NATO STANDARD

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MEDICAL MANAGEMENT OF CBRN CASUALTIES

Edition A Version 1

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NORTH ATLANTIC TREATY ORGANIZATION

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NORTH ATLANTIC TREATY ORGANIZATION (NATO)

NATO STANDARDIZATION OFFICE (NSO)

NATO LETTER OF PROMULGATION

19 June 2018

1. The enclosed Allied Medical Publication AMedP-7.1, Edition A, Version 1, MANAGEMENT OF CBRN CASUALTIES, which has been approved by the nations in the Military Commettee Medical Standardization Board, is promulgated herewith. The agreement of nations to use this publication is recorded in STANAG 2461.

2. AMedP-7.1, Edition A, Version 1, is effective upon receipt and supersedes AMedP-06, Edition C, which shall be destroyed in accordance with the local procedure for the destruction of documents.

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4. This publication shall be handled in accordance with C-M(2002)60.

Zoltán GULYÁS
Brigadier General, HUNAF
Director, NATO Standardization Office

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RECORD OF RESERVATIONS

CHAPTER	RECORD OF RESERVATION BY NATIONS
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RECORD OF SPECIFIC RESERVATIONS

[nation]	[detail of reservation]
FRA	France considers that the risk-benefit ratio of pulmonary lavage is not favourable and will not apply this measure to casualties that it manages.
GRC	Hellenic Armed Forces medical capabilities are considered rather limited for the nonce. Implementation cannot proceed prior to developments in national medical CBRN funding/training policies for deployable formations.
SVK	At present, the Medical Service of the Armed Forces of the Slovak Republic is not equipped with the medical equipment required by this STANAG. The Slovak Republic reserves the right of the future acquisition of the missing equipment in accordance with the national acquisition programs.
USA	(1) Paragraph 2.2. subpara 1. The US reserves the right to not use "CBRNE3T" since it is not an approved US Joint or Army acronym. (2) Paragraph 4.2.1. Replace "Levels of Care" with "Roles of Care" per STANAG 2228 Ed.3, Allied Joint Doctrine for Medical Support, AJP-4.10, paragraphs 1.2.6 through 1.2.10. (3) Paragraph 4.7. Change "within an hour" to "as soon as practical." Per US ratification AJP-4.10(NB) with the following reservations statement "the U.S. does not concur with, nor subscribe to, specific timeframes for the indicated care." (4) Paragraph 4.8, subpara1. Change to: "If casualty hazard management is required, evacuation of the casualty to an MTF that delivers damage control surgery should occur as soon as practical." Per US ratification AJP-4.10(NB) with the following reservations statement "the U.S. does not concur with, nor subscribe to, specific timeframes for the indicated care." (5) Paragraph 6.3.1, subpara 1.a-1.d. The US uses three levels of casualty decontamination; immediate, operational, and thorough. (6) Paragraph 6.6, subpara 5. The US does not use the term "dress state." (7) Paragraph 24.2. In the US, National legal and political frameworks determine whether or how RCA may be used in military operations.

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CHAPTER 1: INTRODUCTION

1.1. AIM

The aim of this allied publication (AP) is to provide guidance to medical personnel on the management of casualties in a CBRN environment.

1.2. SCOPE

1. Previous editions of AMedP-6 series, *Handbooks on the Medical Aspects of NBC Defensive Operations*, have focused on CBRN defensive operations. This new edition applies to any medical support operation and supports the NATO 'defence against terrorism' (DAT) programme even if the CBRN threat is low.

2. This publication is intended to guide CBRN casualty management from point of exposure (PoE) through to a role 3 Medical Treatment Facility (MTF). The guidance uses the 10-1-2 time framework of MC 326/3[1] as a goal for optimal care while understanding the limitations of operating in a CBRN environment. Role 4 care is likely to use home nation health resources as well as international and civilian cooperation and networks and is therefore beyond the scope of this publication.

3. AMedP-7.1 focuses on the delivery of medical countermeasures (MedCM) and casualty care, post-incident response and, in the case of covert CBRN incidents, medical recognition, health surveillance and operational epidemiology by deployed medical personnel.

4. This publication is not intended to provide specific treatment protocols as these remain the responsibility of each member nation. During implementation, nations may customise the treatment protocols shown for illustration by the custodian nation (GBR).

5. AMedP-7.1 is intended to be used as a template to support national CBRN medical training programs in conjunction with AMedP-7.3: *Training of Medical Personnel for CBRN Defence*.

6. Changes and revisions of this publication will reflect:

   a. Scientific advice (✍) from the NATO Science and Technology Organisation including the Human Factors and Medicine Panels (HFM-041, HFM-099, HFM-186, HFM-222, HFM-253[2]), as well as other expert organisations and panels.

   b. Lessons identified or best practice (☑) from national, NATO and international operations and exercises including the NATO Exercise Clean Care 2016.

   c. Peer-reviewed international guidelines and reviews including 'Surviving Sepsis'✍.

1.3. RELATED DOCUMENTS

A list of related documents is provided at the end of the publication.

[1] MC 326/3 NATO Medical Support Principles and Policies.
[2] STO related documents are listed at the end of this publication.

1.4. DEFINITIONS

CBRN and medical terminology is consistent with the NATO lexicons. Where there is no recognised NATO medical terminology, Dorland's Medical Dictionary (2008) has been used unless otherwise referenced.

1.5. NEW CONCEPTS IN AMEDP-7.1

1. AMedP-7.1 is the re-codified version of the AMedP-6 series. It has been written as a series of chapters divided into 5 parts rather than 3 independent volumes. These changes to the structure of the AMedP-7.1 series reflect the change of threat from an obvious nuclear, biological or chemical attack to a more generic all-hazards approach in the management of any casualty in a CBRN environment, incident management and definitive CBRN management (chemical, biological and radiological including nuclear). The changes also reflect a spectrum of hazards from deliberate CBRN weapons through to environmental hazards and accidental or natural exposures.

2. The publication remains a reference for operational planning and the medical logistic support to the CBRN incident cycle described in AJMedP-7 and other publications in the AMedP-7 series. There is a greater emphasis on the CBRN tactical response in Part 2 of this publication for medical personnel deploying to an incident.

3. New concepts include:

   a. Cycle of recognition (intelligence, detection, diagnosis, forensics).

   b. Casualty hazard management including containment, decontamination, isolation and quarantine.

   c. Generic CBRN incident medical management paradigm.

   d. Command guidance on the medical aspects of restriction of movement (RoM).

   e. Management of paediatric CBRN casualties.

   f. Incorporation of STANAG 2242: *Policy for the Chemoprophylaxis and Immunotherapy of NATO Personnel against Biological Warfare Agents*.

   g. Syndromic approach to the CBRN casualty assessment.

   h. 'Surviving sepsis' international guidelines for the management of biological casualties (✍).

4. Standardised documentation derived from this publication include:

   a. CBRN casualty report form.

   b. CBRN medical incident report form (METHANE).

   c. CBRN casualty handover template (AT-MIST-D).

1.6. THE STRUCTURE OF AMEDP-7.1

This publication consists of five parts:

Part 1 – Generic management of CBRN casualties.

Part 2 – Medical management of a CBRN incident.

Part 3 – Management of chemical casualties.

Part 4 – Management of biological casualties.

Part 5 – Management of radiological casualties including nuclear.

1.7. MEDICAL CONTRIBUTION TO CBRN DEFENCE

MedCM are one significant component of CBRN defence. However, CBRN medical support is not limited to this and elements of medical support cross over into the other components. The investigation of an incident or outbreak and the subsequent implementation of interventions as a report of *operational epidemiology* demonstrates the link between CBRN Inform (Detect and Information Management) and CBRN Protective Measures (Physical Protection, MedCM and Hazard Management) – see Chapter 17. Figure 1-1 demonstrates these relationships and interactions.

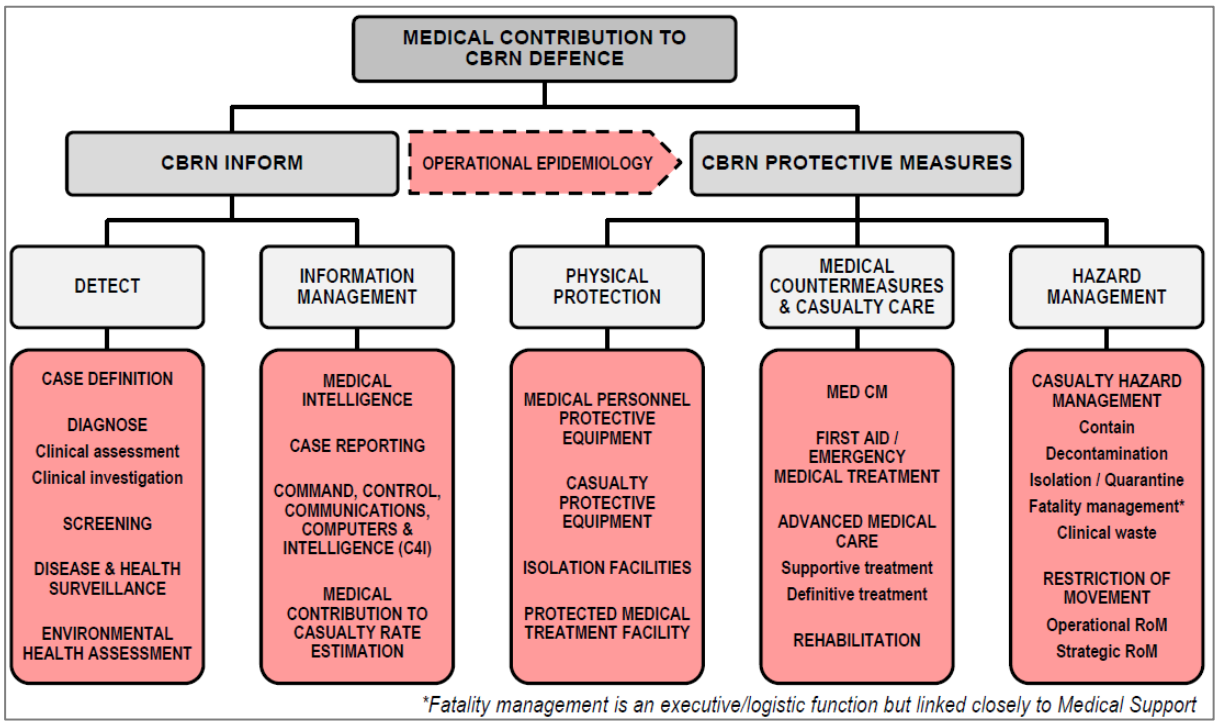

Figure 1-1: Medical contribution to CBRN defence (CBRN medical support).

1.8. CONSIDERATIONS FOR CBRN MEDICAL PLANNING AND DELIVERY

1. The medical planning process for CBRN defensive operations, as well as CBRN casualty care and outbreak response, includes:

a. Threat and hazard assessment.

b. Operational risk assessment.

c. Planning.

d. Force protection.

e. Incident (and outbreak) response.

f. Incident recovery.

2. On CBRN defensive operations with an increased CBRN threat, a CBRN event is likely to be recognised by detectors followed by warning and reporting. The event is recognised and becomes an incident. On low CBRN threat operations, the most likely recognition will be those first responding on the scene and through the medical chain.

3. For biological incidents or covert events, recognition is almost certainly through the medical chain and includes the management of the unusual patient (see Annex 7A). Chapter 5 describes CBRN recognition in detail.

4. Some of the considerations that have a direct effect on CBRN casualty care are described in this AP. Command guidance, casualty estimation and specialist medical investigation and response teams can be found in other APs in the AMedP-7 series.

1.8.1. FORCE PROTECTION CONSIDERATIONS

The considerations for force protection during CBRN defensive operations include:

a. Vaccination programmes.

b. Pre-exposure MedCM.

(1) Pre-exposure prophylaxis.

(2) Pre-treatment.

c. Individual / personal protective equipment (IPE / PPE).

d. CBRN medical force generation.

e. Medical treatment facility collective protection (MTF COLPRO).

f. Medical treatment facility isolation areas.

g. Disease surveillance including human, animal and plant.

1.8.2. INCIDENT RESPONSE CONSIDERATIONS

The considerations for the immediate post-event actions (incident response) cover the saving of life and the continuation of the vital mission objectives, and include:

a. Post-exposure MedCM:

(1) Post-exposure prophylaxis.

(2) Immediate therapy.

b. Casualty care in a CBRN-threat environment:

(1) First aid.

(2) Emergency medical treatment including decontamination.

(3) Advanced medical care.

c. Isolation and quarantine.

d. Implementing restriction of movement (operational and strategic).

e. Outbreak investigation (and escalation to a NATO Bio-Response[3]).

f. Casualty and case reporting.

g. Medical evacuation (MEDEVAC).

h. Accessing CBRN medical stockpiles.

i. Accessing reach back advice and support.

1.8.3. INCIDENT RECOVERY CONSIDERATIONS

The considerations for the later post-event actions (incident recovery) cover the restoration to normality and support to the post-incident investigation, and include:

a. Casualty rehabilitation.

b. Medical treatment facility and equipment hazard management.

c. Exploitation to support knowledge management and forensics.

d. Fatality management (although not a medical task).

e. Review and easing of restriction of movement.

f. Risk communication.

[3] NATO Bio-Responsiveness is currently subject to a Smart Defence Project (SD 1.45).

g. Medical logistics resupply.

h. CBRN clinical waste management.

i. Preventive (occupational) medicine support.

j. Health surveillance.

k. After actions reporting.

l. Lesson learnt process.

1.9. CONTRIBUTIONS, ACKNOWLEDGEMENTS & POINTS OF CONTACT

1. Contributions for this Allied Publication were through the COMEDS CBRN Medical Working Group and Biological Medical Expert Panel. Part Five: The Management of the Radiological Casualty includes content provided by the US Armed Forces Radiobiology Research Institute (US AFRRI).

2. The custodian and point of contact for this Allied Publication is:

Medical Director
Defence CBRN Centre
Winterbourne Gunner
Salisbury
SP4 0ES
United Kingdom

1.10. ADDITIONAL CONTENT

This AP is supported by Quick Response (QR) codes, and Augmented Reality (AR). AR uses a free application that can be downloaded using the QR link below and provides additional content, examples of best practice (☑) and supports the training component of this document.[4]

Links to the AR additional content are identified by the ● symbol.

[4] The additional content of this AP is not covered by the STANAG and should be considered as a vitual standardisation related document (SRD).

AMedP-7.1 PART 1: THE GENERIC APPROACH TO CBRN CASUALTIY MANAGEMENT

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CHAPTER 2: CBRN THREATS AND HAZARDS

2.1. INTRODUCTION

1. Since the end of the Cold War, the threat to NATO nations of a state authorised CBRN attack has reduced significantly. However, the threat of other CBRN incidents such as a non-state deliberate release, accidents and legacy devices found on non-CBRN defensive operations remains credible. In addition, the global deployment of NATO and allied forces means that there may be an increasing number of environmental and industrial hazards (EIH) as well as endemic diseases that may mask any CBRN incident or mimic one.

2. During CBRN defensive operations where the CBRN threat is significant, CBRN incident interventions are focused on both pre-event preparedness (prevent and protect) and post-event mitigation (recovery)[1]. During other operations where the CBRN threat is low, the CBRN defensive posture is likely to be reduced. Any interventions are therefore likely to be a post-CBRN incident response relying heavily on medical support to mitigate, treat and in some cases initially recognise the event. CBRN threat and medical risk are therefore not the same and where there are vulnerabilities the likelihood (or plausibility) and health consequences may be increased.

2.2. ALL HAZARDS SPECTRUM

1. The traditional Nuclear, Biological and Chemical (NBC) threats of a state attack (Article 5 NBC Defensive Operation) has evolved into a CBRNE^{3}T all-hazard spectrum.[2] This concept reflects the additional hazards presented by radiological material, explosives, environmental (including industrial) hazards, and endemic disease (see Figure 2-1). All of these hazards could generate CBRN casualties, or mimic CBRN, that may require a CBRN medical response. For some hazards, a CBRN response may be an appropriate initial response due to safety and forensic considerations.

2. The CBRN weapons end of the spectrum includes:

a. *Chemical agent.* A chemical substance which is intended for use in military operations to kill, seriously injure, or incapacitate personnel through its physiological effects. The term excludes riot control agents, herbicides and substances generating smoke and flame.

b. *Weaponised biological agent.* A biological agent that is deliberately used to produce disease or death in humans, animals, or plants, or which produces materiel deterioration. The biological agent may be a live microorganisms (bacteria, virus or fungus) or a toxin.

c. *Radiological dispersal device (RDD)*. This is a device that causes the overt or covert deliberate spread of radioactive material for the purpose of causing either irradiation, contamination, psychological effect or combination. Some devices may also be dispersed by an explosive device (‘dirty bomb’).

[1] AJP-3.8: *Allied Joint Doctrine for CBRN Defence*.

[2] For medical personnel an all-hazards approach includes chemical, biological, radiological, nuclear & explosives threats and environmental and endemic hazards, and trauma (CBRNE^{3}T).

d. *Nuclear weapon*. A complete assembly (i.e. implosion type, gun type or thermonuclear type) in its intended ultimate configuration which, upon completion of the prescribed arming, fusing and firing sequence, is capable of producing the intended nuclear reaction and release of energy. In terms of casualty care, this will cause a combination of trauma, due to thermal, blast, penetrating and blunt injuries, and radiation casualties.

3. The non-CBRN weapon end of the spectrum includes:

a. *Toxic industrial materials (TIM)*. TIM is a generic term for toxic or radioactive substances in solid, liquid or gaseous form. These may be used, or stored for use, for industrial, commercial, medical, military or domestic purposes. TIM may be chemical, biological or radioactive and described as toxic industrial chemical (TIC), toxic industrial biological (TIB) or toxic industrial radiological (TIR).[3]

b. *Endemic diseases*. Endemic disease is a significant environmental hazard and includes a number of diseases that are present or usually prevalent in a population or geographical area at all times. These diseases form part of a spectrum with weaponised biological agents that can be referred to as *biological agents of operational significance* as they may cause or have historically caused disease that degrades the operational effectiveness of an individual or an organisation. As NATO operations move outside continental Europe, endemic disease will have a greater impact on medical support missions. In some regions, the presence of endemic pathogens (e.g. malaria) may delay the recognition of a biological attack while the severity of some diseases may mimic an attack. NATO nations may also be called upon to support international responses to *public health emergencies of international concern* such as a viral haemorrhagic fever outbreak or emerging disease (see Chapter 17). Some endemic diseases could also be used as a biological weapon.[4]

c. *Emerging disease*. An emerging disease is one that has appeared in a population for the first time, or that may have existed previously but is rapidly increasing in incidence or geographic range [World Health Organisation (WHO)]. This remains a challenge to any medical organisation as the characteristics of the agent will be unquantified until a case series has been established. Laboratory diagnosis will also be difficult particularly where there is a reliance on molecular biology and especially sequencing.

Note: Although some hazards are at the non-offensive end of the spectrum, it does not mean that they are less of a hazard than agents intended for offensive use. In some cases, they may have a greater impact on the deployed force than a limited CBRN attack, e.g. pandemic influenza or viral haemorrhagic fever.

4. *Explosives*. Military explosives as well as homemade explosive (HME) substances are an important component of the spectrum as their use, deliberate or accidental, may:

a. Be a method for the release of a CBRN agent or TIM.

[3] The term *Environmental and Industrial Hazards (EIH)* is sometimes used to refer to this group as well as endemic disease and other non-CBRN hazards such heat, cold and noise.
[4] Toxins may also be considered as an endemic threat although they would be associated with a living organism (e.g. infection, ingestion and envenomation) rather than isolated for weapons use.

b. Allow for the opportunistic absorption of a CBRN agents through wounds with the additional casualty hazard of wound contamination.

c. Compound the effects of attack or the release by masking the non-conventional (non-trauma) effects or increasing the severity of the combined effects.

5. *Trauma*. As well as explosive (blast) injuries, medical personnel should be able to manage any life-threatening trauma (deliberate or accidental) and perform life-saving interventions (LSI) in a CBRN environment. This may be either in the presence of a CBRN agent or in a high-threat environment where there is a requirement to wear protective equipment.

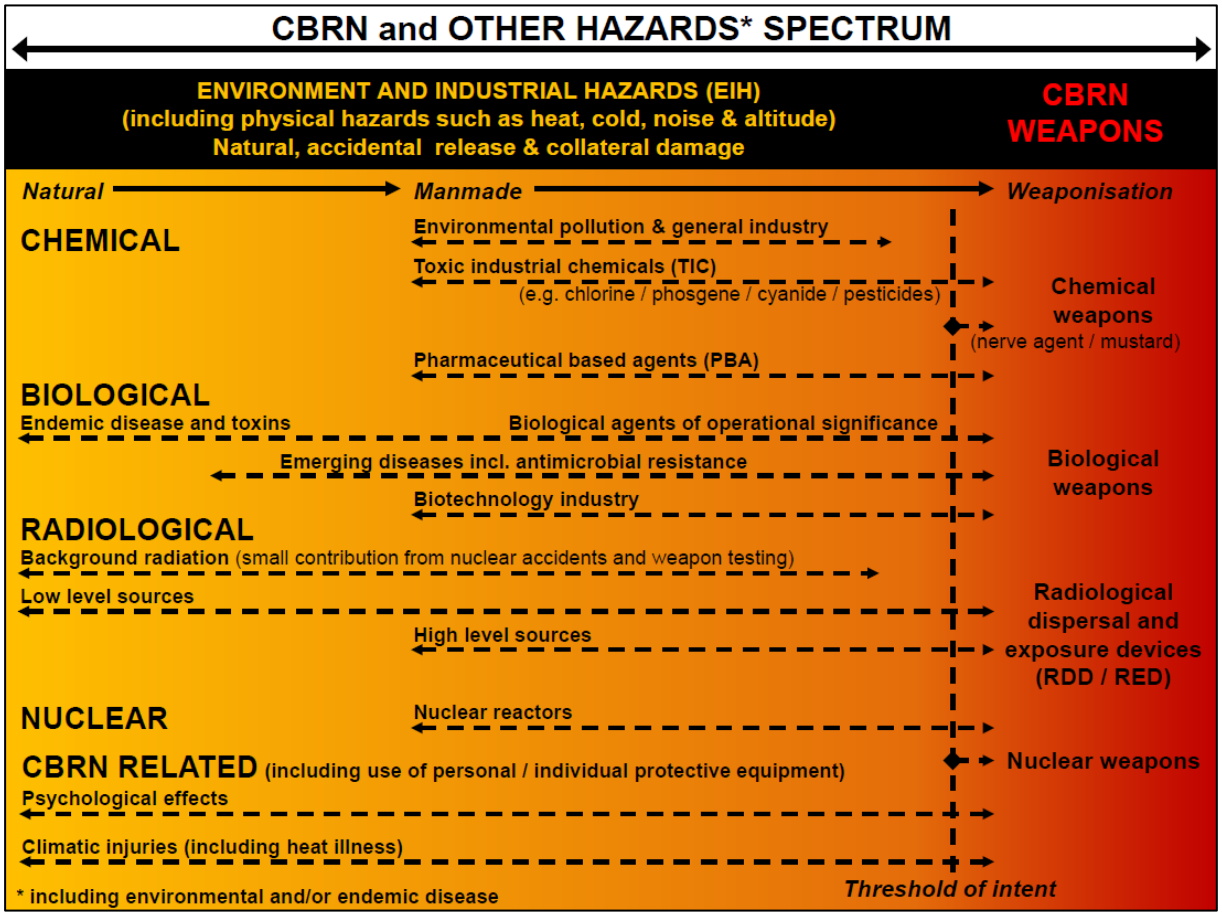

Figure 2-1 – All Hazards Spectrum.

2.3. HISTORICAL REVIEW OF CBRN EVENTS

1. The use of non-conventional weapons dates back to antiquity from the use of poisoned arrows and sulphur. The mass use of chemical weapons was seen between 1915-18 with the use of toxic industrial chemicals (TICs) such as chlorine and phosgene as chemical warfare agents (CWA) and the development of agents with the sole purpose of offensive use (sulphur mustard and lewisite). During the late 1930s and Second World War chemical, biological and nuclear weapon research was conducted with regional offensive use of biological weapons. 1945 saw the use of the first nuclear weapons over Hiroshima and Nagasaki. During the Cold War, CBRN offensive and defensive programs continued and ongoing demilitarisation and disarmament operations continue to date. In the Middle East, the use of CBRN weapons has

been seen in conflicts both between states (Iran-Iraq War), as part of ethnic conflict (use of CWAs against the Kurdish population in northern Iraq, 1988) and internal war (Damascus, 2013).

2. CBRN proliferation may result in the non-state use of CBRN weapons that may be targeted against military and civilian targets. Examples include the use of sarin in Matsumoto (1994), Tokyo (1995), anthrax in the United States (2001), and the opportunistic use of chlorine in Iraq (2007). The use of CBRN agents by insurgents and terrorist organisations should be considered in any medical planning with emphasis likely to be on post-event interventions.

> *"Terrorism poses a direct threat to the security of the citizens of NATO countries, and to international stability and prosperity more broadly. Extremist groups continue to spread to, and in, areas of strategic importance to the Alliance, and modern technology increases the threat and potential impact of terrorist attacks, in particular if terrorists were to acquire nuclear, chemical, biological or radiological capabilities"*
>
> Quote from Active Engagement, Modern Defence: Strategic Concept for the Defence and Security of the Members of the North Atlantic Treaty Organisation (Nov 2010)

3. Non-conventional (CBRN) agents may also be used to target individuals or small groups for assassination as well as trial runs for a larger attack. Examples of agents used for assassination include the toxin ricin and the radioisotope Polonium-210. Water and food security remain important for force protection, and assessment and management of the unusual individual casualty will use many of the principles for any other CBRN casualty.

