**Eye decontamination**
Flush the eyes with plenty of water, Ringer’s solution or isotonic saline. Do not forget to remove contact lenses! For continuous rinsing during patient transport, eye irrigation lenses are most efficient.

**Wounds**
Since WP spontaneously ignites on contact with air (and will reignite after all fire has been extinguished), contaminated wounds need to be kept covered with gauze soaked in clean water, saline or similar until all WP is gone. Do not use an oily or greasy dressing because phosphorous is lipid-soluble and can penetrate into the body tissue.

Carefully decontaminate the patient in a well-ventilated area because the absorbed agent can be released from clothing and skin as a gas. Meticulous surgical debridement of all embedded phosphorus particles is required; wear proper gloves, and remove it using tools such as forceps (if using tooth surgical forceps, be careful not to break the particles) or a spoon. To avoid reignition, store any collected solid particles under water in a canister.
TREATMENT
There is no antidote. Treatment is symptomatic (respiratory support if needed, treating hypotension and arrhythmias, correcting electrolyte abnormalities). Monitor vital signs, cardiac rhythm and blood tests (phosphorus level, C-reactive protein, glucose and electrolytes), and perform 12-lead electrocardiography.

Treat the burn according to international medical guidelines.

White phosphorus burns

The ICRC helps people around the world affected by armed conflict and other violence, doing everything it can to protect their lives and dignity and to relieve their suffering, often with its Red Cross and Red Crescent partners. The organization also seeks to prevent hardship by promoting and strengthening humanitarian law and championing universal humanitarian principles.

People know they can count on the ICRC to carry out a range of life-saving activities in conflict zones and to work closely with the communities there to understand and meet their needs. The organization’s experience and expertise enables it to respond quickly and effectively, without taking sides.