2.3.1. NATO OPERATIONS AND POSSIBLE THREATS

The type of NATO operation will determine the CBRN defence posture adopted for the medical support mission. Military operations range from war with CBRN capable states including "collective defence" under Article 5 through to Non-Article 5 Crisis Response Operations (NA5CRO) which may include civil-military co-operation (CIMIC).

2.4. GENERAL CHARACTERISTICS OF CBRN AGENTS

2.4.1 NON CLINICAL CHARACTERISTICS

These characteristics determine the requirement and type of personal protective equipment, including individual protective equipment, and decontamination.

a. *Physical*. Known chemical agent and radioisotopes cover the whole range of physical properties: *gaseous, liquid or solid*. Biological agents are usually particulates although they may contaminate a liquid. The volatility of a liquid will determine whether the liquid evaporates in the ambient conditions to be an airborne *vapour* hazard, and therefore become an inhalational hazard. Particulate solids and aerosolised liquids may be suspended in the air depending on ambient conditions and be an airborne hazard. The physical properties of an agent determine whether an agent is a persistent hazard and whether direct contact with the agent (contamination) requires decontamination. Further information on *persistency* and *casualty hazard management* is found in Chapter 6.

b. *Chemical*. The inherent reactivity and stability of chemical agents can vary widely. Some chemically reactive agents denature rapidly, whereas other less reactive agents require bleach solutions or other active skin and equipment decontaminants to inactivate

them. Solid adsorbents e.g. fuller's earth can be used as a decontaminant but do not chemically destroy the chemical agent and the potential for off-gassing should be recognised. Chemical properties are not limited to chemical agents and can be applied to toxins and radioisotopes. The chemical properties of radioisotopes can be exploited to chemically remove an incorporated radioisotope (*decorporation*).

c. *Microbiological properties*. Biological agents have properties that are described in more detail in Part 4. Common properties with other CBRN agents include persistency in environment and requirement for decontamination. Other properties such as transmissibility and risk from contagious illness are specific to biological agents alone.

d. *Radiological properties*. Radiological properties are specific to the radioisotope and will be discussed in Part 5. The chemical properties of the stable and radioisotopes of an element are the same and can therefore be exploited for treatment. For example, the chemical properties of radioiodine (^{131}I), including its uptake by the thyroid gland, is the same as stable iodine (^{127}I). Radioisotopes cannot, therefore, be chemically destroyed and will require external decontamination using physical methods, or removed internally by *decorporation*.

Note: Exposure to ionising radiation (irradiation) without contamination does not require decontamination.

2.4.2. PATHOPHYSIOLOGICAL (MEDICAL) CHARACTERISTICS

1. The medical effects of each agent depend on the a number of factors, these include:

   a. Route of absorption.

   b. The behaviour of the agent in the body including absorption, distribution, localisation in tissues, biotransformation and excretion (kinetics).

   c. The pathophysiological effect at the site of effect (dynamics).

   d. Initial state or pre-existing conditions of the exposed person.

   e. Concentration or dose.

   f. Duration of exposure.

2. The medical effects of CBRN agent and trauma exposure will have different clinical effects, although the effects are not exclusive. Clinical assessment of the casualty will look for symptoms and signs specific to each hazard type – the '*casualty effects'* ('Four Is'):

   a. Intoxication (chemical / toxins).

   b. Infection (live biological agents).

   c. Irradiation (radiation / nuclear).

   d. Injuries (explosives / trauma).

3. The severity of the medical effects of CBRN agents can be defined as:

   a. *Lethal*. Examples include nerve agents, cyanide, phosgene, inhalational anthrax, pneumonic plague, toxins and high dose radiation. Lethality can be expressed as LD_{50},

which is the lethal dose to kill 50% of the exposed population, or LCt_{50} for inhaled chemicals which is based on concentration and duration of exposure.

b. *Damaging*. These agents have a low mortality in those exposed to < 5% during conflict, but will have a significant impact on medical support. Examples include sulphur mustard and low dose radiation.[5]

c. *Incapacitating*. These agents cause a reversible mental or physical disability and inability to function. An example of mental incapacitants are BZ and LSD, while examples of physical incapacitants are adamsite (DM) (vomiting agent), Influenza virus and *C. burnetii* (Q fever). For some agents, an incapacitating dose (ID_{50})[6] has been calculated and includes sub-lethal doses of some agents described in 3a.

2.4.3. LATENCY OF EFFECTS

1. The onset of symptoms is important in the recognition and establishment of causation of a CBRN or TIM release. However, delay in these symptoms due to a latency period will confound any epidemiological investigation. For live biological agents, the latency period is called the incubation period. A summary is shown in Figure 2-2.

2. The timeframe for the overt clinical effects to be expressed is categorised as:

a. *Immediate onset*. Symptoms and signs that occur during the exposure that may be early enough to allow for protective measures to be taken such as evacuation, wearing respiratory protection or medical countermeasure use.

b. *Acute onset*. Symptoms and signs that occur usually within six hours[7] after the period of initial exposure. Acute effects are likely to use the same response used for traumatic injuries with a similar clinical timeline and evacuation chain.

c. *Delayed onset*. Symptoms and signs that occur usually after six hours. For these cases, the initial event may not be associated with the delayed effects seen. Agents with delayed onset may require special arrangements to be in place to observe and respond to the delayed effects.

d. *Long-term sequelae*. Symptoms and signs that occur in period after the operational employment window (such as six months) where immediate causation may not be recognised but may be diagnosed as part of post-operational health surveillance provided either by military or civilian primary healthcare systems.

3. The latency period is important as this is sometimes referred to as the '*window of opportunity (WOO)*' for the effective use of early post exposure prophylactic MedCM. The WOO can also be applied to a window of effective medical therapy also. The WOO in both scenarios depends on early detection of the initial release ('detect to protect') or medical

[5] ATP-3.8 does not include damaging as a class, however for medical planning this is an important group of casualties – chemical burns. The use of the term 'lung-damaging agents' to describe chemicals such as phosgene and chlorine is ambiguous and therefore the preferred medical term for these agents is 'pulmonary agents'.

[6] The ID_{50} for incapacitating effects should not be confused with the ID_{50} describing the infecting dose associated with live biological agents.

[7] This period is set for guidance and planning including casualty estimation.

personnel recognising symptoms and signs of the effects (‘diagnosis to treat’), direct or indirect respectively.

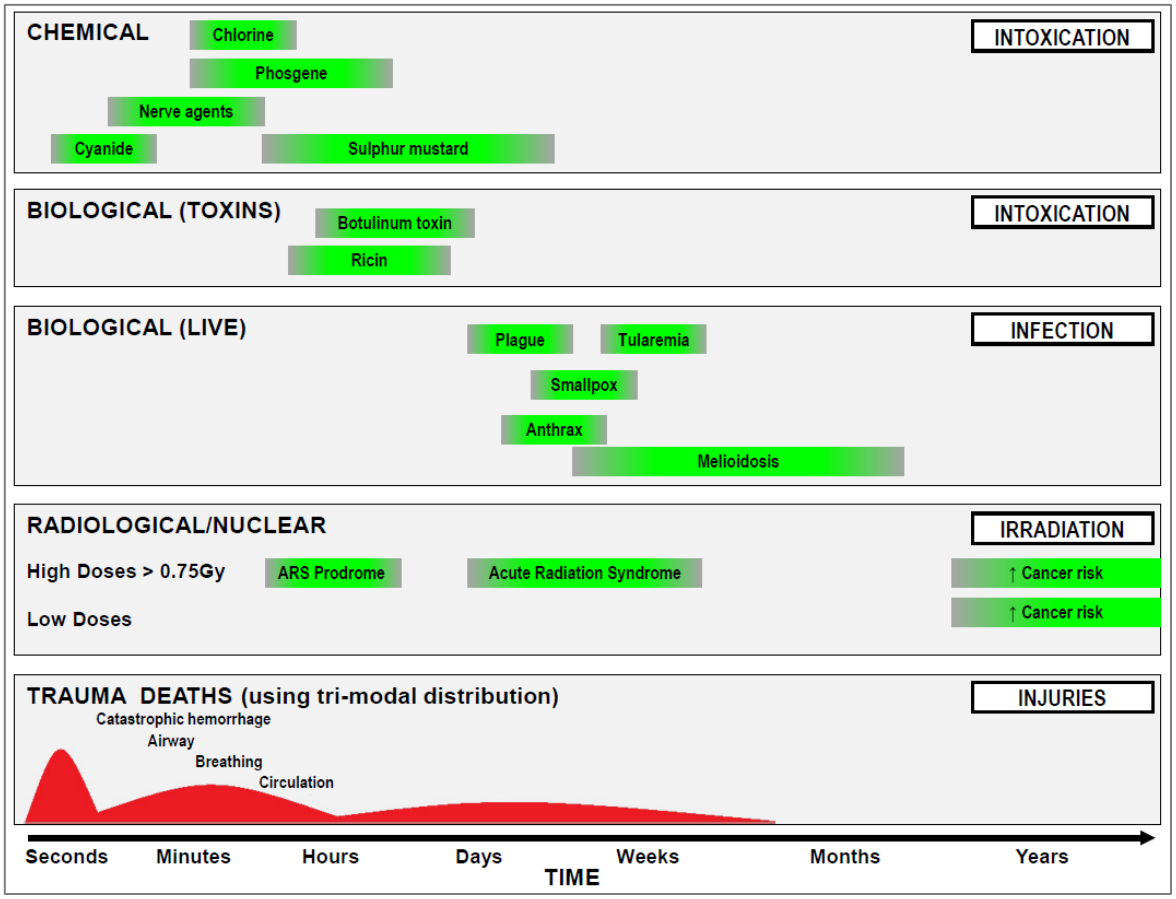

Figure 2-2 – Illustrative Summary of CBRN Onset of Symptoms.

2.5. METHODS OF DELIVERY

1. The characteristics of a CBRN agent, especially physical and stability characteristics and vulnerable routes of absorption, will determine the delivery method used. The optimal method will maximise the number of casualties and the effects of the exposure via the optimal route of absorption and penetration of any physical protective measures. The delivery method used may also cause additional medical effects such as trauma (injuries) or psychological stress. The method of delivery may be:

a. *Overt*. This method may use of an obvious method of delivery such as munitions, bombs, projectiles, spray tanks and warheads. A release of a large quantity of agent may also be indicative of an attack, such as a cloud of chlorine.

b. *Covert*. Covert methods, including the use of insect vectors, mean that casualty presentations have no obvious causation and investigations will require an effective inform process using epidemiological methods as described in Chapter 17. Covert methods may become detected if a real-time detection network is in place with a warning time short enough to ‘detect to protect’ where protection may be physical or post-exposure medical countermeasure.

2. The increasing asymmetrical threat from insurgents and terrorists means that improvised explosive devices (IED) may be used as well as CBRN only overt and covert releases. In cases of explosive incidents, the release of a CBRN agent may be masked by the obvious injuries. CBRN release should be suspected based upon intelligence, environmental assessment and appropriate screening of casualties by detection equipment.

2.6. ROUTES OF EXPOSURE

There are a number of ways that CBRN agents can get into the body. Some routes are quicker than others for the systemic absorption while some routes are easier to protect by physical protection, or are less permissive to some types of agent. Routes of exposure include:

a. *Inhalation*. Agent (gas/vapour and aerosols) breathed in.

b. *Ingestion*. Agent (liquid, solid) eaten or drunk.

c. *Skin (percutaneous)* by three methods:

(1) *Through intact skin (transcutaneous)*. Agent (liquid, gas, solid) is absorbed through intact skin without initially breaking it although absorption may be slower by this route and vary depending on blood flow and thickness. Unbroken skin is resistant to most biological agents. Normal field hygiene should be used.

(2) *Inoculation (or injection)*. Any penetration of the skin with minimal trauma in order to introduce the agent (liquid, solid). This route includes vectors such as arthropods transmitting biological agents and is also a common route for the administration of vaccines and auto-injectors.

(3) *Wound*. Any contamination after significant breaking of the skin barrier following a traumatic event.

d. *Eye (ocular)*. Agents (gas/vapour and aerosol) may have a local effect on the eye, such as miosis caused by nerve agent. Significant systemic absorption is less likely or significantly delayed due to a poor corneal blood supply.

e. *Mucosa*. This route, including the conjunctiva, is susceptible to chemical agents such as sulphur mustard, and live biological agents including blood borne and haemorrhagic fever viruses.

2.7. TYPES OF CBRN CASUALTIES

1. The type of casualties that may be generated by a CBRN attack depends on the hazards present, delivery method, persistency of the agent, presence of trauma and CBRN protective measures used. The presence of a secondary exposure hazard to responders due to persistency should also be noted in any casualty report, as well as the clinical state. The descriptions used can also be applied to other incidents and casualties including exposure to TIMs and endemic disease.

2. Casualty types may be defined by two criteria, the cause of the perceived clinical effects (and probable or confirmed cause) and secondary hazard status (contaminated or contagious). Each casualty type is therefore not exclusive.

2.7.1. CLINICAL CASUALTY TYPES

Casualties[8] classified by the clinical effects are:

a. *Chemical casualty*. A casualty caused by a chemical substance.

b. *Biological casualty*. A casualty caused by a biological agent (including toxins).

c. *Radiological casualty*. A casualty caused as a result of exposure solely to ionising radiation either due to irradiation (acute radiation dose) or treatment for internal contamination (decorporation). Nuclear casualties are excluded from this category (see below) as are any potential long term effects of ionising radiation exposure.

d. *Nuclear casualty*. A casualty caused by a nuclear detonation. These casualties are likely to also have concurrent trauma due to flash, thermal burns and blast (combined injuries).

e. *Combined casualty*. A chemical, biological or radiological casualty with concurrent traumatic injuries.

f. *Psychological casualty* (see section 2.8 and AMedP-8.6[9]).

2.7.2. CASUALTY TYPES DUE TO SECONDARY HAZARD RISK

Casualties (and personnel) classified by their secondary hazard status are:

a. *Exposed person*. This is an asymptomatic person that falls within the population at risk (PAR) following a suspected or confirmed release. Exposed persons may remain unaffected or become casualties during the range of the latency period of the agent. Any significant exposure (suspected or confirmed) should be recorded. Contaminated personnel that have been exposed by a potentially transmissible biological agent or in contact with a contagious patient may require quarantine and would be classified as a casualty. Medical advice and follow-up may be required including initial *screening* or long term *health surveillance*.

b. *Contaminated casualty*. A casualty that has been exposed to a persistent CBRN agent, externally, internally or into a wound, that remains or may be a secondary hazard to responders and is symptomatic. These casualties will require casualty decontamination, and may require decorporation (removal of internal contamination) and wound decontamination as part of casualty hazard management.

c. *Contagious casualty*. A biological casualty that has been infected with a transmissible (person to person) microorganism. These casualties will require assessment for isolation as part of casualty hazard management.

2.8. PSYCHOLOGICAL CASUALTIES

This casualty type is complex. Symptoms range from mild anxiety and acute stress reaction through to mental incapacitation due to reduced level of consciousness, delirium or psychosis. Symptoms may also be delayed or long-term and include post-traumatic stress disorder.

[8] For the purposes of this medical allied publication - a casualty is described as “a person who is lost to an organisation by reason of having been declared dead, wounded, injured, or diseased”.
[9] AMedP-8.6: *Forward Mental Healthcare*.

Psychological effects due to CBRN agents may be direct or indirect. In most cases, symptoms may be present without exposure to an agent and this is called *psychogenic*, or *sociogenic* if presenting as multiple casualties with similar symptoms but no obvious cause.

2.8.1. DIRECT PSYCHOLOGICAL EFFECTS

The direct effects of CBRN agents include:

a. *Delirium*. This is an acute, transient disturbance of consciousness with reduced ability to pay attention to external stimuli, and disorganised thinking with rambling or incoherent speech caused directly by the agent. Some agents may cause hallucinations and are described as psychoactive or psychotropic.

b. *Sedation*. This is a reduction of consciousness that is a form of mental incapacitation. The mild form will lead to psychological impairment and the most severe form is coma and potential death due to the suppression of vital brain functions such as respiratory drive.

2.8.2. INDIRECT PSYCHOLOGICAL EFFECTS

The indirect effects of CBRN agents may be due to a psychological response or actions to protect against their effects, such as wearing protective equipment. They include:

a. *Reactive*. These casualties range from an appropriate level of anxiety with low risk ('worried well') to acute stress reaction, acute stress disorder and post-traumatic stress disorder.

b. *Degradation*. These are the psychological effects of wearing IPE and may be caused by claustrophobia, poor communications and isolation, or a delirium secondary to heat illness (heat stroke).

2.8.3. PSYCHOGENIC (SOCIOGENIC)

These casualties have symptoms and possible signs of exposure but in the absence of any CBRN exposure and are psychological in origin. The simultaneous presentation of a large number of casualties with this phenomenon, *mass psychogenic (sociogenic) illness* is well described and may present in a similar location to or soon after a confirmed or suspected CBRN event.

CHAPTER 3: MEDICAL COUNTERMEASURES

3.1. INTRODUCTION

MedCM are pharmaceutical products designed to diminish the susceptibility of personnel to the lethal and damaging effects of chemical, biological and radiological hazards and to treat the effects arising from exposure to such hazards. MedCM maintain commanders' freedom of action and combat effectiveness. While they may be used as a standalone CBRN protective measure, they will usually be used as a part of the CBRN defence capability especially with other components such as detection, warning and reporting, and individual protection.

3.2. TYPES OF MEDICAL COUNTERMEASURES

1. MedCM are generally pharmaceuticals that have protective properties against specific CBRN agents or types of agents, or non-specific effects such as vomiting or convulsions. Examples of pharmaceutical types include:

a. *Antidotes*. These are pharmaceutical agents that counteract a chemical agent.[1] Antidotes may work in a number of ways. They may act to reverse the effect of the chemical agent at the site of action (toxicodynamics) such as oximes, or may act to antagonise the effects of the chemical agent toxicity (atropine and diazepam). Some antidotes act to alter the distribution or increase the metabolism and elimination of the chemical; examples include chelating agents.

b. *Antibacterials* (commonly called antibiotics). These pharmaceuticals provide inhibit or kill sensitive bacteria. Their use may be either as self-administered pre- or post-exposure prophylaxis, or by medical personnel for casualty care.

c. *Antivirals*. This group of anti-microbials is less well developed than antibacterials but subject to continuing research and clinical trials.

d. *Antitoxin*. These are antibodies (immunoglobulins) raised against specific toxins. Dosage may be based on toxin dose with multiple dosing required. There is a risk of anaphylaxis and serum sickness using animal derived antitoxin. The use is limited to probable or confirmed cases rather than routine empirical prophylaxis and there may be a relatively short window of opportunity.

e. *Passive immunisation*. Immunoglobulins (antibodies other than antitoxin) or parts of them are transferred to a person so that the body does not need to produce these elements itself. This method of immunisation begins to work very quickly, but it is short duration because the antibodies are naturally broken down and they are not further reproduced by the recipients. Antibodies for passive immunisation continue to be developed against certain biological agents and there are currently vaccinia and anthrax immunoglobulin available in some countries.

f. *Active immunisation (vaccination)*. This encourages the recipients to develop their own immune response to a biological agent including toxins. Multiple doses may be required before maximal protection over a long period (up to six months). This is usually administered by medical personnel although authorised by the chain of command. Some

[1] The term antidote is sometimes used to describe antitoxins including those for CBRN as well as drug overdose and other poisons.

vaccines (anthrax) may have a protective effect if given early post-exposure and contribute to greater protection by other MedCM such as antibiotics.

g. *Cytokine (stimulation) therapy*. This treatment uses cytokines to stimulate the casualties own immune system in order to fight the infection. Some of these treatments are also used in the management of acute radiation syndrome.

2. There are four concepts of use for MedCM. A specific MedCM is not limited to one concept type (i.e. antibiotics). The types are defined as:

a. *Pre-exposure prophylaxis*. Pre-exposure prophylaxis (PrEP) describes the administration of MedCM before detection of an exposure in order to prevent the effects of a CBRN agent. These may be given days, weeks or even months in advance. An example would be vaccines to protect against biological agents. Pre-exposure MedCM do not rely on detection to trigger implementation as this is based upon the CBRN threat, risk:benefit and acceptability.

b. *Pre-treatment*. Pre-treatments are therapy enhancers that are administered before exposure to enhance the efficacy of subsequent post-exposure therapy. An example is pyridostigmine for the enhancement of nerve agent immediate therapy.

c. *Post-exposure prophylaxis*. Post-exposure prophylaxis (PEP) is a medical countermeasure used after an exposure has been detected in order to prevent the effects of the CBRN agent. It requires a detect capability in order to take advantage of the *window of opportunity* (WOO) between exposure to an agent and the development of any irreversible consequences. Examples include antibiotics and vaccination after potential exposure to biological agents. The WOO is maximised by early detection or diagnosis and rapid MedCM administration and varies by agent and MedCM type e.g. Longer WOO for live biological agents and antibiotic prophylaxis compared to a shorter WOO for toxins and antitoxin use.

d. *Immediate therapy*. Immediate therapy is a medical countermeasure used to treat the initial effects of a CBRN agent based upon symptoms and signs. Immediate therapies may be administered by non-medical as well as medical personnel. These may be given by the casualty themselves (self-administered) or as first aid by another (buddy-administered). Some training is required in recognising early symptoms and signs, as well as the actual method of MedCM administration (“diagnose to treat”). Examples include self-administered medical products used when the effects of CBRN agents are evident (e.g. nerve agent antidote auto-injectors).

e. *Continuing medical therapy*. This refers to pharmaceuticals that are given within the medical chain. This group of MedCM is important to consider in order to ensure interoperability, along the casualty chain and between MTFs, and prevent adverse drug interactions with self-administered MedCM. Hospital care may be delivered by a different nation to the patient’s nation or a nation providing pre-hospital emergency care.

3. The range of MedCM may be used at any time during the career of a military person from new entry through to post exposure. Some MedCM such as vaccines may be part of the wider Force Health Protection program. For a specific operation, the beginning of the force generation period through to the end of the WOO is described as the MedCM employment envelop. The key events within the envelopment are the time of exposure, detection and diagnosis. The last two events of post exposure triggers for a command decision to be made

on the use of a MedCM; this is unless pre-authorisation has already been made for personnel to self-administer on the appropriate trigger.

4. A summary of pharmaceutical types and concept of use is shown in Figure 3-1.

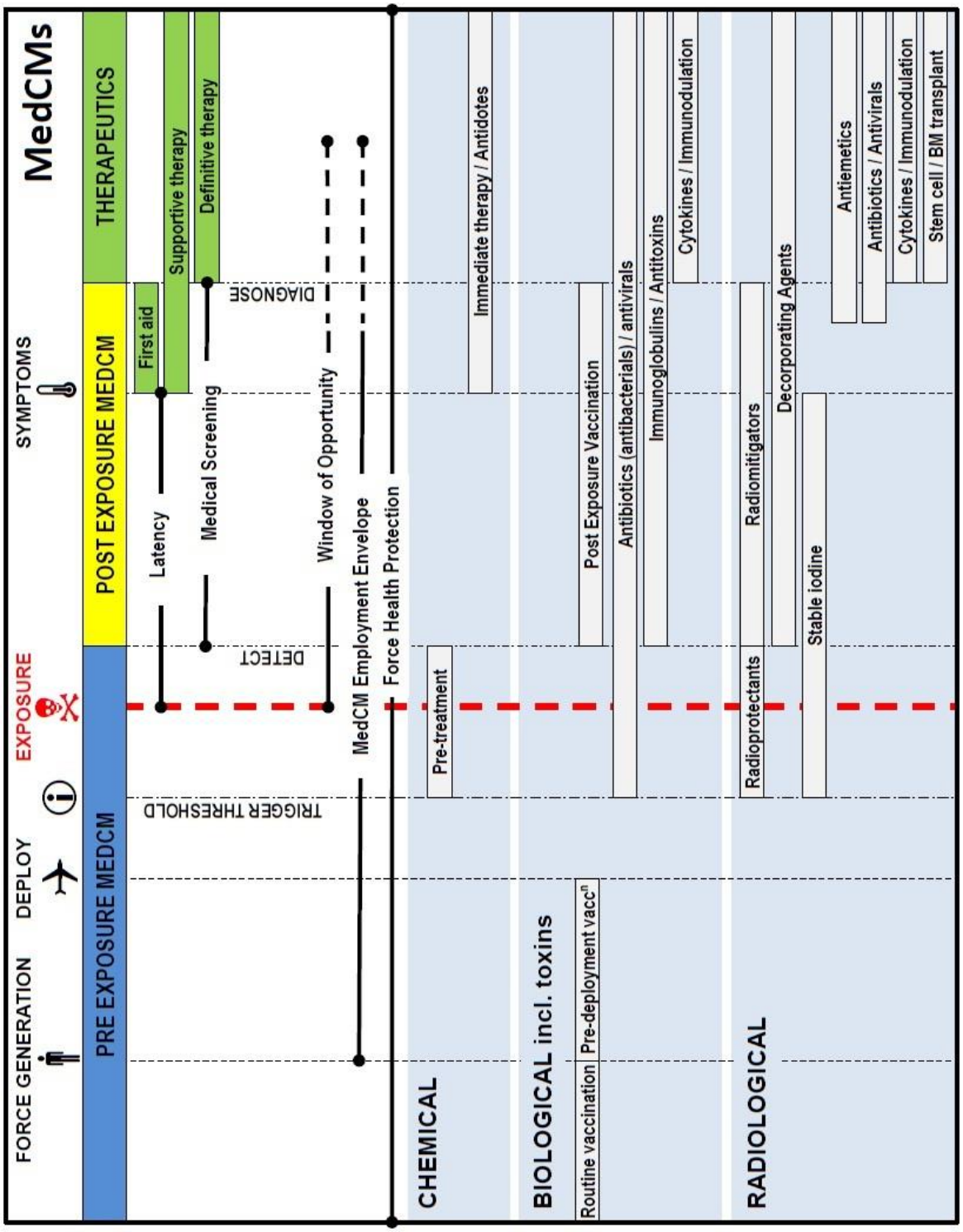

Figure 3-1: Summary of CBRN MedCM types.

3.3. IMPLEMENTATION OF MEDICAL COUNTERMEASURES

1. During any operation or in preparation, there are a number of decision points for the implementation of MedCM. The decision to start MedCM will usually be a command decision supported by medical advice. The exception is the use of immediate therapy for casualty treatment. These decisions include whether and when to:

a. Implementation of a vaccination programme to a specific threat.

b. Training personnel in the use of self-administered MedCM.

c. Training medical personnel in the use of medically administered MedCM.

d. Issuing MedCM to personnel.

e. Starting pre-exposure MedCM.

f. Starting post-exposure MedCM.

g. Stopping MedCM.

2. Further guidance to command on the implementation of MedCM can be found in AMedP-7.6.

3.3.1. VACCINATION

Vaccination policies will vary between nations due to issues such as concept of use, licensing and regulatory constraints. Vaccination provides significant protection for specific biological agents as well as endemic disease. Some vaccines require multiple doses over a period of time that may exceed the time allocated for force generation. Where possible, vaccination should be implemented in conjunction with the immunisation programme as part of wider force health protection before deployment. Some vaccines may not provide 100% protection and may require additional MedCM. For planning purposes including casualty rate estimation, an efficacy of 90% is usually assumed unless supported by other scientific information.[2]

3.4. RECORDING THE USE OF MEDICAL COUNTERMEASURES

1. The use of any MedCM whether self-administered or by medical personnel should be recorded in the person's health record.

2. Any adverse drug reactions (not recognised side effects) should also be reported to the relevant medical/drug regulatory authority in accordance with national legislation and licensing.

3.5. CBRN MEDICAL COUNTERMEASURES PLANNING

Further guidance on CBRN medical planning and the operational considerations for MedCM deployment, issuing and use will be found in medical planning publications of the AMedP-7 series including AMedP-7.5 and 7.6.

[2] AMedP-7.5 provides more specific estimated vaccine efficacy for casualty rate estimation.

CHAPTER 4: PRINCIPLES OF CBRN CASUALTY MANAGEMENT

4.1. INTRODUCTION

1. The management of CBRN casualties requires a standardised framework. This optimises medical response by supporting prioritisation, personal safety and interoperability between units and nations. A common framework supports personnel with different casualty management roles (command, clinical, detection, decontamination and logistics) and capabilities.

2. The principles of CBRN casualty management are:

a. Recognition (Detect & Diagnose).

b. Safety (see Chapter 9).

c. First-aid including self-administered and buddy-administered (see AMedP-7.2).

d. Triage (see Chapter 14).

e. Casualty assessment.

f. Life-saving interventions (LSI).

g. Casualty hazard management (contain, decontaminate, isolate & quarantine) (see Chapter 6).

h. Advanced Medical Care

(1) Supportive treatment, including critical care and replacement therapy.

(2) Definitive treatment, including antidotes and surgery.

i. Rehabilitation.

4.2. LEVELS AND PRIORITIES OF CBRN CASUALTY CARE

The level of care and the ability to provide safe and effective treatment will depend on:

a. The training and competency of the non-medical or medical responder individual (see AMedP-7.2 and AMedP-7.3 respectively); and

b. The presence of a CBRN hazard, described as CBRN functional zones[1], with the requirement to wear IPE/PPE.

[1] The CBRN functional zones are described in detail in Chapter 10 but are summarised as:
Hot zone. This is a non-permissive area where there is a direct hazard (primary exposure and contamination) to the responder from the environment.
Exclusion zone. This is a type of hot zone where despite protective measures a significant risk to the responder remains that can only be reduced by avoidance (e.g. high dose radiation source or explosive device).

4.2.1. LEVELS OF CARE

The delivery of casualty care is a continuum of care starting at the point of exposure (PoE) through to a Role 4 hospital and rehabilitation. The level of care able to be provided will vary. It depends on the operational environment, availability of medical resources and the requirement to wear personal protective equipment due to a CBRN hazard. The levels of care are:

a. (Enhanced) First Aid.

b. Emergency Medical Treatment (EMT).

c. Advanced Medical Care (AMC).

d. Rehabilitation.

4.2.2. PRIORITIES FOR TREATMENT IN A CBRN ENVIRONMENT

1. A generic all-hazards approach adapted from the management of trauma is applied to CBRN casualty management including the management of trauma in a CBRN environment. The priorities for treatment through the continuum of care, although more limited in the hot zone, are:

<C>atastrophic haemorrhage.

Airway and **a**ntidote (MedCM) administration.

Breathing and oxygen delivery, where appropriate.

Circulation.

Decontamination (and **d**isability).

Evacuation to a more permissive environment.

2. The times to reach each level of care described follow a similar timeline as conventional casualty management set by MC 326/3.[2] These timings are for medical planning purposes and it is accepted that they will be limited by a CBRN environment. For CBRN defensive operations, medical planners should consider the forward deployment of medical personnel and equipment although any damage control surgery within 2 hours will be a challenge where there is a contamination hazard delaying medical evacuation.

4.3. RECOGNITION

This is the first indication of a CBRN event and may be initiated by any persons recognising environmental signs or casualties, casualties themselves experiencing symptoms (self-aid) or

---

Warm zone. This is a semi-permissive buffer area where there is a secondary contamination hazard due to contaminated equipment, personnel or casualties leaving the hot zone. The warm zone is bounded by a cordon that is called the clean / dirty line (CDL).
Clean (Cold) zone. The clean zone is the area beyond the CDL with optimal access to casualties following external decontamination. A residual risk from contaminated wounds and a contagious patient still exists in this zone.

[2] MC 326/3 NATO Medical Support Principles and Policies.

medical personnel recognising the symptoms described by the casualty or observing the signs seen during casualty (clinical) assessment. Once a CBRN event (release) has been recognised, CBRN medical incident management may begin working with the chain of command (see Part 2).

Note: For biological events, the first recognition is most likely to be through the medical chain either by the diagnosis of an individual case or recognition of an outbreak due to an increase casualty rate or disease pattern recognition.

4.4. TRIAGE

1. Although triage is generally used only for situations with multiple casualties, the use of a triage category is useful for prioritisation of treatment, decontamination, and casualty transport as well as reporting. The same triage categories (T1 (immediate), T2 (urgent), T3 (delayed), T4 (expectant) and Dead) used for conventional casualties are recommended, especially when operations may have conventional, CBRN and combined casualties. The triage criteria for each category and event may differ.

2. Further details of CBRN Triage and categories are in Chapter 14.

4.5. CASUALTY ASSESSMENT

1. Following the recognition and identification of T1 casualties during triage, assessment of chemical and trauma casualties is critical to diagnose and treat any life threatening condition. This early diagnosis of a chemical agent by casualty assessment focused on the assessment of the casualty's Conscious level, Respiratory pattern, Eyes, Secretions and Skin (CRESS) and is called 'Quick Look'.

2. Specific indicators will be given of each chemical agent type in Part 3 of this publication but a short version is given in Table 4-1 highlighting agents (and MedCM side-effects) with specific toxidromes and potential for MedCM and other interventions in the hot and warm zone.

Note. Symptoms and signs may vary with specific agents, doses and routes of exposure.

Table 4-1: Abbreviated 'Quick Look' Assessment (CRESS).

	NERVE	CYANIDE	OPIATE	ATROPINE	SEPSIS	HEAT STROKE
Conscious	Convulsions	Unconscious / Convulsions	Reduced / Unconscious	Agitated / Confused	Normal, reduced or agitated	Altered
Respiration	Increased, reduced or stopped	Increased or stopped	Reduced / Stopped	Increased	Increased	Increased
Eyes	Pinpoint pupils*	Normal / Large pupils	Pinpoint pupils	Large pupils / Blurred vision	Normal	Normal / Large pupils
Secretions	Increased	Normal	Normal	Dry mouth / Feeling of thirst	Normal / sputum	Normal
Skin	Sweaty	Pink then Blue	Normal / Blue	Flushed	Warm / Pale (cool)	Varied
Other potential features	Vomiting Incontinence Slow pulse	Sudden onset			Fast pulse Fever (>38.3°C) Bio-syndrome*	High temperature (>38°C)

* Pinpoint pupils may not be present immediately if skin absorption or eye protection worn.
* Bio-syndromes include respiratory, cutaneous, lymphadenopathy, haemorrhagic, gastrointestinal & neurological syndromes.

3. In the presence of trauma, a rapid trauma primary survey will also be carried out with emphasis in the hot zone on catastrophic haemorrhage, airway and breathing difficulties, or respiratory distress/respiration problem including respirator fault (when unable to assess airway and differentiate from breathing difficulty or chest trauma). Both concepts are also part of first aid assessment in a CBRN environment (see Figure 4-1).

4.6. FIRST AID IN A CBRN ENVIRONMENT

1. First aid is the provision of care to trauma and CBRN casualties to save life or sustain an operation by non-medical or medical personnel. First aid should be delivered as soon as safe to do so and ideally within the first 10 minutes of recognition. In a CBRN environment this is likely to be in the most contaminated, high-risk or even non-permissive environment (hot zone).

2. First aid interventions should focus on the control of catastrophic haemorrhage, basic airway management, early MedCM (antidote) delivery including oxygen (where available), basic chest (breathing) injury management and evacuation to a more permissive environment. This can be summarised as <C>AaB-Evac as part of the continuum of care described above.

3. Additional first aid skills (enhanced first aid) may be considered for CBRN specialists at greater risk of encountering a CBRN incident. Further details of the requirement for first aid in a CBRN environment including training requirement and first aid materials can be found in AMedP-7.2: *CBRN First Aid Handbook* and supporting Standardisation Related Document (see QR code link).

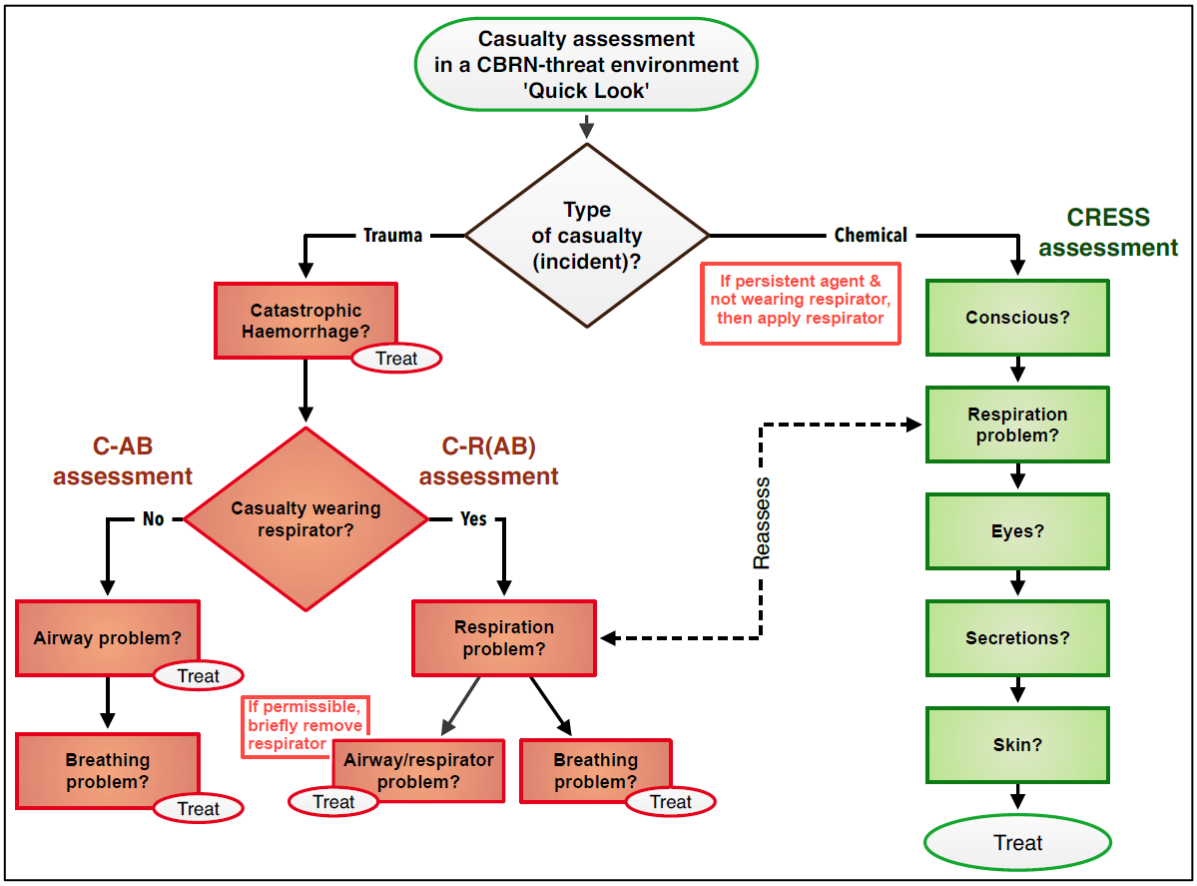

Figure 4-1: CBRN First Aid Assessment 'Quick Look' and Initial Management.

4.7. EMERGENCY MEDICAL TREATMENT

1. The provision of forward life-saving treatment to CBRN casualties including trauma, before and during any decontamination, is described as Emergency Medical Treatment (EMT). EMT provision to T1 casualties should start within an hour of recognition in a semi-permissive (warm or decontamination zone) CBRN environment area. EMT includes:

a. Triage in order to identify T1 casualties.

b. Rapid casualty assessment ('Quick Look').

c. Provision of LSI to trauma and CBRN casualties (see below).

d. Casualty hazard management; those providing EMT may also be required to provide casualty decontamination as part of casualty hazard management or work with those providing that capability (see Chapter 6).

2. EMT (care under fire) in a non-permissive (hot zone[3]) area is limited due to a significant hazard including CBRN agent (primary exposure), explosive device, effective fire or environmental hazard. Treatment may be restricted by personal protective equipment and access to the casualty due to distance or the casualty wearing IPE and combat body armour.

[3] In the context of a CBRN incident, a hot zone is defined as an area where there is a direct exposure or contamination hazard from the environment (for further details see Chapter 10).

Within the hot zone, EMT may be limited to first aid with the priority to evacuate to the warm zone and a casualty collection point or Role 1 MTF. A summary of EMT is in Figure 4-2.

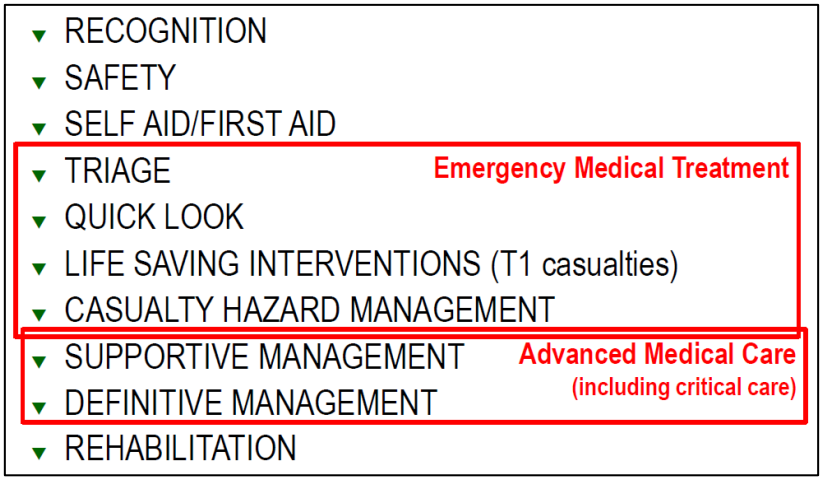

Figure 4-2: CBRN Emergency Medical Treatment and Advanced Medical Care Concepts.

4.7.1. LIFE THREATENING CONDITIONS

Immediate life-threatening conditions that may require immediate treatment and be a challenge in a CBRN environment include:

a. Catastrophic haemorrhage.

b. Airway obstruction.

c. Breathing difficulty.

d. Cyanosis.

e. Respiratory distress.

f. Tension pneumothorax.

g. Sucking chest wound.

h. Circulatory failure.

i. Shock due to fluid loss, cardiac toxicity, sepsis and vasodilatation.

j. Bradycardia (< 40 beats per minute) due to nerve agent.

k. Neurological disability

l. Unconsciousness.

m. Convulsions.

n. Other signs of severe chemical intoxication, where an effective MedCM exists and a delay in administration may cause death.

o. Severe sepsis or septic shock.

p. Heat stroke.

4.7.2. LIFE-SAVING INTERVENTIONS

1. Life-saving Interventions (LSI) are actions that can be performed by any person appropriately trained to reverse any life-threatening condition or prevent further deterioration in a casualty due to a CBRN agent and/or trauma. In a CBRN environment, LSI should only be performed on the most severe casualties (T1) with immediate life-threatening conditions and include:

a. Removal from the hazard (self-extraction or rescue).

b. Application of pressure dressing (and haemostatic dressings[4]).

c. Application of tourniquet(s).

d. Basic airway management including suction.

e. Ventilation (as resources allow).

f. Early MedCM/antidote administration including anticonvulsant.

g. Application of oxygen.

h. Management of tension pneumothorax, such as needle decompression.

i. Management of sucking chest wound, such as application of dressing with valve.

j. Fluid resuscitation (trauma, sepsis or combined injuries).

k. Management of severe sepsis and septic shock (fluids, oxygen and antibiotics).

l. Management of heat stroke.

Note. *In the CBRN environment, a tension pneumothorax should be suspected in any casualty with a penetrating chest injury (or suspected blast injury) AND respiratory distress or circulatory shock.*

2. The levels of care including likely triage points and delivery of LSI are shown in Figure 4-3.

4.8. ADVANCED MEDICAL CARE

1. This is the provision of supportive and definitive care including critical care and surgery by hospital personnel. Although the timeframe of 2 hours to reach a MTF that delivers damage control surgery is set by MC 326/3, this timeframe may be a challenge if there is a requirement for casualty hazard management. Advanced management consists of:

a. *Supportive management*. During CBRN casualty management, the causative agent may not be known or suspected. Treatment may still be effective by adequate supportive treatment focused on managing the observed clinical symptoms and signs. In some

[4] The application of haemostatic dressings may be impractical in a CBRN environment and for catastrophic limb injuries early use of a tourniquet is advised. For torso injuries, tourniquets are not an option and pressure dressings should be the mainstay of treatment within national guidelines.

cases even when the agent is known, there may be no definitive treatment such as an antidote and treatment may remain supportive throughout the casualty continuum of care including critical care with ventilation, circulatory and renal support.

b. *Definitive management*. Definitive treatment is the final level of comprehensive care provided to return the patient to the highest degree of mental and physical capability possible. After the definitive treatment period the individual may undergo rehabilitation before being returned to duty or discharged from military service. For some casualties, definitive care may only be available at Role 4 or in an allied nation. Types of definitive treatment include further antidote treatment, replacement therapy, as well as surgery and burns management.

3. Specific supportive and definitive treatments including critical care and paediatric patients are discussed in Chapter 7.

4.9. REHABILITATION

Rehabilitation (physical, mental and social) for CBRN casualties is outside the scope of this AP. However, medical services should give consideration to the long term impact of a CBRN incident leading to chronic or long term disease such as burns (thermal, chemical and radiation), eye injuries, psychological effects, increased risk of cancer and potentially teratogenesis. These effects will require involvement with rehabilitation services and specialities including plastic surgery, ophthalmology, mental health services, health (genetic) counsellors, occupational physicians and veteran agencies. Social rehabilitation is also an important consideration especially overcoming stigma and reintegration with family, work and society. This may require risk communication provision not only to the patient but other parties.

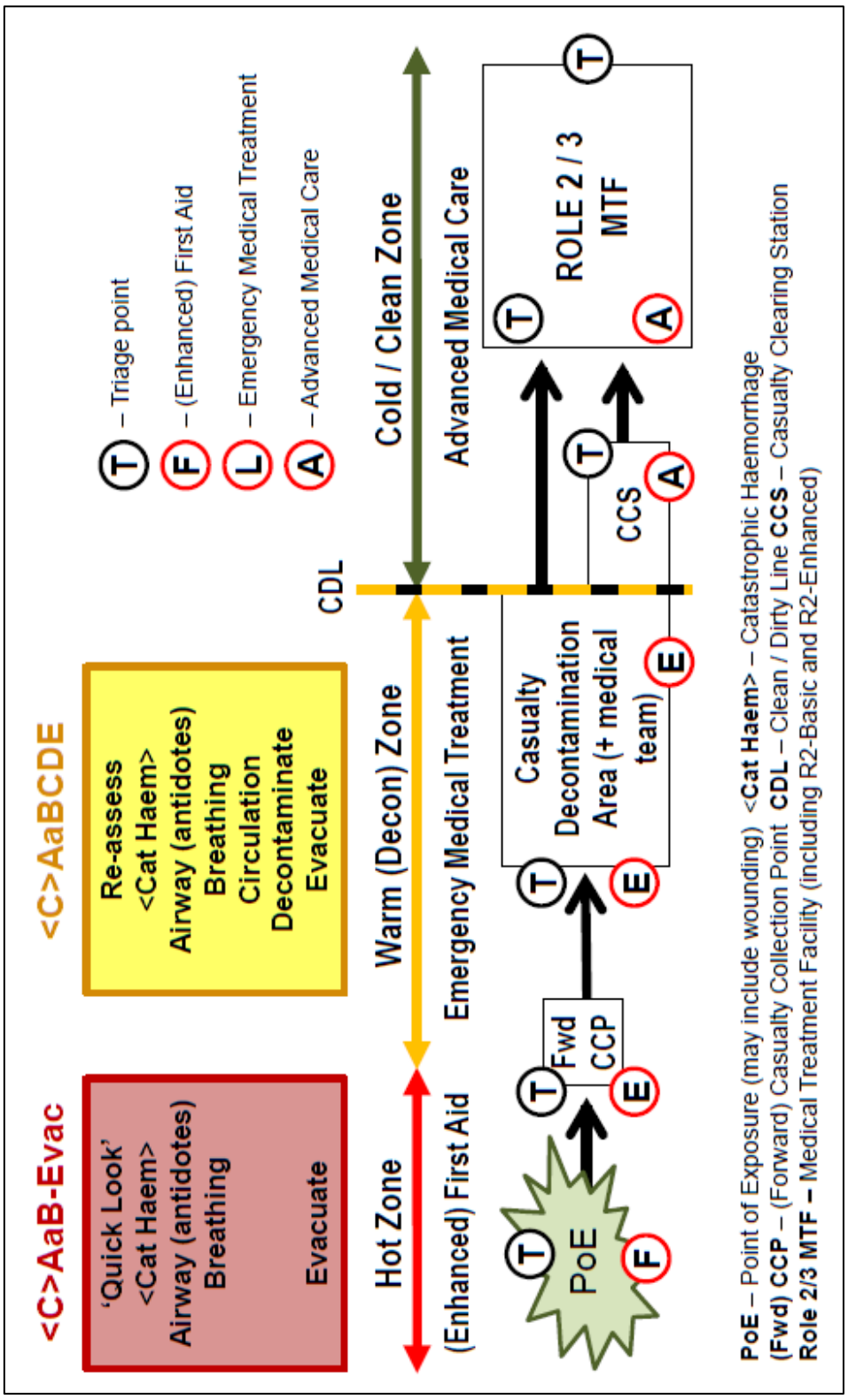

Figure 4-3: Summary of Casualty Management Points and Levels of Care.

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CHAPTER 5: CBRN RECOGNITION

5.1. INTRODUCTION

1. A response to a CBRN event requires an ability to recognise the event. While on a CBRN Defensive Operation, the main element contributing to this will be detection either with equipment carried by personnel or as a deployed sensor network. The priority for these operations is "*detect to protect*" either by use of physical protection (individual (donning respiratory) or collective protection (taking shelter)), and/or by administering MedCM. Medical personnel may also recognise the effects of the CBRN agents as symptoms and signs and therefore start specific treatments; this is "*diagnose to treat*".

Note: For low CBRN threat operations, medical personnel may be the first to see the effects of a CBRN release. In order to optimise recognition, all operational medical personnel that may be involved in casualty management should be aware of the effects of CBRN agents and how they may initially present. For biological agents, medical recognition either by clinical assessment or clinical investigation may be the only method.

2. Indicators of an environmental or CBRN hazard include:

   a. Any symptoms involving incident response personnel or Reconnaissance teams.

   b. STEP 1-2-3 Rule. This is an escalated defensive response based on the number of casualties presenting with similar symptoms and signs.

   c. Unusual taste, smell or mist.

   d. Unexplained dead animals.

   e. Unexplained symptoms including altered vision, eye pain, headache, chest tightness, difficulty in breathing, excessive secretions, non-thermal burns.

   f. Anything unusual or unexplained symptoms and signs.

5.2. CYCLE OF CBRN RECOGNITION

1. A cycle of CBRN recognition is described as a cyclical relationship between the elements of recognition (Figure 5-1). These elements are:

   a. Intelligence, including medical.

   b. Detection (scene assessment).

   c. Diagnosis, consisting of:

      (1) Casualty (clinical) assessment.

      (2) Clinical investigations including diagnostic imaging and laboratory services.

   d. Forensics.

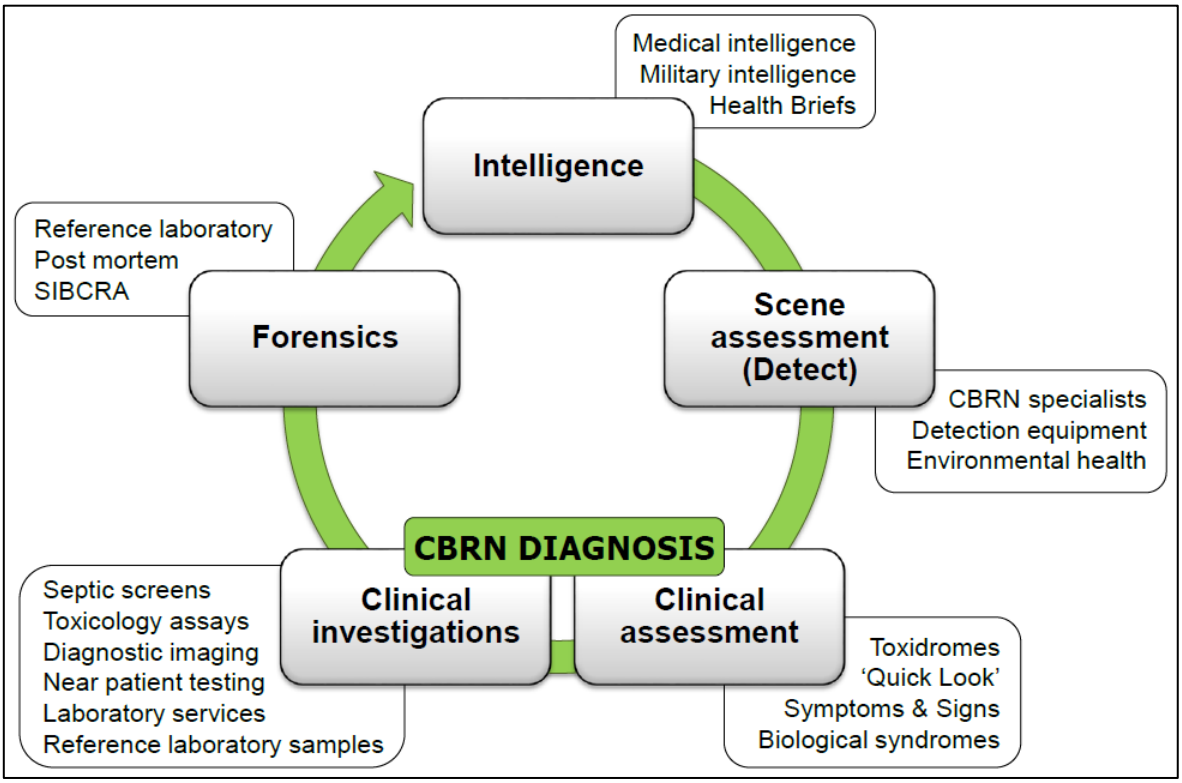

Figure 5-1: Cycle of Recognition. (SIBCRA: Sampling and Identification of Biological, Chemical and Radiological Agents)

2. The initial CBRN defence posture of an operation or mission is based upon the intelligence and subsequent risk assessment of the CBRN threat. This in turn contributes to the decision to deploy a detection capability and what agents are specifically being looked for. In addition, intelligence will inform the medical planners who will raise awareness of certain CBRN agents as well as medical intelligence that will also inform medical personnel of spectrum of hazards including environmental and endemic disease. If subsequent agents or an event are recognised either through detection, the medical chain by diagnosis or forensic analysis, the intelligence assessment will be updated and the risk assessment revised.

3. As part of a CBRN incident response, detection and clinical assessment will contribute to the 'assessment' component[1] of the CBRN incident response paradigm, as scene assessment and casualty assessment respectively. This is discussed further in Part 2 of this publication.

5.3. CBRN DETECTION

1. CBRN detection is one of the five elements of CBRN Protection contributing to CBRN Inform. It includes detection, identification and monitoring (DIM).

2. Detection may be carried out by any force trained and equipped with CBRN detection equipment, CBRN specialist units or other technical units such as environmental health teams depending on whether the principle hazard being assessed is CBRN or environmental. In some cases, the type of agent may not be known and a multidiscipline approach to detection must

[1] The components are safety, cordons, command, communication, assessment, triage, treatment, transport, exploitation and recovery.

underpin CBRN recognition. Endemic disease is more likely to be recognised (diagnosed) through the medical chain and for outbreaks among livestock veterinary personnel have a key role.

3. The methods of detection and identification are subject to sensitivity and specificity constraints that may generate 'false negative' and 'false positive' information respectively. The threat state affects the pre-test probability of any detection method. As such, response to a positive detection result will depend on the CBRN threat state as well as other information such as situational awareness and medical reporting.

5.3.1. DISEASE SURVEILLANCE

Disease surveillance is the observation of patterns of disease in plants, animals and humans. This is part of the medical contribution to a detection capability provided by environmental health, public health, veterinarians and clinicians. Human health (health surveillance) is monitored in the deployed population at risk (PAR) based on the reporting of specific conditions or syndromes (syndromic surveillance). The 'EpiNATO system' is a NATO sponsored monitoring system using multiple categories that include those that may be relevant to CBRN incidents. Further details are found in AMedP-5.1: *Deployed Health Surveillance*.

5.4. CBRN DIAGNOSIS

1. Diagnosis contributes to the Detect element of CBRN defence and is delivered by medical (or other medically trained) personnel. Diagnosis consists of two elements that are part of the cycle of CBRN recognition:

a. Clinical assessment of a CBRN casualty.

b. Clinical investigations.

2. Diagnosis may be concurrent with Detect where that capability is deployed i.e. nerve agent symptoms (described by casualty) and signs (seen by clinician) supported by chemical agent detection.

5.4.1. CLINICAL (CASUALTY) ASSESSMENT

1. This element of CBRN Diagnosis is delivered by a clinician such as a doctor, nurse or medic, who has direct access to the casualty. Clinical assessment includes history and examination. Symptoms (i.e. described by the casualty) might suggest a possible CBRN exposure or direct questioning may be required. Signs of potential CBRN exposure (i.e. seen by the clinician) may suggest the agent used and possibly at exposures levels less than detection (for example, miosis related to nerve agents and some biological agents). It may be necessary to ask direct questions related to a CBRN exposure by the clinician, such as time of onset and possible effects not immediately attributable to the illness, based upon intelligence or detection warnings that are earlier parts of the cycle of recognition.

2. Clinical assessment may be delivered from point of exposure through to rehabilitation for diagnosis, monitoring treatment efficacy and long-term effects. At point of exposure, clinical assessment will support first-aid management although provided by non-medical personnel and very important for CBRN agents with a short latency (chemical agents) and trauma.

3. A focused clinical assessment described in Table 4-1 is part of recognition, first aid and EMT in response to a CBRN incident. Certain chemical agents and some MedCM have a

characteristic pattern of symptoms and signs (syndrome) and this is sometimes referred to as a 'toxidrome' with a relatively high level of sensitivity and specificity; an extended Quick Look is provided in Part 3 of the publication. Clinical assessment of a possible biological casualty is also fundamental to the 'syndromic approach' to biological agent exposure and diagnosis (see Chapter 27).

5.4.2. CLINICAL INVESTIGATIONS

1. Clinical investigations are the continuing evaluation of a symptomatic casualty after the initial or repeated clinical assessment and include biochemical, haematological, radiographic, microbiological, immunological, chromosomal and toxicological assays. Clinical investigations are especially important for agents with a longer latency, where causation may not be suspected, or agents with non-specific signs and symptoms (biological and radiological). As well as recognition, these investigations may also be used for triage classification and monitor treatment efficacy. The investigations may be ordered by the clinician based upon clinical suspicion (possibly based upon medical intelligence) or as part of an investigation protocol based upon general intelligence or epidemiological guidance.

2. Investigations are likely to require medical personnel to take clinical samples such as blood and process them. However, some samples such as urine may not need specific medical training. It is likely that more sophisticated sample handling and testing would require laboratory facilities or a medical treatment facility (Role 2 and above). Some samples may require specific laboratory facilities that are only available or appropriate at a recognised reference laboratory either due to limitations or confidence in identification, biosafety, security, legal and/or political issues.

5.4.3. LEVELS OF CERTAINTY OF DIAGNOSIS

1. Clinical investigation methods also contribute to formal agent identification (confirmation), forensics, medical screening and health surveillance. The levels of certainty for diagnosis are:

a. *Suspected*. A case in which a potentially exposed person is being evaluated by medical personnel for CBRN medical effects, but no specific credible threat exists or detection warning has been made.

b. *Probable*. A clinically compatible case (including biological syndromes and toxidromes) in which a high index of suspicion (i.e. a credible threat or detection warning) exists for exposure to a particular CBRN agent, or a case with an epidemiologic link to a laboratory-confirmed case. (The latter may be based upon a case definition derived from previous cases).

c. *Confirmed*. A clinically compatible case with laboratory confirmation by using clinical, veterinary or environmental samples. In some instances, the case can be confirmed if laboratory testing was not performed because either a predominant amount of clinical and non-specific laboratory evidence of a particular CBRN agent was present or a 100% certainty of the aetiology of the agent is known.

Note: Confirmation of some selective agents will require reporting to national, regional and international governing bodies e.g. World Health Organization (WHO).

2. Specific information on clinical investigations is provided in each chapter for each agent.

5.5. FORENSICS

1. CBRN forensic recognition (or exploitation) uses scientific methods and techniques as part of the investigation of a CBRN event, to determine causation and possible criminal activity. CBRN forensics draws upon a variety of scientific principles, including biology, physics, chemistry and medicine. The level of confidence required must be irrefutable and therefore requires a documented chain of evidence confirming origin, evidence handling, validated analytical methods and that there has been no cross-contamination or tampering. As all CBRN incidents will be treated as a scene of crime, forensic principles and internationally standardised processes might also influence medical procedures and documentation.

2. Forensic assessment is unlikely to contribute directly to the initial CBRN event but will influence further incidents through intelligence and subsequent CBRN recognition. Human, veterinary and environmental sampling may provide samples for immediate or provisional detection and identification, as well as forensic evidence as long as there is no cross-contamination and the chain of evidence is maintained. All samples for clinical investigation may become a forensic sample. Medical personnel may also be required to provide a legal statement on the clinical assessment and investigation. All elements of the cycle of recognition could therefore be used for legal proceedings and international security measures.

Note: The term bioforensics should not be confused with the taking of clinical samples to investigate any CBRN incident. Bioforensics refers to the forensic investigation of a biological incident which may include human, plant and animal sampling and analysis.

5.5.1. SAMPLING IDENTIFICATION OF BIOLOGICAL, CHEMICAL AND RADIOLOGICAL AGENTS

Within NATO, a capability exists to collect and transport CBRN forensic evidence. This is called Sampling and Identification of Biological, Chemical and Radiological Agents (SIBCRA). This is provided by specialist teams from contributing nations. Further details are described in the AJP 3.8 CBRN defence series. Medical personnel may have a role but within the constraints of national legal frameworks and the protected status of non-combatants under the Geneva Convention.

5.6. POST-INCIDENT MEDICAL FOLLOW-UP

5.6.1. MEDICAL SCREENING

Medical screening is the active assessment at a specific time of a *well person* who may have been exposed to a suspected hazard or have an epidemiological link to a probable or confirmed case. Screening is vital so that MedCM that cannot be used empirically but have a post-exposure WOO, and early treatment can be started as soon as possible. For biological agents, this may be as simple as a rise in body temperature. The period of the medical screening required will be influenced by the latency or incubation period of the agent, exposed dose and the certainty of the time of exposure. For contagious biological agents, medical screening will also be part of the quarantine process. Not all screening requires medical personnel and it is not the sole responsibility of the medical chain of command.

5.6.2. HEALTH SURVEILLANCE

Post-exposure health surveillance is the long-term monitoring of the health (physical and mental) of an exposed population for predictable and unknown sequelae and may use tools such as a health registry and screening.

5.7. CBRN INCIDENT ASSESSMENT

Elements of the CBRN Cycle of Recognition contribute to the assessment part of the generic approach to a CBRN incident. Chapter 13 on CBRN Incident Assessment highlights the importance of concurrent scene and casualty assessment.

5.8. CBRN MEDICAL SENSE CAPABILITY

Figure 5-2 summarises the components of the medical contribution to the overall CBRN inform capability. Depending on the initial threat state, the actions in response to a warning or report of an individual case or outbreak may vary and include automatic or pre-determined protective measures or require further analysis to quantify the validity of the report and the plausibility of an attack. This response to CBRN threats and medical sense with protective measures is described further in AMedP-7.6.

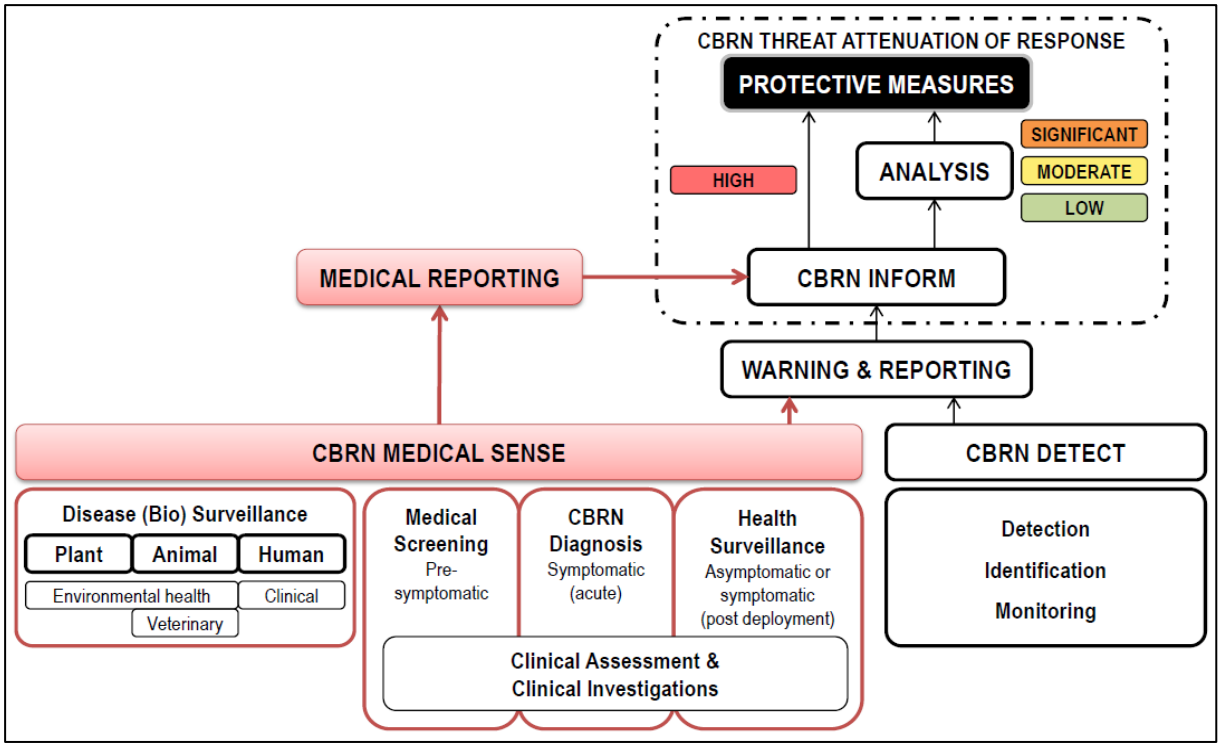

Figure 5-2: CBRN 'Medical Sense' Concept.

5.9. THE UNUSUAL PATIENT

During medical support operations, casualties may present with unusual illnesses to both hospital and primary care facilities. For these cases where the illness does not match the normal endemic pattern of illness or there is no obvious cause, an environmental or CBRN exposure should be suspected. Further guidance on the management and investigation of the unusual patient is covered in Annex 7A.

CHAPTER 6: CASUALTY HAZARD MANAGEMENT

6.1. INTRODUCTION

1. Casualty hazard management is the decision making process for the handling of casualties with a secondary exposure risk due to either contamination or a contagious illness (Figure 6.1). Contamination may be classified as:

a. External contamination.

b. Internal contamination.

c. Wound contamination.

2. Personnel decontamination is the responsibility of the operational unit and casualty hazard management is the responsibility of the medical chain of command, with support from other units. The elements of casualty hazard management are:

a. Containment.

b. Decontamination.

(1) External.

(2) Internal (Decorporation).

(3) Wound.

c. Isolation.

d. Quarantine.

e. Restriction of Movement (RoM).

(1) '*Medical RoM*' (isolation, quarantine).

(2) Operational RoM.

(3) Strategic RoM.

6.2. CONTAINMENT

1. Containment is the immediate on scene management of persons that may have been exposed to a CBRN agent in order to prevent further spread. It should not however prevent the clearance of persons away from a continuing hazard. For Improvised Explosive Devices (IEDs), the four C's approach (Confirm, Clear, Cordon, Control)[1] is used to describe the initial actions on discovering a device. For those that may have a CBRN component to the device, the approach is extended to include *Communicate* and *Contain* (the six Cs). This approach is compatible with the CBRN incident management paradigm described in Part 2 of this publication (see para 9.2. & Figure 9-1).

[1] STANAG 2294Ed1

2. Containment of personnel on scene may be an early form of RoM and is controlled by the Incident Commander under advisement from a CBRN advisor or reach back.

3. In some cases, the mission may need to carry on for operational reasons and containment may therefore be delayed. All mission personnel must take appropriate measures to limit any cross-contamination, spread or further exposures.

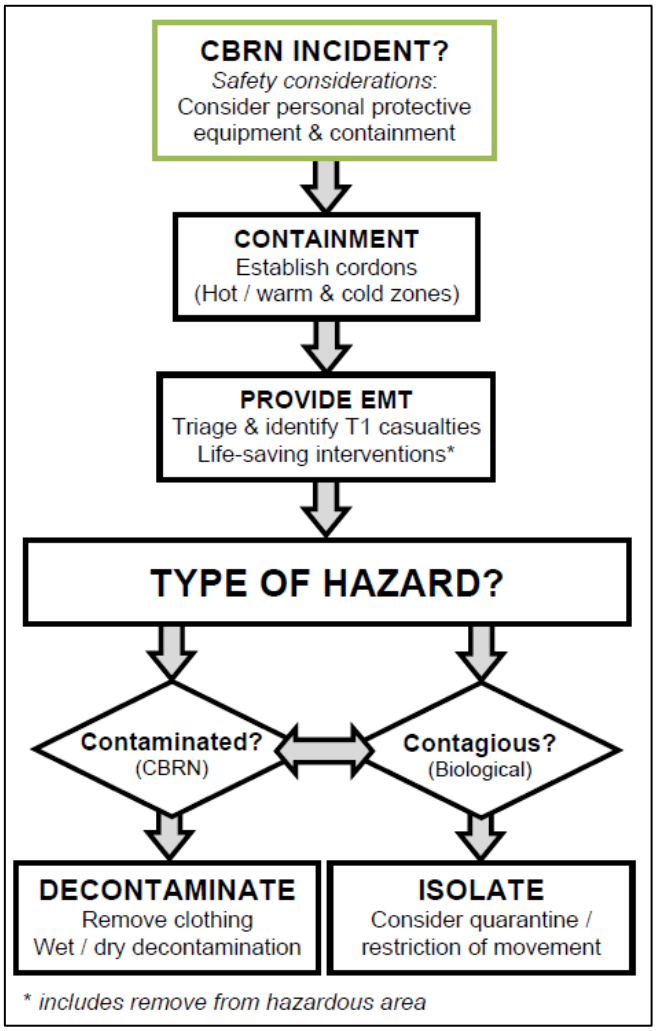

Figure 6-1: Casualty Hazard Management Algorithm.

6.3. EXTERNAL CONTAMINATION

1. External contamination is the coating of an external surface such as the skin, hair or clothing by a persistent agent. The agent may be liquid (including theoretically condensed vapour) or dry particulate. Management of external contamination (decontamination) includes the removal of clothing, gross physical removal of the contaminant and skin decontamination. Owing to the possibility of hair contamination, appropriate management should be considered including hair washing and potential shaving of the head.

2. Where a significant CBRN threat exists, pre-event CBRN defence against external contamination will include individual protection equipment maximising skin coverage. For non-CBRN defensive operations, the only external contamination protection may be conventional

clothing and post-exposure interventions will therefore rely on clothing removal and skin decontamination. The resources for decontamination should be prioritised for casualties, responders and then medical infrastructure. The latter is especially important for the decontamination of particulate contaminants such as contaminating CBRN particulates or robust hazards such as spores. This is important due to the risk to other casualties that may be vulnerable due to open wounds, respiratory sensitivity or other vulnerability.

3. Removal of chemical external contamination should be a priority as absorption through the skin may continue increasing systemic toxicity and localised skin damage. High dose beta and gamma radiological hazards should also be prioritised due to continuing local damage and possible whole body irradiation; however life-saving interventions should take greater priority. Biological external contamination is less of a priority for decontamination as the skin is relatively impermeable to biological agents. During decontamination, respiratory protection for responders and casualties should be risk assessed as volatile liquids will continue to evaporate and particulate contaminants could be re-suspended during the procedure.

4. Decontamination methods for chemical agents include:

a. *Physical removal*. This is the mechanical method of removing a persistent contaminant. The most common mechanical methods used individually or in combination are:

(1) Removal of clothing.

(2) Irrigation with copious amounts of water at a recommended temperature of 35°C.

(3) Dry adsorbents. Adsorbents physically draw in liquid contaminants and examples of adsorbents include fuller's earth and powdered resins. It should be noted that the agent is not destroyed and an off-gassing hazard from the adsorbent material and residual contamination remains after dry decontamination.

b. *Chemical destruction*. This method involves the deactivation of an agent by altering its structure. This can be achieved by chemical reactions such as:

(1) *Hydrolysis*. Water has both a mechanical and chemical action. The chemical action is slow but useful for deactivation of susceptible chemicals once in the wash off decontamination solution. This mechanism is most effective in an alkali pH due to the act of OH^- on phosphorous containing agents.

(2) *Oxidation*. This method includes the use of active chlorine solutions on some chemical agents including sulphur mustard and VX. Hypochlorite ($-OCl^-$) solutions vary in strength and pH and depend on the chlorine forming product used. The solutions should be fresh (daily) and alkaline. Active chlorine solutions also have a disinfecting action on live biological agents.

(3) *Active decontamination compounds*. This form of decontaminant uses agents that have a specific action against certain chemical agents.

5. Physical removal is also a significant method for the decontamination of biological and radiological contamination either using water with soap, and can be enhanced by the use of skin disinfectant (inactivation). Common biological skin disinfectants include chlorhexidine, iodine and 0.5% bleach.

6.3.1. LEVELS OF CASUALTY DECONTAMINATION

1. NATO publications refer to four levels of decontamination (immediate, operational, thorough and clearance). However, these levels cannot be applied directly to casualty decontamination and the following levels of casualty decontamination are used instead:

a. *Immediate*. This is the immediate application of adsorbent and/or removal of exposed clothing from an unprotected individual to prevent further absorption and reduce secondary contamination risk. This is considered a first aid measure.

b. *'Expose to treat' (clinical decontamination)*. This level of decontamination is applied during EMT to T1 casualties only. It is the removal of clothing and protective equipment in order to allow medical personnel to provide treatment to the upper end of the body. The term 'expose to treat' is a medical order to another medic or decontamination team member, potentially from another nation, to remove a respirator and expose the upper torso, and arm or lower leg. This allows the assessment of the airway, respiration and allows intravenous or intraosseous antidotes administration. This level of decontamination will precede the complete removal of clothing and full casualty decontamination. These may be delayed if there is a continuing risk of primary contamination. For other non-T1 casualties the respiratory protection would be the last item of clothing to be removed.

c. *Removal of clothing (disrobing)*. This is the removal of IPE or standard clothing as the first stage in full decontamination. This stage of decontamination is the most important as it may remove most of the contaminant. In some circumstances, such as exposure to a non-persistent agent or IPE use, this may be the only decontamination stage required followed by the option of a superficial wiping of the skin.

d. *Full decontamination*. This may be a single or two stage method depending on the agent and its physical properties. The requirement for full decontamination may be due to no IPE being worn followed by exposure to a persistent agent. Even if IPE is worn, any damage, such as following an explosion, may lead to external or wound contamination. The precise method whether dry or wet will vary between nations. The choice of dry or wet method following an incident may depend on the availability of water and infrastructure, the presence of particulate or solid debris as contamination, and contamination not amenable to chemical destruction or adsorption such as biological and radiological.

2. Responders should also limit the risk of environmental injury such as hypothermia or sunburn. Casualties during the decontamination process should be sheltered and then covered (re-robe) after decontamination either with blankets or replacement clothing.

3. The decontamination of civilian populations may also need to consider cultural needs such as language, religion and gender to minimise the distress and reluctance to decontaminate. Additional decontamination techniques or resources may be required when decontaminating vulnerable populations such as children, the elderly and casualties with existing disabilities including prostheses and poor mobility.

6.3.2. CASUALTY DECONTAMINATION RISK ASSESSMENT

1. The requirement and level of casualty decontamination depends on the hazard, the resources available as well as the medical state of the casualty. The presence of IPE while protecting the individual may cause difficulty in casualty assessment and in the worst case

cause life-threatening problems due to airway obstruction (especially if vomiting or airway trauma) and increased airway resistance. Factors that affect the decision, level and method of decontamination are:

a. IPE worn by the casualty.

b. Persistency and cross-contamination risk of the agent.

c. Weather conditions (ambient temperature, humidity).

d. Effects of the agent (lethal or non-lethal).

e. Risk to responders.

f. Operational environment and mission.

g. Decontamination facilities available, including water supply and control of run-off.

2. Following exposure to a CBRN agent, the possible outcomes of the risk assessment after assessing the agent, delivery method (if known) and scene are:

a. *No decontamination required*. This is most likely after exposure to non-persistent agents such as a gas that is lighter than air, or to an intact radiation source where there is no contamination hazard. Removal of clothing may still be a requirement to allow access to the casualty for treatment.

b. *Limited decontamination / removal of clothing*. This is likely if exposed to vapour as removal of clothing (disrobing) will remove the majority of the any trapped vapour thus removing any off-gassing hazard. Although there is little residual skin contamination, a light skin wipe may be considered to reassure the casualty.

Note: A person exposed to condensed vapour in an open area is highly unlikely to present an off-gassing hazard. Casualties being managed in a confined space must be disrobed and managed in a well-ventilated area.

c. *Full casualty decontamination*. This is for persons contaminated with persistent agents with symptoms or signs of intoxication, infection, irradiation or injuries. It consists of two decontamination methods depending on whether the casualty can walk and/or is compliant.

(1) *Walking (ambulatory) decontamination*. This method may be used for the walking and compliant casualties and is similar to personnel decontamination. Casualties with injuries or minor incapacitation may require assistance and all casualties should be escorted and observed.

(2) *Stretcher (non-ambulatory) decontamination*. This method is used for non-ambulatory casualties (T1 or T2) and may also be used for any other casualties, including incapacitated, as a safer and easier method of casualty handling. T1 casualties may receive concurrent Emergency Medical Treatment (EMT) while undergoing stretcher decontamination.

3. A summary of the various decontamination methods is in Figure 6-2 and may include the use of active decontamination compounds also either in isolation or as an additive to water

during wet decontamination. Following the use of active decontaminants or adsorbents, a brief rinse may be required to remove the decontamination materiel.

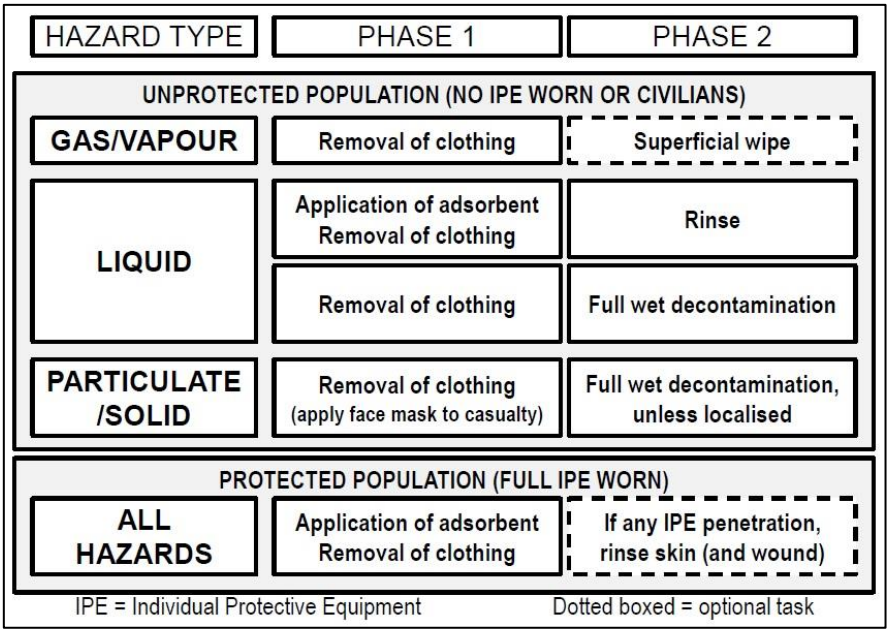

Figure 6-2: Example of Casualty Decontamination Methods.[2]

6.4. INTERNAL CONTAMINATION

1. Internal contamination occurs by inhalation, ingestion, transcutaneous (fat soluble liquids only for the latter) or wound. Relatively small quantities of agent are internalised by inoculation and are unlikely to represent a secondary hazard for responders. For chemical agents, internal contamination is not a significant issue as most of them will be reacting with biological molecules and water inside the body and will not be present in the original active form. Skin reservoir, described with agents such as sulphur mustard, may produce off-gassing even when no more agent is available to skin decontamination. Manage of these casualties is recommended in a well-ventilated environment. Some agents including radiological will require removal enhanced by medical intervention.

2. The removal of internal contamination is sometimes referred to as *decorporation*. Methods used include:

a. The exploitation of the agent's chemical properties and binding them using chelating agents. These form either less toxic complexes or enhance the agent's removal as a complex from the body. Chelation is also applicable to radionuclides based on their chemical properties.

b. Increasing elimination of the agent (reducing the biological half-life).

c. Increasing metabolism of the agent (reducing the biological half-life).

[2] This figure is for guidance and each nation will have its own methodology. Following exposure to gases, full decontamination is not required but disrobing should be performed. Active decontaminants may also be used instead of adsorbents with a Phase 2 rinse requirement dependent upon the manufacturers' recommendations.

d. Blocking the target site of the agent (i.e. stable iodine preventing absorption of radioactive iodine in the thyroid).

e. Physical removal of the agent from the respiratory or gastrointestinal tract.

3. Specific MedCM and other antidotes are described in the relevant chapter for each specific agent and in Chapter 35 for radiological agents.

6.5. WOUND CONTAMINATION AND MANAGEMENT

1. Wound contamination is the introduction of an agent into an area of traumatised tissue. A secondary hazard to responders including surgical teams may be present, however the greater the potency of the agent the less likely the casualty is to survive before surgery and therefore the risk tends to be self-limiting. Devitalised tissue may also act as a buffer to limit systemic absorption of an agent.

2. *Pre-hospital management of wounds*. The management of wounds and the use of tourniquets and dressings mean that the potential for external contamination exists and all dressings and tourniquets must be either replaced or covered in a clear protective dressing or wrap.[3] Wound irrigation fluid ranges from copious amounts of water or saline to weak hypochlorite (bleach; 0.3%) solution. The low concentration may only have limited or delayed effect on chemical agents and be disinfection only. Wound decontamination may occur at point of exposure (wounding), during full casualty decontamination or at the receiving MTF. However, where, when and the specific decontamination process, is the responsibility of individual nations.

Note: Other skin decontaminants such as adsorbent powders (e.g. fuller's earth) and some active decontaminant compounds are not recommended and may complicate wound healing.

3. *Secondary exposure risk*. There is a theoretical risk that contaminated wounds (munitions fragment or impregnated clothing) may pose a threat to a surgical (2.5-10%) team. For simple clean wounds such as an incision, this is very unlikely. All dressings may be contaminated and should be removed and disposed of in concentrated hypochlorite solution or a vapour-proof container. Standard surgical procedures including the use of aseptic techniques and surgical instruments, such as forceps, will minimise any risk. A single pair of latex surgical gloves may not provide adequate protection from some chemical agents where there is direct contact e.g. wound probing, and double nitrile gloves are the minimum recommendation.

4. *Radiological wounds*. Solid radiological shrapnel may present a potential but quantifiable hazard and can be managed by early removal or shielding until haemostasis or proximal vascular control is achieved. Significant exposures to gamma radiation can be quantified by dosimetry and the dose rate to the clinician assessed. Any exposure can be mitigated by time, distance and shielding supported by dose rate monitoring. Any radiological shrapnel should be removed as soon as possible and safely disposed of in a shielded container. Casualties exposed to a high enough dose rate that presents a significant hazard to the surgeon are likely to have been already exposed to a severe localised dose as well as potential lethal dose. Surgery should be limited to damage control surgery and where necessary the surgeon rotated

[3] ☑ ***Lesson Learnt***. Observations during the Exercise Clean Care series noted that decontamination teams were reluctant to change dressings. Medical or first aid trained personnel should be available to assist non-medical decontamination teams with this task.

to limit radiation exposure. An early wound reassessment ('relook') is suggested after local irradiation due to a high risk of tissue necrosis and wound failure.

5. *Surgical guidance*. Surgical management of any battle injury should include a debridement extensive enough to remove any residual contamination. Wide debridement is recommended for all contaminated wounds, whether CBRN or conventional. Irrigation with low concentration decontamination solution has been suggested but remains controversial; bicarbonate solution causing alkaline hydrolysis of some agents has been suggested. There is a potential vapour risk from impregnated clothing that has entered the wound. However for lethal agents such as the nerve agents, contamination levels enough to be a threat to the surgeon will already be fatal to the casualty. Sulphur mustard in a wound will bind rapidly to the surrounding tissue. Clothing impregnated with mustard may still off-gas when removed from the wound and should be transferred rapidly from the wound to a decontamination liquid such as hypochlorite solution. Tissue should also be placed in 2.5-5% hypochlorite solution and radiological shrapnel should be shielded, marked and removed to a safe distance in accordance with local ionising radiation regulations.

6.6. CASUALTY DECONTAMINATION FACILITIES

1. All MTFs should have a casualty decontamination capability and a capacity that is appropriate to the CBRN threat analysis, and reflect the increased or decreased capability in other areas of CBRN defence.

2. Casualty decontamination facilities should operate in such a way as to optimise interoperability with personnel decontamination, other nations' casualty and personnel decontamination facilities and MTFs. In order to achieve a common capability, a casualty decontamination facility should:

a. Be rapidly deployable depending on the CBRN threat analysis.

b. Provide Emergency Medical Treatment to T1 casualties.

c. Provide appropriate decontamination within the constraints of available resources, including water.

d. Manage any hazardous waste generated by casualty decontamination.[4]

e. Operate and mitigate the effects of the climates of the operational area.

f. Operate at night.

g. Prevent secondary injuries due to environmental exposure e.g. heat and cold.

3. Types of casualty decontamination facilities include:[5]

[4] See Chapter 16 – Exploitation and Recovery, and STANAG 2150: *Management of Clinical Waste*.

[5] ☑ ***Lesson Learnt***. Variation in the naming of casualty decontamination facilities exists. It is recommended for operational planning that there is consistency between contributing nations during the planning process. The responsibility and delivery of casualty decontamination may vary between nations but may include the CBRN Decontamination Platoon and an enhancement to a MTF such as R2E.

a. *Casualty Decontamination Area (CDA)*. This is a generic overarching term that describes a location in the warm zone of varying complexity for the decontamination of any casualties including CBRN and trauma and the provision of EMT.

b. *Casualty Decontamination Centre*. This is a standalone casualty decontamination facility with its own logistic support.

c. *Casualty Decontamination Unit (Cell)*. This is a casualty decontamination facility that operates within a larger personnel decontamination unit or a MTF and relies on logistic support from the larger unit. A unit may be forward deployed in order to enable casualty evacuation using ground and air assets (see Chapter 15 – Transport) but would have limited capacity.

d. *Decontamination Team*. This is a team trained in personnel and casualty decontamination that may be medical or non-medical. Some CBRN and trauma first-aid training should be provided, and the team should be interoperable with the EMT team for stretcher (non-ambulatory) casualty decontamination.

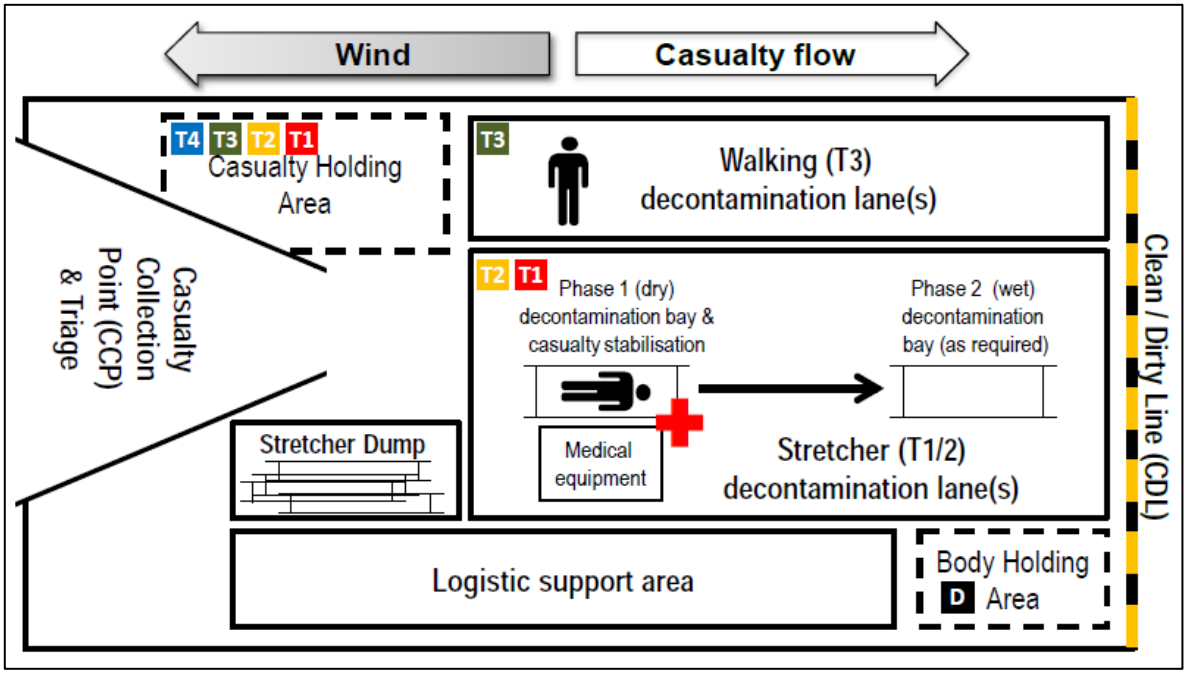

Figure 6-3: Basic Layout (1 walking and 1 stretcher) of a Casualty Decontamination Area.[6]

4. Elements of a CDA include:

a. Casualty Collection Point[7] & Triage.

b. Ambulatory (walking) lanes(s) (optional – varies between nations).

[6] Capability is described by the number of stretcher lanes and number of walking lanes i.e. 1 + 0 (light), 1 + 1 (basic) and 2 + 3 (standard).

[7] ☑ ***Lesson Learnt***. A Forward CCP is an initial *ad hoc* area where casualties will be taken after evacuation from the hot zone, most likely in the hot / warm zone interface where EMT can be started. This is based on previous incident responses, both military and civilian counter-terrorism.

c. Stretcher lanes(s) with provision for EMT.

d. Body handling area (temporary mortuary).

e. Casualty holding area providing first aid, as required.

f. Equipment decontamination either within logistic support area or stretcher dump.

g. Logistic support area, as required.

h. Miscellaneous, including scribes (clerks) and runners.

5. The foremost personnel element of the CDA is the identification of a Commander (Officer or NCO in charge), which may be secondary role. Where possible, personnel should be identifiable especially those in a command, safety or medical role. An example basic layout is shown in Figure 6-3 while a suggestion of roles and identification is described in Table 6-1.

Table 6-1 – Examples of CDA Roles and Identification Colours.
(Command – yellow, Medical – blue, Safety – red and Logistic support – white)

ROLE		RESPONSIBLE FOR
CDA Commander		Personnel safety & dynamic risk assessment Selecting appropriate IPE dress state or PPE Establishing and maintaining cordons Establishing work / rest regime and rehydration regime Selecting appropriate decontamination technique Maintaining casualty flow including casualty reports and MEDEVAC requests Organising resupply
Triage		Triage of casualties for prioritising treatment & decontamination If delay in casualty flow and no more triage required, providing first aid in casualty holding area
Sentries	**Entry**	Sanitising casualty for weapons or ordnance Checking for contamination Reporting contaminant type to CDA commander
	CDL	Controlling CDL Security within CDA Implementing new CDL upon breach
Medical Team Leader (MO, NO or Senior Medic)		Performing CBRN emergency medical treatment Performing conventional treatment Confirming death
Medic		Assisting EMT Team Leader Treating and escorting T1 casualties
Decontamination operative	**Dry stretcher decontamination**	Transporting casualties within CDA to wet stage Removing contamination Transferring casualty over CDL, if required Preparing decontamination solutions
	Wet stretcher decontamination	Transporting casualties from wet to CDL Removing residual contamination and decontaminant Decontaminating equipment, as required Transferring casualty over CDL
	Walking decontamination	Escort casualties along walking decontamination lane Monitor and support T3 decontamination, incl. wet if wound Prepare decontamination solutions
Logistic support		Storage and safety of weapons Removing and bagging of equipment Issuing of equipment Preparing decontamination solutions Arranging decontamination/disposal of equipment
Scribe / Communications (optional)		Time keeping Completing medical documentation for handover at CDL Casualty reports and MEDEVAC requests (for CDA Commander)

6.7. ISOLATION

1. Isolation is the separation of ill or contaminated persons in such a manner as to prevent the spread of infection or contamination [adapted from IHR, 2005]. In the context of CBRN casualty hazard management, this refers to casualties with an illness from a suspected transmissible biological agent. Early isolation of personnel during any CBRN incident is referred to as containment.

2. The principles of isolation include protective measures such as physical protection and hazard management. These overlap with some CBRN defence capabilities as shown in Figure 1-1. Collective protective measures including negative-pressure isolation differ from those that protect from an external CBRN threat (positive-pressure). Barrier nursing methods will also be used and are the same as those used for infection control and prevention (ICP).

3. The implementation of isolation is a clinical decision and remains a medical responsibility, although the operational commander may need to be informed as this is the first stage of a wider operational RoM that is followed by quarantine.

6.7.1. TYPES OF ISOLATION PRECAUTIONS

The requirement and types of isolation are determined by transmissibility, infectivity and consequence of the disease. They are detailed in Table 6.2 and are based on WHO guidance.

Table 6-2 – Isolation Requirement and Precautions.

Isolation requirement	Contagiousness of case	Route of transmission	Type of protective measures (also see Chapter 4 for infrastructure and PPE requirements)	Examples of diseases (deliberate and natural)
Standard precautions	**Moderate**	Direct or indirect contact with faeces, urine, blood, body fluids and contaminated particles	Hand-washing, safe disposal of contaminated articles	Most infectious diseases except those in the categories below. Also used for toxin only exposures
Enteric isolation (contact precautions)	**High**	Direct contact with patients and with faeces and oral secretions	Isolation room*, contact precautions	Cholera, *E.coli*, rotavirus, shigellosis, typhoid fever
Respiratory (droplet) isolation	**High**	Direct contact with patients or oral / respiratory secretions and droplets	Isolation room*, masks, contact precautions	Influenza
Airborne isolation	**High**	Direct contact with patients or oral / respiratory secretions and droplets	Negative-pressure isolation room*, high-specification masks, contact precautions	Smallpox
Strict isolation	**Very high (extremely low ID_{50})**	Direct contact with infected bloods, secretions, organs or semen	Isolation room*, clean / dirty entry & exit routes, strict precautions (see Table 9-1)	Contagious viral haemorrhagic fevers (e.g. Lassa, Ebola, Marburg)
	Disease associated with high mortality and/or no effective or limited treatment			

* Isolation room may be extended to be an isolation (cohort) ward for multiple patients with the same infectious disease.

6.7.2. DEPLOYED ISOLATION AREAS

The type of isolation areas within an MTF depends on the index of suspicion for a contagious disease, level of isolation required, type of PPE to be used and clinical interventions required. The types of isolation areas are:

a. *Infectious disease assessment unit* (IDAU). This is an assessment area with a separate entry to the MTF. It is usually co-located with the PHC facility or deployed Emergency Department. It is for the initial assessment and emergency medical treatment of patients that may require fluid resuscitation and early management of sepsis including investigation. The IDAU is required on all operations and is most likely to be used for gastrointestinal diseases with standard and contact (enteric) precautions. The use of additional respiratory PPE will also allow droplet precautions to be taken.

b. *Isolation room*. An isolation room is a single-occupancy patient-care room used to isolate persons with a suspected or confirmed contagious (transmissible) disease. The room should have an anteroom for donning and doffing PPE and its own ablutions. For strict isolation, required for highly infectious disease such as VHFs, a one way system for entry (donning) and exit (doffing) is recommended.

c. *Airborne isolation room*. A negative pressure patient-care room used to isolate persons with a suspected or confirmed airborne infectious disease. The room should have an anteroom to act as an airlock for donning and doffing PPE, and its own ablutions.

d. *Cohort isolation ward*. An isolation ward for multiple casualties with the same (usually confirmed) infectious disease. For strict isolation, required for highly infectious disease such as VHFs, a one way system for entry (donning) and exit (doffing) is recommended. Bed separation should also be maintained to reduce cross-infection with secondary and opportunistic infections.

e. *Infectious disease cohort treatment unit*. This is a disease-specific MTF to manage an outbreak of an infectious (and usually) contagious disease. The outbreak is usually on a scale that is beyond the capacity of a conventional field hospital or requires specialist isolation facilities or waste management. The requirements for an infectious disease cohort treatment unit include:

(1) Reception area(s) – patient, staff and/or visitors.

(2) Patient circuit, including reception and triage, suspect area, confirmed area and recovery area.

(3) Staff circuit, including PPE storage and issuing, PPE donning area, PPE doffing and PPE disposal.

(4) Diagnostic laboratory facility with direct access to clinical area and sample reception, if supporting other MTF.

(5) Clinical waste management.

(6) Medical equipment waste including hazardous waste such as batteries.

(7) Incinerator.

(8) Mortuary.

6.8. QUARANTINE

1. *Quarantine is only required for contagious diseases with a risk of person-to-person transmission*. Quarantine is the restriction of activities and/or separation from others of suspect persons who are not ill in such a manner as to prevent the possible spread of infection or contamination [adapted from IHR, 2005]. In the context of CBRN casualty hazard management, this refers to the separation and assessment over a period time (usually the latency period) of a well person who may have been exposed to a suspected hazard or have an epidemiological link to a probable or confirmed case.

2. Quarantine is a medical decision but with increasing Command involvement on operations and is closely linked to *medical screening*. Depending on the type of agent, severity, transmissibility and numbers exposed, the quarantined person may be under the care of medical personnel or the operational unit with medical support or augmentation. Any quarantine area must have medical cover appropriate to the severity of the disease ranging from remote advice to full resuscitation facilities.

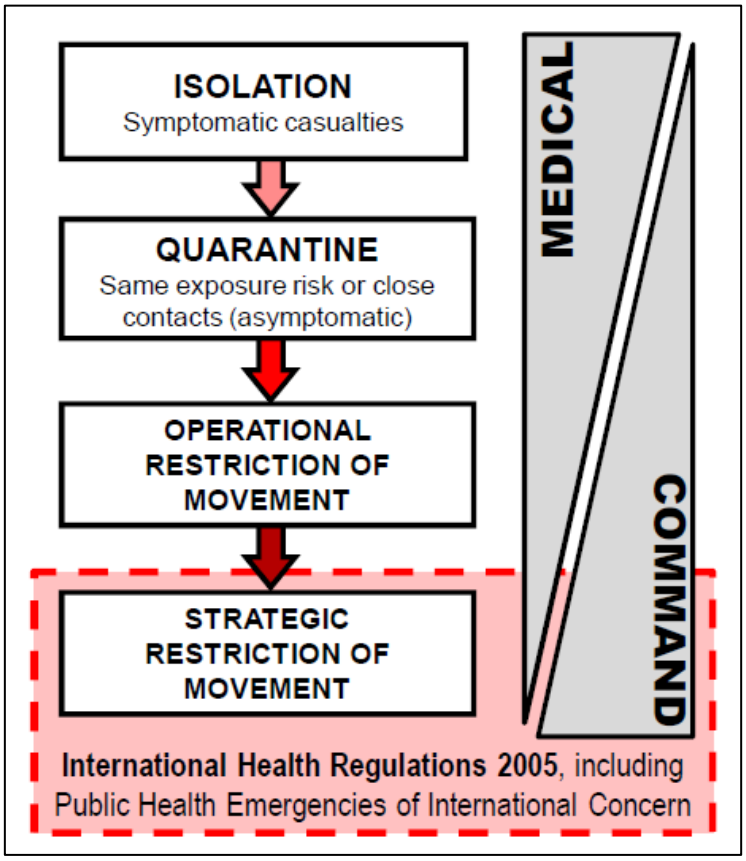

Figure 6-4: Summary of Restriction of Movement.

6.9. RESTRICTION OF MOVEMENT (RoM)[8]

RoM is a hazard management intervention for controlling the spread of a contagious disease by restricting contact with effected personnel and units. This separation may be geographical or operational and is implemented in order to maintain the mission. Figure 6-4 shows the escalation from medical restrictions (isolation and quarantine) through to formal RoM (operational and strategic RoM) as well as the medical and Command relationship.

6.9.1. OPERATIONAL ROM

A commander will need to consider formal RoM following credible evidence that there has been biological weapon use or indications from health surveillance or medical reporting that personnel have been exposed to a contagious disease with significant mortality or morbidity, or operational impact. This is a Command decision supported by advice from the Medical and Scientific Advisers as well as reach back. The decision will take into account situational awareness of the likely biological threats and hazards, severity of the illness and impact on the military mission as well as transmission into the local (host nation) and allied populations. Operational RoM can also be applied to non-biological incidents in order to control the spread of any hazard.

6.9.2. STRATEGIC ROM

For large outbreaks with a high morbidity or mortality, and person to person spread, international restrictions may be required to prevent the disease from being carried out of the operational theatre and across international borders. This is likely to involve civil health departments, regional health organisations and the World Health Organisation (WHO) as required under the International Health Regulations (IHR 2005) as a Public Health Emergency of International Concern (PHEIC). PHEIC, as defined in IHR 2005, means an extraordinary event which is determined:

a. to constitute a public health risk to other States through the international spread of disease, and

b. to potentially require a coordinated international response.

6.9.3. COMMAND GUIDANCE FOR ROM

Further information and Command guidance on the implementation and easing of RoM can be found in AMedP-7.6.

[8] RoM is the recommended abbreviation for Restriction of Movement as RM may be confused with Risk Management.

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CHAPTER 7: ADVANCED MEDICAL CARE

7.1. INTRODUCTION

1. There are a number of scenarios that would require a CBRN critical care response both for CBRN defensive operations, DAT and other CBRN incidents. These examples include:

a. Presentation of a single casualty with multi-organ failure (ricin, sepsis or radiation).

b. Presentation of multiple casualties with respiratory support requirements (pulmonary and nerve agents).

c. Presentation of a single casualty (botulin) or multiple casualties (chemical burns) with long term critical care or specialist needs.

d. Presentation of casualties with high bio-security needs including negative-pressure barrier nursing (contagious casualties).

2. CBRN incidents are sometimes referred to as 'special incidents' and are likely to be resource intense. However, the principles of incident and casualty management reflect conventional best practice but with additional safety issues and treatment regimens depending upon the specific CBRN agent (all-hazards approach). In many cases, treatment is supportive or follows guidelines that can be applied to scenarios irrespective of whether they are in a military or deliberate release context. An example of this is the application of the 'Surviving Sepsis' guidelines to a biological agent such as anthrax.[1]

3. Critical care patients are likely to have received a greater dose of a CBRN agent and present earlier than other casualties. Early diagnosis of these critical care patients may allow for more timely treatment of latent cases. This is also important for potential contagious or long latency illnesses where the critical care patient may be the index case or first confirmed diagnosis in the early stages of an outbreak.

7.2. AIRWAY CONSIDERATIONS

1. The management of the airway is critical following a chemical attack as well as trauma and is a priority. Airway compromise is likely for a number of reasons. Most of the lethal chemical agents may affect airway patency and protection due to:

a. Reduced level of consciousness (nerve agents, cyanide and some incapacitating agents)

b. Direct mucosal injury (sulphur mustard)

c. Increased secretions and vomiting (nerve agent)

2. The goals of initial airway management should therefore be:

a. Removal of any airway secretions or vomitus

b. Adequate oxygenation

[1] www.survivingsepsis.org

c. Adequate ventilation

3. Management of the airway depends on the environment (permissive or non-permissive), level of protective equipment worn and responder skills. While endotracheal intubation is the gold standard for a compromised airway, lessons identified from previous chemical incidents have demonstrated a shortfall in basic airway management including suction and simple airway manoeuvres during the pre-hospital response. In order to establish a definitive airway, anaesthetic drugs are likely to be required. When dealing with chemical casualties, medical staff should be aware that there are potential drug interactions. These are not only for CBRN casualties but also military personnel taking the nerve agent pre-treatment, pyridostigmine. If resources allow, casualties in respiratory arrest should be considered for resuscitation and would tolerate intubation without drugs. The benefits of a secured airway are:

a. Airway patency and protection;

b. Adequate suctioning;

c. High ventilation pressures and feedback on the adequacy of atropinisation for nerve agent bronchospasm; and

d. Capnography.

4. Some response organisations and recent papers highlight the benefits of laryngeal mask airways (LMA) for hot/warm zone management. These airway adjuncts should be used with caution as an LMA is not a definitive airway. Cases with bronchospasm and airway secretions (nerve agent) may not be able to be ventilated with high enough positive pressures or cases with pulmonary oedema (chlorine) be delivered positive end-expiratory pressure (PEEP). The LMA may also increase the risk and mask any aspiration of gastric contents as it sits over the larynx and upper oesophagus while limiting effective airway suction. The device however has a role as a rescue airway especially if there is a single clinical provider as it is easier to insert in PPE, relatively well tolerated by an unconscious patient without drugs and can be easily removed and reinserted.

7.3. BREATHING (RESPIRATORY) CONSIDERATIONS

1. Exposure to CBRN agents has specific effects on the respiratory system. These effects can be categorised based upon the mechanism of action:

a. Direct

b. Indirect

c. Non-specific

2. Direct respiratory effects are localised and include bronchospasm, acute lung injury (ALI) and infection; examples being nerve agent, chlorine and pneumonic plague respectively. The level of the respiratory tree affected by the agent depends on its nature, particle size and water solubility. The respiratory tract can be divided into central and peripheral. The central part of the respiratory tree includes the upper airway and bronchial tree. Central respiratory dysfunction tends to lead to a ventilatory (type II respiratory) failure and CO_2 retention. The peripheral part consists of the lung parenchymal including the alveoli where gas exchange occurs. Damage to this part of the respiratory tract will lead to Type I respiratory failure leading to oxygen desaturation.

3. Chlorine is water soluble and has significant effects in the upper airway as well as lung, while phosgene is less water soluble and has little effect on the upper airway but severe effects in the lower respiratory tract. Indirect effects on the respiratory system and therefore gaseous exchange can be due to systemic toxicity such as the paralysis of the diaphragm (nerve agent/botulin exposure) or pulmonary fibrosis by some TIC. The final non-specific effects on the respiratory system do not necessarily have a dose-response relationship. The effects may be due to systemic inflammatory response syndrome (SIRS) or sepsis causing adult respiratory distress syndrome (ARDS). Some agents may have a combined effect on the respiratory system; sulphur mustard and inhaled ricin both exert their effect by local damage and a systemic inflammatory response.

4. The effects of some pulmonary agents are still not widely understood. Chlorine appears to have a local effect that is dependent upon both concentration and duration of exposure. Local effects include direct damage to the intercellular junction and contraction of the intracellular cytoskeleton. Either mechanism will cause greater permeability of the alveolar-endothelial membrane leading to non-cardiogenic pulmonary oedema, alveolar collapse and a type I respiratory failure pattern with desaturation. Specific respiratory management includes reducing the closing capacity and recruiting more alveoli either with continuous airway positive pressure (CPAP) or positive pressure ventilation with PEEP. If there is also circulatory compromise, both should be used with caution due to the potential of reducing cardiac preload. Patients should therefore be optimally filled to maintain venous return. The mechanism of phosgene toxicity appears to be inflammatory and free radical mediated and may account for the relatively longer latency period (up to 24 hours). The degree of respiratory compromise is also greater reflected by a lower lethal concentration time when compared to chlorine. Phosgene accounted for the majority of the chemical fatalities during WWI, while mustard accounted for the greater number of casualties. Chronic effects of chemical exposure include reactive airways dysfunction syndrome (RADS) and fibrosis.

5. The role of inflammatory mediators following pulmonary agent exposure raises the question of anti-inflammatory prophylaxis such as inhaled steroids. The role of systemic corticosteroids is more controversial due to potential immunosuppression especially following chemical agents that may also cause bone marrow suppression such as sulphur mustard. The role and benefits of acetylcysteine is less clear and further research is required.

6. Ventilation strategies for CBRN exposures depend on the respiratory manifestations and the type of respiratory failure (type I, type II or mixed). Surrogate ventilation strategies based on those used for more common disease processes could be used as many of the CBRN agent manifestations mimic these conditions.

a. *Asthma*. The initial ventilation requirements for nerve agent are similar to an asthmatic with features of bronchospasm and high airway pressures. Capnography will assist in the titration of atropine as will the reduction in peak airway pressures. Ventilation may require lower respiratory rate to prevent air trapping and auto-PEEP. The mainstay for nerve agent treatment however is aggressive antidote management with a standard dose of oxime and rapid atropinisation titrated to the end-points of reversal of any bradycardia, drying of secretions and breaking of the bronchospasm.

b. *Low tidal volume (permissive hypercapnia)*. This ventilation strategy is recommended for ARDS. ARDS is likely to be a complication of any toxic insult that may lead to SIRS, this includes sulphur mustard and some of the toxins (ricin), as well as sepsis. The strategy uses low tidal volumes at the expense of retaining off carbon dioxide in order to reduce barotrauma and alveolar damage.

c. *Positive end-expiratory pressures*. For cases of type I respiratory failure, increasing PEEP while reducing inspired oxygen may optimise gaseous exchange while reducing further lung damage from oxygen toxicity. The role of CPAP in a mass casualty event remains unclear but may buy time and assist triage during the initial surge. Effectiveness may also depend on the agent used and pre-existing respiratory and cardiac disease.

7. Some toxins such as botulin and saxitoxin cause an uncomplicated type II respiratory failure (similar to neurotoxic snake venoms) and require standard adult ventilation strategies unless a complication occurs. Any ventilated patient should be monitored for conventional complications of mechanical ventilation including pneumothorax and secondary infections.

7.4. CARDIOVASCULAR CONSIDERATIONS

Most CBRN agents do not cause a direct or immediate effect on the cardiovascular system. Nerve agent may cause a bradycardia that is responsive to atropinisation as well as other dysrhythmias. Concurrent trauma may be present and normal guidelines on fluid resuscitation apply and take precedence after the airway and breathing have been secured. Biological agents may cause septic shock and this is discussed later. Patients requiring CPAP or PEEP should be adequately filled. There is no specific guidance on the choice of resuscitation fluid and crystalloid would seem to be appropriate. Fluid resuscitation for chemical burns may not need to be as aggressive as for thermal burns as the presentation of blisters is slower and therefore timed fluid replacement regimes such as the Parklands formula are not validated. Strict fluid balance and the monitoring of urine output is recommended. Some antidotes rely on renal function for the elimination of some agents as well as their antidote complexes (thiocyanate, cyanocobalt and radioisotope chelation). Urine output (0.5-1ml/kg/h) is therefore a useful guide to circulatory status.

7.5. DRUG INTERACTIONS WITH CHEMICAL AGENTS

Although the evidence base for chemical agent and pharmaceutical drug interactions is limited, there are some important interactions that need to be considered. Most of the data comes from animal models. Clinicians should also be aware of interactions between some MedCM and other pharmaceuticals i.e. pyridostigmine and some anaesthetic agents. Further details are found in Chapter 18 and agent-specific chapters in Part 3.

7.6. BIOLOGICAL AGENTS AND SURVIVING SEPSIS

1. Biological agents used maliciously may be associated with high mortality and potential person-to-person spread. However, the management of resulting sepsis should still follow the surviving sepsis guidelines. Although these guidelines apply specifically to live agents, the early management of a systemic inflammatory response associated with a toxin such as ricin would be beneficial. In many cases, the causative agent may not be known and the source identification guidance within the guidelines is all the more important for deliberate release contributing not only to the clinical investigation but also to the forensic investigation. Early treatment of sepsis and specialist support is vital in order to prevent patient deterioration and the development of multi-organ failure (MOF). The presence of at least three-system failure is associated with at least 50% mortality in the intensive care population.

2. Further details for the management of the septic casualty are covered in Part 4 of this publication.

7.7. CLINICAL INVESTIGATIONS

1. Assessment of a casualty will start with clinical examination following a history either provided by the casualty or pre-hospital team. Initial investigation will include normal biochemistry and haematology as for trauma. Early signs of chemical agent exposure may be an abnormal lactate level; this would be especially high for cyanide. Chest radiographs may be useful for respiratory syndromes (chlorine, pneumonia and pulmonary anthrax), however there may be a lag period between clinical and radiograph signs. Clinicians should be cautious at excluding one type of CBRN agent too early and previous case reports have demonstrated that there can be overlaps in syndromes during the early stages of illness. Examples include the presentation of paralysis (neurotoxins/Guillain-Barre syndrome), sepsis (live agent, biotoxin and heavy metal poisoning) and radiation both, systemic and local manifestations (thallium/gastroenteritis and dermatitis/vesicant/ cutaneous anthrax & leishmaniasis).

2. Point-of-care testing is still subject to research but may help guide antidote treatment while the mainstay of treatment is supportive management. Nerve agent intoxication is likely to be a clinical diagnosis and the severity assessed using clinical parameters. Diagnosis is likely to be supported by investigations such as red cell AChE activity levels and normal levels may be useful as a discharge criterion although lack of pin-point pupils is also a useful indicator.

3. Recommended investigations include baseline and specialised (focused) depending on the scenarios. Full details are given below.

4. For a CBRN incident, where there may be a violation of the Chemical or Biological Warfare Conventions or other criminal investigation, any sample may also be used for forensics and a chain of custody should be supported with at minimum appropriate documentation or witness statements.

7.8. COMBINED INJURIES – CRITICAL CARE CONSIDERATIONS

1. In cases of combined injury, the mortality and morbidity will be significantly increased. During a mass casualty incident, triage should be modified to reflect the effects of CBRN exposure (see Chapter 14). This is especially true for irradiation, where triage for surgery may be required. This will allow cases to be managed within a window of surgical opportunity before the risk of infection and clotting disorders increases.

2. *Wound contamination*. There is the potential for casualties with contaminated wounds to be admitted to a critical care unit to await surgery and debridement. Although there is a theoretical risk of secondary exposure, it is very low. Any lethal agent, such as VX, that is in significant quantity to cause harm to staff will have killed the casualty. Biological toxins do not present a significant risk if standard precautions are adhered to. Sulphur mustard is absorbed by tissue rapidly and would only be a hazard if a large wad of sulphur mustard saturated material remained in the wound. The blister fluid from sulphur mustard exposure does not pose a chemical hazard to staff although it remains a biohazard. Lewisite (another blister agent based on arsenic) blister fluid may be toxic, but standard precautions will also reduce the risk. Radiological shrapnel may be present but the dose rate from the wound is quantifiable and by reducing time, increasing distance and shielding where possible, the risk to staff can be mitigated and quantified.

3. *Combined chemical casualties*. Where there is concurrent trauma and chemical agent exposure clinicians should be aware of potential interaction and synergistic effects. Some of these are listed in Table 7-1.

Table 7-1: Examples of Chemical Agent and MedCM Effects on the Traumatised Casualty.

CHEMICAL AGENT	POTENTIAL COMPLICATIONS DURING COMBINED INJURY
Nerve agents (acute)	Excessive airway secretions and bronchospasm may further compromise airway obstruction and failure to ventilate lungs. Increased airway pressures due to bronchospasm may mimic tension pneumothorax after positive pressure ventilation. The pupillary effects of NA (miosis) and its treatment with atropine (mydriasis) will affect head injury assessment. NA effects on the autonomic nervous system (including bradycardia) may cause cardiovascular compromise in a compensating casualty.
Nerve agent (late)	Intermediate effects of NAs may lengthen time required in intensive care because of muscle weakness.
Sulphur mustard (late)	Acting as an alkylating agent, systemic toxicity may include bone marrow suppression and increase the likelihood of infection.
Pulmonary agents (choking agents)	Pulmonary agents, such as chlorine and phosgene, intoxication may present as pulmonary oedema. When encountered with chest trauma, type I respiratory failure will be exacerbated. Radiological investigations and cardiovascular monitoring may assist in differentiating non-cardiogenic pulmonary oedema from pulmonary contusion, adult respiratory distress syndrome (ARDS) and cardiogenic pulmonary oedema. Phosgene should be noted for its delayed onset and potential for misdiagnosis.
Cyanide	Severe cyanide casualties are unlikely to present beyond Role 1, as they are likely to self-select. Moderate casualties that survive beyond the pre-hospital environment may have a significant metabolic (lactic) acidosis. This may be misleading when assessing shock and oxygen delivery. Specific antidote treatment is unlikely to be required at this stage and may be contraindicated. Supportive management with high flow oxygen is recommended.
MEDCM	**POTENTIAL COMPLICATIONS WITH TRAUMA**
Atropine	Inadvertent use of atropine may lead to anticholinergic toxicity. This typically includes dry mucosa, mydriasis, tachycardia and confusion. The last three signs may confuse clinical assessment in the presence of trauma, especially when assessing shock and head injuries. Thermoregulation may also be disturbed because of reduced sweating, leading to hyperthermia especially in a warm environment.

4. *Management of non-thermal burns*. Most non-thermal burns should be treated using the same principles as thermal burns. However, there are a few caveats:

a. *Sulphur mustard burns*. Cooling the skin for several hours following initial exposure may reduce the initial damage. The requirement for fluid replacement should follow the Parkland formula, although the speed of the infusion will not be as rapid due to the latency of the blister formation. Re-epithelialisation of skin damaged by sulphur mustard is enhanced by treatments such as dermabrasion and laser ablation.

b. *Arsenical burns (Lewisite)*. Unlike sulphur mustard burns, vesicants containing arsenic have significant systemic toxicity with burns of < 5% BSA being potentially fatal. All casualties with rapid onset chemical burns and systemic features should be assessed for potential topical and systemic antidotes.

c. *Hydrofluoric Acid (HF) burns*. HF burns also have systemic toxicity due to hypocalcaemia. Treatment includes topical and intravenous calcium solutions.

d. *Phosphorous burns*. These burns are a combination of chemical and thermal. Any residual agent may continue to self-ignite and should be managed with wet dressings, irrigation and debridement as required followed by conventional burns management.

e. *Radiation burns including beta irradiation*. These will be discussed in Part 5.

5. *Trauma and nuclear detonation*. Following the detonation of a nuclear device, the following “conventional” traumatic injuries will be seen:

a. *Flash* – causing afterimages, retinal damage and blindness.

b. *Blast injuries* – caused by the initial positive and negative overpressures, and blast winds. In addition, further injuries will occur due to flying debris and crush injuries due to falling masonry. Treatment will follow conventional guidelines.

c. *Thermal injuries* – these are due to the initial intense flash and fireball with further injuries secondary to any fires ignited. These thermal injuries are different to burns caused by ionising radiation.

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ANNEX 7A – MANAGEMENT OF THE UNUSUAL PATIENT

1. The presentation of a casualty or multiple casualties with unexplained symptoms and signs, especially in the context of high morbidity or mortality, should be investigated in safe and standardised way. In all cases, standard precautions should be taken and where a contamination or contagious hazard is suspected, casualty hazard management should be carried out. Most of the assessment of an unusual casualty will be made at a Role 3 MTF although a strategic medical evacuation to a specialist unit may be required.

2. For multiple casualties over a period of time (emerging scenario), epidemiological study is recommended. This is initially supported by *Operational Epidemiology* (see Chapter 17). Consideration should also be given to requesting a Rapid Deployable Outbreak Investigation Team (RDOIT) or Medical Radiological Incident Investigation Team (MRIIIT). Guidance on both specialist teams is found in AMedP-7.7 and AMedP-7.4 respectively, and.

3. *Baseline clinical investigations*. All casualties should have baseline investigations at Role 2 Enhanced or Role 3. These will provide information to assess the critical care needs of the casualty as well as suggest possible cause. Recommended baseline investigations are listed in Table 7A-1 and should then be repeated at six hours so that a trend and serial assessment can be made. All samples should be appropriately marked if a significant hazard is suspected. Certain results from the baseline might suggest a possible cause for the disease and these are listed in Table 7A-2. However, diagnosis should not be based on a single non-specific test and correlation with clinical assessment, intelligence and other information should be carried out.

Table 7A-1: Recommended Baseline Samples for Clinical Investigations.

BASELINE CLINICAL INVESTIGATIONS (Repeated at 6 hours)
Full (complete) blood count and differential white cell count
Arterial or venous blood gas measurement including lactate Blood glucose level Renal function Liver function (includes clotting) Bone profile (includes calcium) Inflammatory markers (vary with nations but include C-reactive Protein)
Clotting screen (includes International Normalised Ratio (INR))
Chest radiography
Consider brain imaging, if reduced level of consciousness

4. *Specialised (hazard specific) clinical investigations*. Table 7A-3 provides a list of recommended samples to be taken in the event of a suspect illness or unusual illness. Where a type of agent is suspected, a focused series of clinical investigations can be taken. For a casualty with no clues to the type of agent, all of these investigations should be considered although there will be some overlap. The following should be considered with regard to sample handling:

a. Samples taken should be clearly documented in the casualty documentation.

b. A chain of evidence for samples should be maintained, as appropriate, so that they remain suitable for forensic examination and contribute to any subsequent security or legal investigation.

c. Hazard markings should also be used to warn the receiving laboratory.

d. Some of the investigations will be beyond the capability of the deployed medical treatment facility and require reach back and transportation to a specialist or reference laboratory.

5. Any sample should appropriately be labelled and packaged to meet international regulations such as UN3373 for Category B samples (including patient specimens) for Class 6.2 Infectious Substances (International Air Transport Association).

Table 7A-2: Baseline Clinical Investigations and Possible Causes.

BASELINE CLINICAL INVESTIGATION FINDING		POSSIBLE CAUSE
Full (Complete) Blood Count	Haemolytic anaemia	Heavy metals such as arsenic
	Low white cell and platelets	Significant radiation exposure
	Low platelets (and pyrexia)	Malaria (included for differential diagnosis)
	Low platelets (and coagulopathy)	Disseminated Intravascular Coagulopathy (DIC)
Chest radiograph	Unilateral pulmonary infiltrates	Pneumonia
	Bilateral pulmonary infiltrates	Pulmonary oedema due to pulmonary agents Viral pneumonitis Inhaled toxin (such as ricin, SEB)
	Widened mediastinum	Inhalational anthrax
Brain imaging	Localised lesion (and pyrexia)	Encephalitis Cerebral abscess

Table 7A-3: Agent Type Clinical Investigations.

SPECIALIZED CLINICAL INVESTIGATIONS (CHEMICAL)
10ml blood in plastic (lithium anticoagulant) heparin tube
5ml blood in glass (lithium anticoagulant) heparin tube (plastic to be used if glass not available)
10ml blood in plastic (EDTA anticoagulant) tube
30ml urine without preservative
SPECIALIZED CLINICAL INVESTIGATIONS (BIOLOGICAL)
Blood cultures for aerobic and anaerobic organisms
Serum sample for serology and toxin assays (repeated serology (convalescent))
Whole blood (EDTA anticoagulant) for molecular-biological investigations
Urine (microscopy, culture and sensitivities, and storage)
Source sampling including respiratory tract, skin, wound, nose and throat swabs, pus or vesicular fluid, faeces, cerebrospinal and pulmonary effusion fluid, and tissue biopsy material.
SPECIALIZED CLINICAL INVESTIGATIONS (RADIOLOGICAL)
Serial full (complete) blood counts
Serum amylase
Serum citrulline (where available)
Nasal swabs (within first hour following potential internal contamination)
Urine
Faeces (if gastrointestinal route)
Whole blood (storage for HLA cross-matching) recommended within 12 hour
Whole blood (for cytogenetics/dicentric assay) recommended at 24 hours

EDTA – Ethylenediaminetetraacetic acid
Specific assays will be discussed in relevant chapters

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ANNEX 7B – MANAGEMENT OF PAEDIATRIC CBRN PATIENTS

1. During NATO operations and in accordance with the Geneva Convention, medical personnel may be expected to treat paediatric casualties, including CBRN casualties. Historically, children have become casualties either accidentally or by deliberate targeting by both conventional and CBRN weapons. Medical personnel involved in NATO operations, where there is a significant CBRN threat and a requirement to treat the civilian population, should be prepared to manage paediatric CBRN casualties.

2. Children differ from adults in a number of ways which make them particularly vulnerable to attack, especially to an attack employing CBRN agents:

a. *Anatomical differences*:

(1) *Body weight*. Body weight among children varies greatly with age and nutritional status; this influences the calculation of drugs, including antidote doses based upon weight.

Note: Care should be taken using estimated weight calculations based on data from other regions, cultural and economic populations.

(2) *Body-surface area (BSA)*. Children possess a greater BSA to volume ratio than do adults. This provides relatively greater absorptive surface area for the action of dermally-active agents (such as vesicants), system absorption (such as percutaneous nerve agents (VX)) and also dictates that fluid losses will have a proportionally greater impact on children. Similarly, children are more susceptible to heat loss during decontamination.

(3) *Skin immaturity*. Children possess a thinner and less well keratinised epidermis, leading to an increased susceptibility to dermally-active agents and absorption.

(4) *Airway calibre*. Children possess a smaller airway calibre such that any small reduction in airway diameter, due to bronchospasm or secretions, has a disproportionately greater impact on airway resistance (in accordance with Poiseuille's Law).

(5) *Body water*. Children have a greater proportion of body water compared to adults leading to altered toxicokinetics.

b. *Physiological differences*:

(1) *Ventilation*. Children have greater minute ventilation than adults, leading to a proportionally increased vulnerability to the effects of gases, vapours and aerosolised agents.

(2) *Cardiac output*. The ability to increase cardiac output among children relies predominantly on heart rate and on a limited ability to increase stroke volume. Fluid loss and bradycardia are thus more dangerous in children and can rapidly lead to shock.

(3) *Functional residual capacity (FRC)*. A reduced pulmonary FRC predisposes children to early oxygen saturation.

(4) *Renal function*. Immature renal function in children results in compromised elimination of toxic substances.

(5) *Liver function*. Immature hepatic function in children results in reduced ability to detoxify toxic substances.

(6) *Glycogen stores*. A lower capacity for glycogen storage within the liver predisposes to early hypoglycaemia during physiological stress including fitting and sepsis.

(7) *Plasma proteins*. Fewer plasma proteins diminish the capacity for the binding of some toxic substances.

(8) *Blood brain barrier*. Greater blood brain barrier permeability heightens the potential for agents to gain access to the central nervous system.

(9) *Central nervous system*. The developing central nervous system of children may be more susceptible to certain exposures or to secondary brain injury resulting from hypoxia or hypovolemia.

c. *Developmental considerations*:

(1) *Height*. Children, being shorter, 'live closer to the ground' than do adults, increasing their exposure to radioactive fallout, heavier than air gases and re-aerosolised particles.

(2) *Mobility*. Children are often unable to flee the incident scene.

(3) *Compliance*. Children often lack the developmental ability to follow the instructions from responders and other on scene personnel.

(4) *Comprehension*. Children often cannot distinguish reality from fantasy and are thus prone to consider each repeated media portrayal of a disaster as a new event. Similarly, children are more likely to be frightened at the appearance of responders in PPE.

(5) *Psychological impact*. Children may be more vulnerable to PTSD than adults.

d. *Relative therapeutic contraindications*. Although the benefits of therapy would readily outweigh the risks in the event of a CBRN incident, providers of healthcare to children are nonetheless often unfamiliar with the use of certain therapeutic agents:

(1) Fluoroquinolones are often recommended as drugs of choice in the prevention and treatment of anthrax and plague, yet experience with their use in children is limited.

(2) Tetracyclines are also recommended for a number of biological agents including anthrax, plague, tularaemia, brucellosis and Q-fever; their use in children is often avoided.

e. *Immunisation considerations*. Immunisation against various biological agents is especially problematic in children:

(1) Anthrax vaccines in most nations are not licensed for persons less than 18 years of age.

(2) Smallpox historical vaccines may produce more frequent and serious complications in children compared to adults.

(3) Yellow Fever vaccine similarly produces serious complications more frequently in children.

f. *Chemical agent specific vulnerabilities*:

(1) *Nerve agents (NAs)*. Most NAs are absorbed through the skin. The thinner, less keratinised, more abundant (in terms of BSA to volume ration) skin of children makes them significantly more susceptible to the effects of such agents. Moreover, NAs cause bradycardia, bronchospasm and copious airway secretions, all of which are less well tolerated in children. Children may also have a lower seizure threshold and thus a greater risk of convulsions following NA exposure.

(2) *Pulmonary agents*. These substances are generally heavier than air and thus may disproportionately affect children who 'live closer to the ground'. The reduced FRC of children exacerbates the risk from these agents.

(3) *Vesicants*. The greater BSA to volume ratio of children dictates that systemic absorption of vesicants will be greater in children than in adults. Moreover, the fluid losses associated with these agents will likely have more immediate cardiovascular consequences.

g. *Biological agent specific vulnerabilities*. Sepsis is less well tolerated in children for multiple reasons as noted above. Moreover, an immature immune system hampers children's abilities to ward off infection with biological agents and potential vaccination efficacy. In addition, several potential biological agents have specific effects on children:

(1) *Venezuelan Equine Encephalitis (VEE)*. VEE is temporarily incapacitating in adults, but can be fatal in young children.

(2) *Smallpox*. Smallpox potentially presents an increase risk to children compared to older generations owing to residual immunity among older adults due to the discontinuing of childhood smallpox vaccination programme.

(3) *Melioidosis*. In some regions, children infected with melioidosis develop a peculiar parotiditis not seen in adults.

h. *Radiological specific vulnerabilities*. Children are more susceptible to the medical effects of ionising radiation, with a greater incidence of cancer including haematological malignancies, as well as thyroid cancers following exposure to radioiodine. Furthermore, radiation can be assumed to have disproportionate adverse effects on all growing tissues.

i. *Miscellaneous considerations*:

(1) Drugs and antidotes against CBRN agents are often unavailable in liquid paediatric preparations.

(2) Rescue equipment is often unavailable in paediatric sizes.

(3) Many hospital systems have limited numbers of paediatric acute and critical care beds.

(4) Many emergency responders, especially military, are unfamiliar or uncomfortable with the care of children.

(5) Paediatric disaster response doctrine is scarce.

3. *Paediatric CBRN medical support*. A number of conventional paediatric emergency care paradigms exist within NATO nations. Most of these systems cover the management of a seriously ill child and include trauma, toxic exposures and sepsis. Where there is significant CBRN involvement, supportive management and the use of MedCM would be appropriate for these challenging situations.

4. *MedCM*. Appropriate MedCM use in children should ideally be formulated as liquid oral preparations and prescribed based on weight (i.e. on a mg/kg basis). Recommended paediatric dosing regimens for the treatment of certain CBRN exposures are listed in the relevant chapters but are subject to national licensing and/or endorsement. Administration of MedCM in the context of a military operation for paediatric casualties will be for emergencies only and ideally should be reflected in any regulating requirements. The dosing of some MedCM may be difficult (auto-injectors) and instead of reducing the dose, the frequency of administration could be reduced.

5. *Psychological*. Management of the psychological consequences of trauma is a key component of paediatric disaster response and applicable to CBRN incidents. Children should be:[1]

a. Informed about an event as soon as possible.

b. Helped to understand the event by stating the basic facts in simple, direct and clear terms.

c. Sheltered from detailed information and graphic media images when developmentally unable to process such information properly.

d. Reassured they should feel safe in their schools, homes and communities.

e. Reassured regarding their lack of responsibility.

f. Observed for signs of guilt and anger.

g. Included in conversations involving the sharing of feelings of fear, sadness and empathy.

h. Offered the opportunity to discuss terrorist events (older children and adolescents), but not forced into such conversations.

i. Provided counselling in anticipation delayed and anniversary reactions (sadness or fear on the anniversary of a tragic event).

j. Provided concrete advice on how to make participation in commemorative events meaningful.

[1] Based on Schonfeld DJ. Supporting children after terrorism events: potential roles for paediatricians. Pediatr Ann. 2003;32(3):182-87.

AMedP-7.1 PART 2: THE MEDICAL MANAGMENT OF CBRN INCIDENTS

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CHAPTER 8: INTRODUCTION TO THE MEDICAL RESPONSE TO A CBRN INCIDENT

8.1. INTRODUCTION

1. A CBRN incident has specific security and intelligence significance. For medical personnel, incident management in the presence of a CBRN agent or TIM is simply another hazard that requires mitigation. During a combined (explosive and CBRN) incident, the presence of the CBRN agent may not be initially recognised. A conventional incident response, at the tactical level, includes safety considerations and a standardised 'METHANE report'[1] with a hazard assessment. CBRN and TIM incident management including the medical response should use the same paradigm but optimised to provide the best medical care for conventional and CBRN casualties.

2. This part of AMedP-7.1 focuses on the tactical element to the response part of the CBRN incident cycle and post-event interventions, both for CBRN defensive operations and in response to the asymmetric threat from insurgents and terrorists on any NATO operation and as part of the NATO Defence Against Terrorism (DAT) program. The CBRN response described is also compatible with most civil response organisations and where there are operational, regional and national incident command structures.

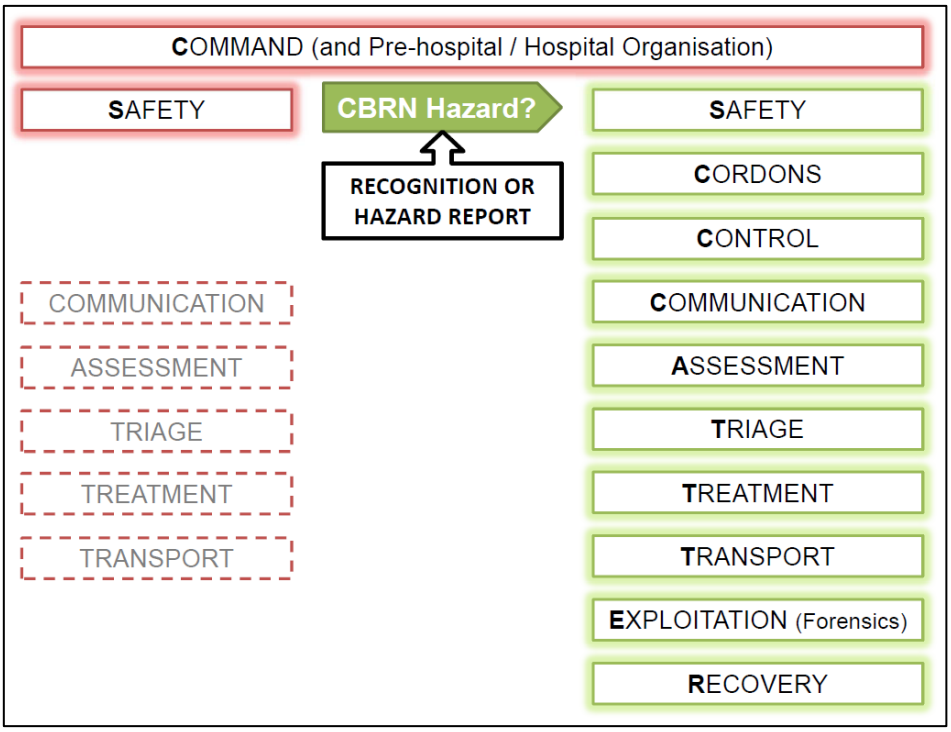

Figure 8-1: The Interface between Conventional & CBRN Medical Incident Management.

3. AMedP-1.10: *Medical Aspects in the Management of a Major Incident/Mass Casualty Incident* provides information on the standardised response to a conventional incident. Conventional major incident management uses Command, Safety, Communications, Assessment, Triage, Treatment and Transport (CSCATTT), to prioritise response and

[1] METHANE is a standardised incident report used by NATO and some member and other nations (see Section 12.2.).

resources. During a CBRN incident, this response is adapted with specific attention to safety, including physical protection and hazard management including establishing cordons and containing the scene. The METHANE report used for conventional events and any standard CBRN report will trigger the move from a conventional response to the modified CBRN response (see Figure 8-1).

8.2. TYPE OF CBRN MAJOR INCIDENTS

CBRN incidents, as for other medical major incidents, can be classified by the onset and timeframe of the incident and the adequacy of the response, using a combination of the following definitions:

a. *Sudden onset*. This type of incident is immediately apparent due to either the cause of the incident (explosive incident or release of an agent with immediate or acute onset effects), or where there is a detection capability. The incident response is likely to be concentric around the incident site or follow a plume if there is a downwind hazard.

b. *Slowly evolving*. This type of incident develops over a period of time so that the time of exposure or cause is not immediately apparent. Incident response is more likely to include operational epidemiology and specialist investigation teams.

c. *Simple*. The organisations and infrastructure involved in the incident response are not compromised by the event.

d. *Compound*. The organisation and infrastructure involved in the incident response is compromised by the event resulting in a degraded response or requiring extra resources to compensate. For a CBRN event, this might include critical infrastructure such as the HQ or MTF being in the downwind plume. The CBRN operational risk may require COLPRO for these mission-critical infrastructure sites.

e. *Mass Casualty* (MASCAL). A mass casualty incident is one in which there is an overwhelming number of casualties relative to the medical capabilities locally available. Depending on the available resources, the exact number is scalable. The senior medical commander (Comd Med) may authorise the use of the T4 (expectant) triage category or a ceiling of therapy. For CBRN incidents, this may be a relatively small number of critically ill casualties or a significant number of resource dependent casualties with specialist requirements including burns or radiation MedCM, critical care or renal support. During an outbreak, the number of casualties may be catastrophic or require finite resources or capabilities such as critical care, ventilation and renal replacement therapy. Further details on conventional MASCAL management may be found in STANAG 2879.

8.3. PRIORITIES FOR THE MEDICAL RESPONSE TO A CBRN INCIDENT

The priorities for the medical response, within the constraints of the mission, to a CBRN incident are:

a. *Safety*. CBRN is an additional on-scene hazard with the potential for contaminated and contagious casualties. Initial safety considerations are adapted from C-IED drills as the six Cs: *confirm, clear, cordon, control, communicate* and *contain*. If IPE is carried, part of the confirm drill will be to don IPE. Communicate and contain are additional to the conventional four Cs and reinforce the requirement to inform other units and HQ and contain any potential secondary hazards on-scene. For all incidents, personal and

collective safety is of paramount importance and safety assessments should be dynamic.

b. *Cordons*. This includes establishing hot, warm and clean zones, decontamination areas and a CDL (see Chapter 10). For a significant biological incident with risk of person to person spread, cordons may be supported by RoM but with significant tactical and operational, and potentially strategic resource implications.[2]

d. *Control & Communications*. Each zone and area will require a vertical and horizontal command structure, with control of each cordon, to support interoperability between specialist units and the chain of command. The chain of communication parallels the chain of command and the same principles of CBRN warning and reporting apply to the medical management of an incident. Similar or adapted formats used for conventional incidents are recommended (see AMedP-1.10).

d. *Assessment*. Assessment supports the cycle of recognition described. It highlights the importance of a joint scene and casualty assessment, using detect and diagnostic capabilities respectively. Any report of a CBRN agent must include the source of the information (environmental sampling, diagnosis) and the confidence of the assessment (suspected, probable or confirmed).

e. *Triage*. Any incident with multiple casualties requires triage. Conventional triage is adequate for broad casualty discrimination. However as resources and casualty care differ for CBRN, a modified CBRN triage will optimise the provision of casualty care. In extreme situations, such as MASCAL, triage will also support decision making including the use of the expectant (T4) triage category (see Chapter 14).

f. *Treatment*. At the individual level, casualty care follows the principles described in Part 1. The specific treatment for each chemical, biological and radiological casualty is given in Parts 3, 4 and 5 for this publication respectively.

g. *Transport*. The presence of a CBRN agent and risk of secondary contamination or infection has significant implications for MEDEVAC especially by air. Some transport methods may also be limited by aviation and international regulations and require specialist equipment such as casualty protective equipment (CPE) including an air transportable isolator (ATI).

h. *Exploitation*. Once the scene is clear of live casualties, the next phase is a transition to incident recovery. Before this can take place, the incident scene should be secured for forensics in order to investigate the circumstances of the incident (forensics) as well as to gain insight into the agent and/or delivery system used.

i. *Recovery*. Another implication of a CBRN event is the potential loss or compromise of a MTF. Incident recovery will require an assessment of the impact of the incident on the medical support capability for both CBRN and conventional incidents. This is discussed in more detail in Chapter 17.

[2] Conventional major incident response structure uses a colour coded system based on bronze within an inner cordon, silver (prioritising on scene management) and gold (headquarters level).

8.4. TYPES OF CBRN INCIDENTS AND RESPONSES

The type of incident response will depend on the type of incident and to some degree the timeframe. Not all incidents will require a major incident response even if CBRN or hazardous materials are present. A summary of the various types of contingency plans is provided in Figure 8.2. The use of colour coding of plans is recommended.

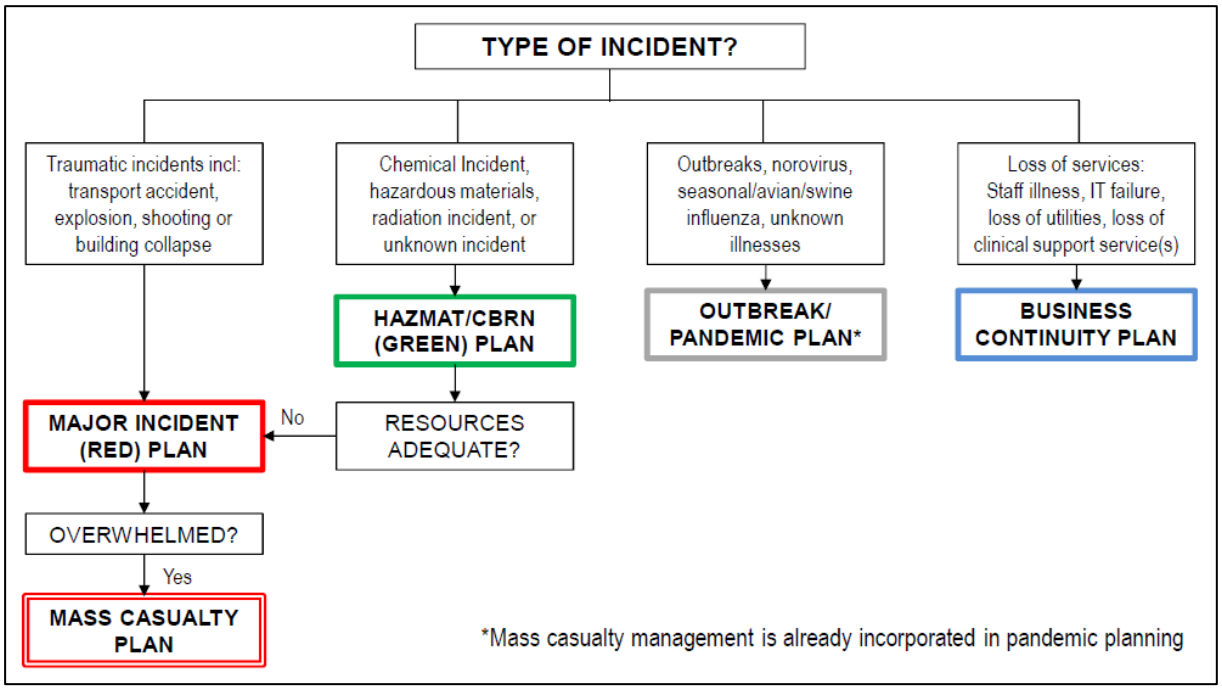

Figure 8-2: Incident Types by Scenario with Illustrative Examples including an Example of Colour-coded Contingency Plans.

8.5. CBRN INCIDENT RESPONSE & FORCE PROTECTION

1. For CBRN defensive operations, CBRN defence and the traditional five components will underpin both pre-event preparations, and post-event interventions including the response phase. For other operations, the conventional incident response and the CBRN modified paradigm will be used for post-event interventions including mitigation. The CBRN medical incident response should therefore be consistent with both a conventional incident response and the five components, described in the Section 1.7 and the medical support of CBRN defence; these are shown in Figure 8-3. Further command guidance can be found in AMedP-7.6.

2. The mission and tactical priorities for on scene commanders include:

   a. Mission-critical objectives (level of risk depends on Command priorities).

   b. Safety of personnel.

   c. Saving of life (casualty care).

   d. Exploitation by the collection of forensic evidence.

   e. Recovery to the pre-event state to continue the Mission.

8.6. SMALL SCALE INCIDENTS

As well as multiple casualty incidents, CBRN agents may be used to target individuals (poisonings and assassinations). These events may not require a full major incident response although some aspects such as safety, cordons and treatment may still be required. These events will still be managed as a criminal act and care should be taken to maintain a chain of evidence. Treatment options however will be the same as larger CBRN incidents.

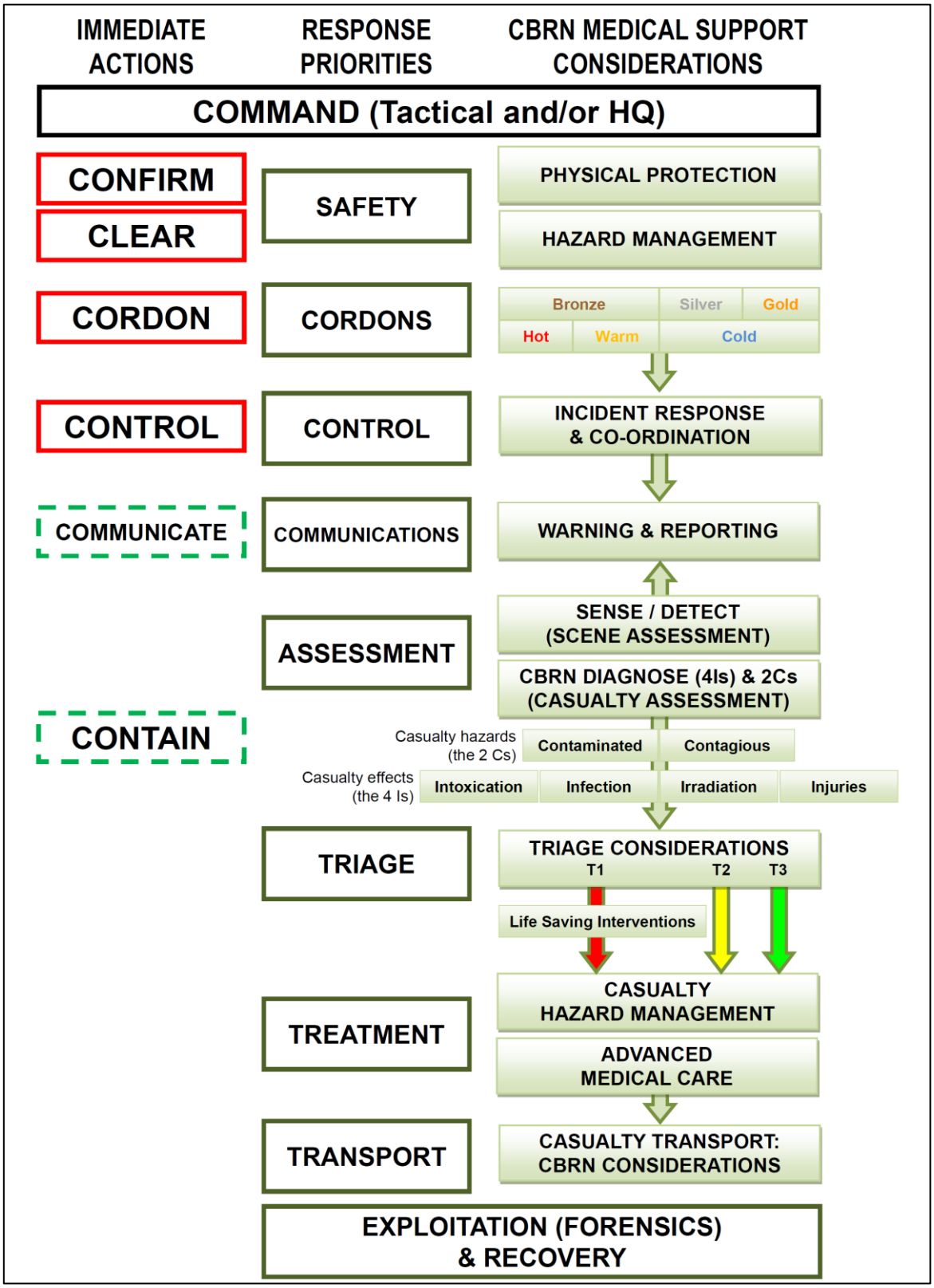

Figure 8-3: CBRN Medical Incident Response and CBRN Defence Capabilities.

CHAPTER 9: SAFETY

9.1. INTRODUCTION

1. For any major incident, safety is paramount and should be the first priority for all commanders. An all-hazards approach applies to both conventional and CBRN incidents and these incidents are not mutually exclusive. Particular attention should be paid to the potential for secondary devices targeting responders using an alternative type of device.

2. Incident safety is an extension of force protection and for CBRN this includes physical protection and hazard management appropriate to the scene and hazard. For the initial management of a CBRN scene, collective protection and thorough decontamination is unlikely to be available. Incident safety will therefore rely on containment, cordon control, individual protection and minimising the spread of contamination.

3. Safety is considered throughout the collective response through to individual casualty management and includes:

a. Donning individual (personnel) protective equipment (IPE) or protective equipment to enable a safe escape (where available).

b. Withdrawing from the immediate hazardous area (‘confirm and clear’).

c. Incident management (‘cordon and control’).

d. Reporting the incident and any associated hazards (‘communicate’).

e. Casualty hazard management (initially ‘contain’).

9.2. INITIAL ACTIONS

The initial actions, based on those for C-IED and shown in Figure 9-1, are the 6C’s:

(1) *Confirm*. Where possible, a CBRN incident should be confirmed using visual assessment or appropriate detection equipment. Respiratory protection if available should be worn immediately followed by a warning to others such as “gas, gas, gas”. If the incident was recognised due to casualties, where possible, the route of exposure (food, water, air or skin) should be identified and reported.

(2) *Clear*. All personnel should clear the immediate area until a formal assessment has been made. The distance and safe direction will be determined by the incident type, type of hazard and wind direction. Where there may be an airborne hazard, the scene should be cleared either in an upwind direction if moving away from the hazard, or perpendicular to the wind direction if in or under a plume.

(3) and (4) *Cordon and Control*. Once the immediate area has been cleared, cordons should be established (physically, virtually or conceptually) to control entry and exit into the hazardous zone. A series of cordons around the scene will be used based upon hazard and security constraints. Each zone within a cordon will have a commander under the overall command of the Incident Commander (see Chapter 10 and 11 respectively).

(5) *Communicate*. Command will be informed of the type of incident (explosive, chemical, biological, radiological and nuclear) and where possible the specific agent. Any

incident update should include location, wind direction and casualty numbers. The report format will be appropriate to the role of the person making the report (first responder, commander, medical personnel) and line of communication. It is vital that an initial report is made as an initial action to ensure the safety of other responders and the integrity of other units including the Headquarters and MTFs (see Chapter 12).

(6) *Contain*. In order to prevent secondary contamination and identify any early medical effects, a containment area should be established for personnel and equipment after clearing the immediate area. All personnel involved in the initial incident should be assessed by an appropriate person for potential exposure and contamination risk before being allowed to leave the scene. During the initial response to a sudden onset incident, there will be no risk of a contagious illness in those personnel exposed to a biological agent. Depending on the incident, the assessment does not need to be medical. Further guidance for casualties is provided in Chapter 6 - Casualty Hazard Management.

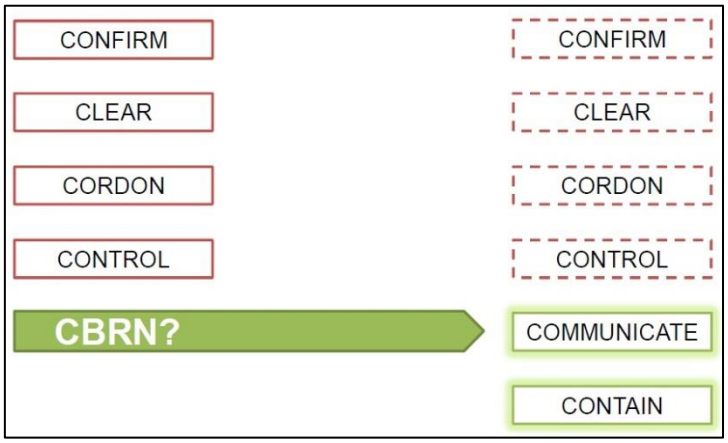

Figure 9-1: The six Cs – CBRN IED and CBRN Incident Initial Actions.

9.3. INDIVIDUAL / PERSONAL PHYSICAL PROTECTION

IPE is a form of physical protection and is a CBRN protective measure. It includes respiratory, eye, hand (gloves) and skin protection and is considered a military form of personal protective equipment (PPE). The selection of IPE / PPE should be based upon a risk assessment. The risk of physical degradation exists in all IPE / PPE. All personnel should be trained to work in the IPE / PPE provided and to monitor for the effects of degradation including physical (heat illness) and psychological stress. ATP-65: *The Effect of Wearing CBRN IPE on Individual and Unit Performance during Military Operations* provides more details specific to IPE use.

9.3.1. RESPIRATORY PROTECTION

The selection of respiratory protection depends on the type of hazard (gas/vapour, liquid and solid aerosol, droplets and large dry particulate). Most forms of respiratory protection require training and in some cases fit testing to ensure an adequate seal and protection; specific attention will be required for personnel with facial hair. The types of respiratory protection include the following and with exception of the first may be a combination:

a. Self-contained breathing apparatus (SCBA). This is the highest level of protection and delivers breathable air for environments with a non-breathable atmosphere.

b. Vapour filters using activated charcoal.

c. Chemical specific filters including those used to protect against toxic industrial chemicals.

d. Particulate filters, protecting against droplets and particulate material including biological agents and radiological particulate material.

9.3.2. PROTECTIVE SUIT (ENSEMBLE)

The selection of suit depends on the type of hazard, concentration, wet or dry contamination risk and risk of physical degradation especially heat illness. Suits include:

a. Surgical gowns.

b. Chemical resistant clothing, optional splash protection or have a splash-proof apron.

c. Splash proof suits with SCBA.

d. Gastight suits with SCBA.

e. For a significant radiological hazard, lead aprons should be considered.

9.3.3. GLOVES

The selection of gloves depends on the degree of manual (medical) dexterity, degree of protection based upon type of agent and concentration and any decontamination role. Examples include:

a. *Butyl gloves*. These are suitable for work in the hot zone and decontamination. When of appropriate size, they can be used for medical procedures.

b. *Double/triple nitrile gloves*. For contact with decontaminated or protected skin within the warm zone, double nitrile gloves provide a balance between chemical resistance and medical dexterity. It is recommended that the outer (sacrificial) layer is changed at least every 15 minutes and between casualties whichever is more frequent. Latex gloves should be avoided. These may also be used when caring for contagious patients with the use of sacrificial layer being important during doffing procedures (strict precautions).

Note: The use of a different coloured inner glove assists in the rapid identification of the inner layer, and any penetration or tearing of the outer layer. Responders should maintain their hands in a surgeon-like sterile stance with hands held forward at 90°.

c. *Single nitrile gloves*. These are suitable for general use (standard precautions).

d. Barrier lotions may be used in addition to the above to provide better protection against penetrating persistent chemical agents such as sulphur mustard and VX nerve agent.

9.3.4. EYE PROTECTION

For respiratory protection that does not come with integral eye protection, this will be an additional protective equipment requirement and is particularly important against biological agents including viral haemorrhagic disease and blood borne viruses.

9.3.5. PERSONAL PROTECTIVE EQUIPMENT FOR MEDICAL PERSONNEL

1. IPE describes the issued personal protective equipment for use in an operational environment. PPE is a more general term and can apply to IPE and other equipment used to protect medical personnel as well as other occupations. The choice of PPE for medical personnel should also include benefits such as human factors, non-verbal communication, visual fields and direct visual contact with casualties. The latter is an important contribution to reducing casualty psychological stress especially when dealing with vulnerable casualties including children.

2. The warfare canisters of military respirators provide protection from traditional chemical warfare agents such as pulmonary agents, either by adsorption or chemical reaction. Some models will also have toxic industrial chemical (TIC) variants. However only limited or temporary protection against high concentrations of TICs and products of combustion can be assumed, and are not to be used in oxygen-depleted or carbon monoxide environments. Self-contained breathing apparatus should therefore be worn by emergency responders. This additional PPE requirement makes medical management more difficult due to a loss of medical dexterity and limited to casualty assessment and very basic interventions (first aid) such as tourniquet application, administration of auto-injectors and basic airway management. For this reason and depending on casevac timelines, medical personnel may be better positioned to receive casualties at the hot-warm zone interface (fwd CPP) in a more permissible PPE for effective medical management.

9.3.6. CBRN HYGIENE AND DRILLS

The same requirement for CBRN hygiene is required by responders and includes restricting eating, drinking and smoking. Responders must decontaminate, remove IPE before entering the clean area or shelter in collective protection before resting, eating and drinking according to the demand and resources available to the response.

9.3.7. LEVELS OF INDIVIDUAL (PERSONAL) PROTECTIVE EQUIPMENT

A standardised classification for IPE (PPE) is recommended and is summarised in Table 9-1:

a. *Level A*. This level provides the greatest level of skin and respiratory protection, and includes SCBA. It is intended for use in a non-breathable or unknown enclosed environment. Disadvantages include limited effective time on scene, increased logistic resources (provision of air supply) and increased physical degradation.

b. *Level B*. This level provides the greatest level of respiratory protection (SCBA) but with a reduced (chemical resistant or splash-proof) skin protection.

c. *Level C*. This level is worn when the type and concentration of the airborne hazard is known and the criteria for using filtered (air-purifying) respirators are met. Respirators may be powered (PR) or unpowered I. Powered respirators will reduce the work of breathing but have time constraints and logistical implications. This level of protection is also applicable to casualty bags and may be powered or unpowered filter protection.

d. *Level D*. This is the lowest level of protection and includes standard precautions. This PPE has respiratory, eye and glove protection and will provide significant protection against droplet and aerosolised biological hazards as well as dry particulate radiological material. The type of respiratory hazard (droplet or airborne/aerosolised) will determine

the level of respiratory particulate protection i.e. surgical mask or N95/EN:FFP2 or 3)[1]. Any level D protection should therefore be caveated by the level of respiratory protection required (none, standard / droplet precautions (surgical mask), airborne hazard (high specification mask)).

Table 9-1: Summary and Examples of Individual (Personal) Protection Equipment.

LEVEL			PROTECTION			
			RESPIRATORY	SKIN	GLOVES	EYE
A			SCBA	Gas-tight suit	Integral	Integral
B			SCBA	Chemical resistant or splash proof	Integral	Integral
C	IPE		Respirator	Chemical resistant	Butyl	Integral
	EMT medics in warm zone		Respirator	Chemical resistant	Triple/double nitrile*	Integral
	Decontamination operative		Respirator	Chemical / splash resistant	Butyl	Integral
D	Standard precautions		(optional)	Physical barrier only	Nitrile	-
	Respiratory precautions	Droplet respiratory hazard	Surgical mask	Physical barrier only	Nitrile	Risk assess
		(with aerosolising procedure)	High specification mask			
		Airborne hazard	High specification mask	Physical barrier only	Nitrile	Risk assess
	Strict precautions - High risk biological agent (e.g. Ebola, Marburg, or blood borne virus and trauma)		High specification mask, or full face respirator	Splash resistant	Double nitrile	Full face coverage / visor
			Strict precautions require strict adherence to donning and doffing drills including the use of sacrificial layers and clinical waste management			
	Radiological particulate hazard		Surgical mask	Physical barrier only	Double nitrile	Yes

Adapted from WHO Infection Control Guidance and US Occupational Safety and Health Administration (OSHA) criteria.
*Triple/double nitrile gloves are used with the outer glove being changed between casualties or every 15 minutes whichever is shortest.

[1] N95 and EN:FFP1-3 are US and EU regulatory classifications of respiratory protection respectively based upon efficacy i.e. N95 (95%) and FFP3 (99%). EU legislation requires fit testing for FFP2 and FFP3 respiratory protection.

9.3.8. HIGH DOSE RADIOLOGICAL PERSONAL PROTECTION

It should be noted that the individual physical protection described above does not provide adequate protection against high dose point source gamma radiation. The mainstay of protection will be based upon reducing time on scene and maintaining distance (exclusion zone).

9.4. COLLECTIVE PHYSICAL PROTECTION

1. Collective Protection (COLPRO) during the initial response to a CBRN incident is unlikely unless a CBRN defensive operation and the pre-event threat was significant. However, some COLPRO or ad hoc shelter may be available depending on the responding unit and vehicles, and nearby infrastructure. Shelters may be hardened against CBRN hazards by enhancing and/or filtering building ventilation with positive pressure, avoiding downwind structures and posting sentries with appropriate monitoring equipment.

2. Where limited COLPRO exists, priority should be given to the Incident Command Post including communications. While an ad hoc medical treatment facility such as a casualty clearing station may benefit from COLPRO, limited casualty decontamination may limit its effectiveness and integrity. MTF COLPRO may therefore not be required if there is an adequate method for casualty transportation to an established MTF with casualty decontamination facilities in a suitable medical timeframe.

3. For CBRN defence operations COLPRO may be deployed for key infrastructure and its requirement is set in STANAG 4639: *Collective Protection in the CBRN Environment.*

9.4.1. REVERSE COLLECTIVE PROTECTION

Isolation facilities or biocontainment are required to keep the hazard inside especially for the management of contagious patients. Ventilation may be under negative pressure, as well as barrier nursing, and support areas providing either a patient care or laboratory function. Laboratories may make use of negative pressure glove boxes or be entirely enclosed, with supporting control measures. The size of a patient care area may either be at:

a. An individual level (barrier nursing/isolation room);

b. A cohort within a MTP (isolation ward); or

c. A separate infectious disease cohort MTF.

9.5. BIOSAFETY LEVEL LABORATORY FACILITIES

Laboratory facilities are classified into four levels of biosafety from BSL-1 being the lowest and BSL-4 being the highest. The core standard is BSL 1-2. But a bioresponse may require more secure facilities on site. BSL-4 facilities are generally used for lethal agents with airborne transmissibility, and/or limited or no effective vaccination or treatment; agents include smallpox and VHF including Ebola, Lassa and Marburg.[2]

[2] CDC HHS (CDC) 21-1112, and EU Council Directive 90/679/EEC.

CHAPTER 10: CORDONS

10.1. INTRODUCTION

1. For any incident involving a conventional or CBRN hazard, cordons are established to control and monitor entry and exit. The presence of a CBRN agent requires additional on scene hazard management to minimise the risk to responders and mitigate the effects of the hazard on casualties and exposed persons. The shape of the zones and surrounding cordons will depend on the type and quantity of hazard, and wind direction and speed. For CBRN defensive operations, ambient data such as wind direction and speed as well as detection equipment will inform the incident commander's decisions while non-CBRN defensive operations will require a risk assessment with a greater margin of error.

2. For each cordon and zone, priorities for casualty management are set (see Chapter 4) but the ultimate aim is the evacuation of the casualty to a more permissive environment and one with the optimal medical capability. Medical planning should ensure that each transfer is to a medical facility or unit with at least the same or greater capability including those used for medical evacuation.

10.2. CBRN ZONES

Some NATO nations use incident command systems although the terminology may vary. Most use concentric incident geometry around an incident site and use hierarchy such as *bronze, silver* and *gold*. The bronze zone is often associated with on scene hazards contained inside an *inner cordon*. For CBRN incidents, the bronze zone may be further split by an additional boundary (hazard threshold) generating a buffer zone acting as a decontamination zone (see Figure 10-1).

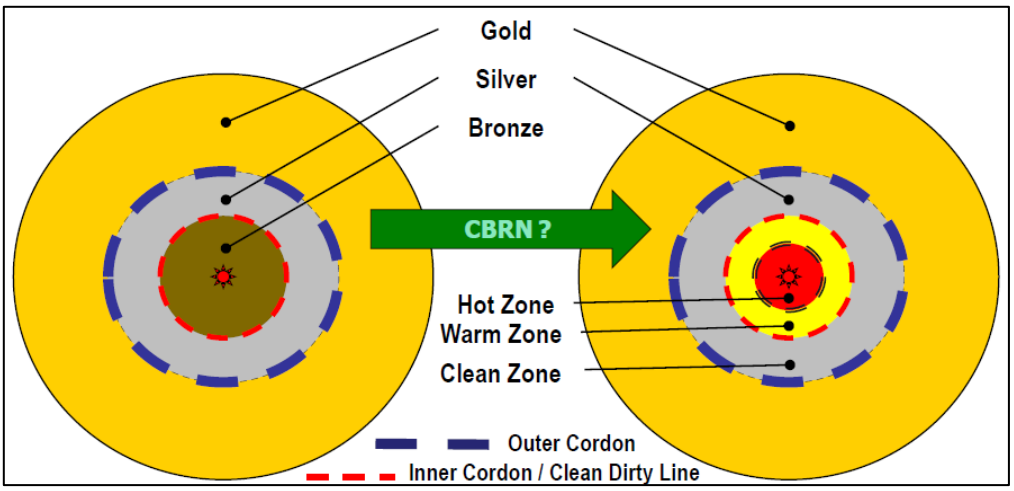

Figure 10-1: Zone Comparison for CBRN Incident Response (Example: MIMMS[1] system).

10.2.1. HOT (EXCLUSION) ZONE

This is a non-permissive area where there is a direct hazard (primary exposure and contamination) to the responder from the environment. This will be mitigated by appropriate

[1] AMedP-1.10: *Medical Management of a Major Incident and Mass Casualty Situation*.

protective measures including individual protective equipment and time management. Casualty management in this zone will be limited to:

a. Life sign assessment.

b. First aid – Trauma and CBRN.

c. Triage.

d. Casualty assessment ('Quick Look') – T1 only.

e. Life-saving interventions (CAaB) – T1 only.

f. Analgesia to facilitate extrication – entrapped casualties only.

g. Evacuation.

10.2.2. EXCLUSION ZONE

1. An exclusion zone is a hot zone where despite protective measures a significant risk remains to the responder that can only be mitigated by clearing the area. Examples of the use of an exclusion zone would be an area around an explosive device, high dose rate radiation source or nuclear weapon. Chemical incidents may require an exclusion zone depending on concentration of an agent, lack of oxygen and potential explosive chemical reactions. The use of an exclusion zone around a biological hazard is unlikely.

2. The criteria for the radii(*r*) of the cordon from the incident site depends on a number of factors such as type of hazard, size (yield) of an explosive or nuclear device and dose rate from a radiological point source. Common default cordons include:

a. Large (car-sized) explosive device	400m
b. Radiological point source	100µGy/hr
c. Explosive radiological dispersal device	a. or b. whichever the greater
d. Chemical spill	See Downwind (Plume) Hazard below.

3. Any work within the exclusion zone that carries a health risk such as radiation exposure must be recorded and appropriate medical screening or health surveillance must be arranged.

10.2.3. WARM ZONE

This is a semi-permissive or buffer area beyond a primary hazard threshold, where there is an indirect hazard to responders due to secondary cross-contamination from equipment, personnel and casualties transiting from the hot zone. Casualty management in this zone will include:

a. Triage and prioritisation for decontamination, if required.

b. Casualty assessment ('Quick Look' and primary survey).

c. Life-saving interventions (CAaBC) – T1 only.

d. MedCM administration to prevent deterioration depending on agent.

e. Management of T4 (Expectant) casualties.

f. External decontamination, as required.

g. Wound decontamination and dressing change, as required.

h. Evacuation.

10.2.4. COLD (CLEAN) ZONE

This zone allows for supportive and definitive care to be undertaken. Casualties may still have residual contamination (wound and internal) and depending on timescale may have the potential to be contagious. For the latter, isolation may still be required.

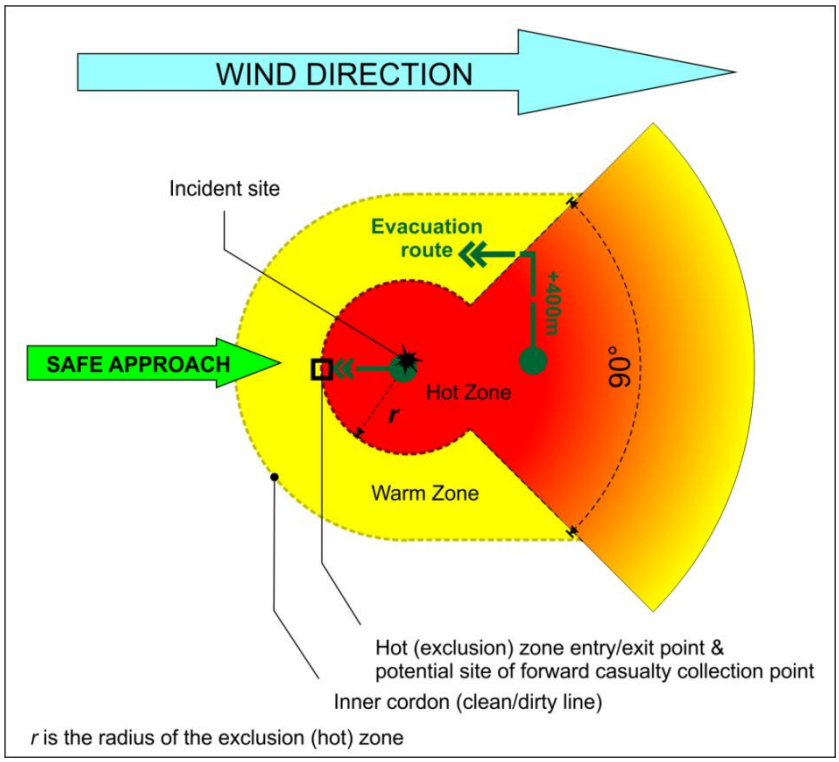

Figure 10-2: Incident Management and Downwind Plume.

10.3. DOWNWIND (PLUME) HAZARD

1. Where the wind speed is greater than 10km/h a downwind hazard will be assumed. For CBRN defensive operations or where the force protection has access to a cell controller, plume modelling will be based on ATP-45: *Warning and Reporting and Hazard Prediction of CBRN* and *2012 Emergency Response Guidebook* (ERG) – see Table 10-1.

2. Where hazard prediction (plume modelling) is not available a downwind hazard with a wider safety margin and 90° arc is recommended. An approximate wind direction can be estimated by the use of signal smoke. All medical personnel are recommended to approach the scene from upwind. Evacuation from in or under the plume should be perpendicular to the wind direction, away from the midline and at least the radius of any exclusion or hot zone (+400m) (see **Error! Reference source not found.**10-2).

10.4. WARM ZONE INFRASTRUCTURE

Within the warm zone the following elements are required:

a. Forward Casualty Collection Point (Fwd CCP) at the hot/warm interface.

b. Casualty decontamination area (centre or unit) with:

(1) Casualty Collection Point (CCP)

(2) Triage point.

(3) Walking (ambulatory) channel.

(4) Stretcher (non-ambulatory) channel with EMT provision as required.

(5) Body handling area.

c. Personnel decontamination area

d. Equipment decontamination area.

e. Personnel entry point(s).

10.5. CLEAN DIRTY LINE

1. The clean / dirty line (CDL) is the cordon after casualty decontamination (and personnel / equipment) has taken place. The CDL is effectively the edge of the hazardous zone, sometimes referred to as the bronze zone and the inner cordon.[2]

2. Although crossing the CDL implies that full casualty decontamination has taken place, this should also be formally declared for each casualty. In some cases, residual hazards may exist and include:

a. Wound or dressing contamination.

b. Radiological contamination in the T1 surgical casualty requiring damage control surgery before full casualty decontamination.

c. Contagious casualty.

3. For casualty management, the CDL may be the first point that casualty documentation may have been generated and a formal handover of care should take place. The handover should include as much information as possible and the minimum of an AT-MIST report (see Chapter 12 - Communications).

10.6. RESTRICTION OF MOVEMENT AND CORDONS

Implementation of restriction of movement is unlikely to require a specific cordon to be raised as it will use established boundaries such as a perimeter fence or geographic feature. Strategic

[2] ☑ ***Lesson Learnt***. The CDL should be well marked and delineated by suitable CBRN warning symbols (e.g. orange or specific hazard tape) to prevent accident crossing.

RoM is likely to use national borders and ports of entry. Sentries to control and monitor operational RoM will be required and the requirements are listed in Chapter 6.

Table 10-1 – Chemical Downwind Plumes.

Chemical agent	Small spills*		Large spills*	
	Hot zone		Hot zone	
	Day (m)	Night (m)	Day (m)	Night (m)
Tabun	200	200	500	600
Sarin	400	1100	2100	4900
Soman	400	700	1800	2700
VX	100	100	400	300
Sulphur mustard	100	100	300	400
Lewisite	100	300	500	1000
Chlorine	400	1500	3000	7900
Phosgene	800	3200	7500	11000+
Hydrogen cyanide	300	1000	3700	8400
Cyanogen chloride	1000	3800	5700	11000+
BZ	400	1700	2200	8100

* Small spills are considered as coming from a single small package (e.g. a drum up to 208 litres), a small cylinder, or a small leak from a larger package. For nerve agents, vesciants and BZ, small spills are considered up to 2kg.
* Large spills are considered as coming from a large package, or multiple spills from small packages. For nerve agents, vescicants and BZ, large spills are considered up to 25kg.

The QR code provides a link to the ERG guidance in accordance with ATP-45.

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CHAPTER 11: COMMAND AND CONTROL

11.1. INTRODUCTION

Command is an important enabler for any incident response after scene safety and appropriate cordon(s) have been established to ensure the safety of the personnel and optimal casualty care. Once cordons have been established, control of the entry and exit of personnel is vital to maintaining safety and overall command of the scene. Lines of command and control include vertical command within unit and chain of command, and horizontal control to support joint or multi-agency response and maintain the integrity of the scene management and safety.

11.2. ON SCENE COMMAND AND CONTROL

The on scene command structure for a CBRN incident should follow that for any other major incident with a commander responsible for each zone or element within the zone. Control is deferred to each of the cordons established with particular emphasis on safe entry and exit as well as the control of any contamination hazard at the CDL or movement of a contagious patient.

11.3. STRATEGIC COMMAND

1. For CBRN incidents, there will be significant interest that may be disproportionate to the incident, especially if it is a release of a contagious biological agent or nuclear incident. These interests include:

    a. International health regulations.

    b. International travel regulations.

    c. Media interest.

    d. Potential contravention of CBRN related arms control.

    e. Post incident recovery including decontamination.

2. The strategic command of a CBRN incident is likely to include representation from organisations including:

    a. Host nation.

    b. NATO.

    c. Allied National Commands and Governments.

    d. International bodies such as the World Health Organisation, United Nations and International Atomic Energy Agency.

    e. Governments of neighbouring and regional nations.

3. The actual composition and organisation of the strategic command structure in response to a large CBRN incident will depend on the type of incident, NATO operation and pre-planning, as well as political context and regional stability.

11.4. CBRN MEDICAL DECISION POINTS

During any operation a number of scenarios may occur. Pre-event, response and recovery planning will identify significant key decision points (DP) that may require a command or medical decision to be made. Examples of these CBRN medical related DP are:

a. Implementation of a vaccination program to a specific threat, if available.

b. Mobilisation of a CBRN capable (COLPRO) MTF.

c. Mobilisation of CBRN Medical personnel.

d. Mobilisation of CBRN Support personnel – decontamination teams.

e. Issuing of MedCM to personnel.

f. Starting pre-event MedCM.

g. Starting post-event MedCM.

h. Closing an MTF due to contamination, if required.

i. Requesting mutual aid from Host Nation, other partners or networks.

j. Implementation of quarantine.

k. Implementation of RoM.

l. Requesting an RDOIT, MRIIT, other deployed specialised team or reach back support.

m. Easing RoM.

n. Re-opening an MTF after decontamination, if required.

11.5. MEDICAL AND CBRN ADVISORS TO THE COMMAND CHAIN

1. Each level of Command requires incident specific advice and this is especially important for CBRN. During NATO CBRN defensive operations, CBRN and medical advisors will be appointed to the CBRN and medical chain of commands as well as CBRN specialist units such as the Joint Assessment Team, CBRN Battalion, RDOIT and MRIIT.

2. For conventional operations, the role of CBRN Advisor is unlikely to be an individual role and will be incorporated into a force protection role and medical operations role for Command and Medical CBRN advisors respectively.

11.5.1. AD HOC CBRN MEDICAL ADVISORY GROUP ON OPERATIONS[1]

1. During a CBRN event, there is potential for conflicting medical advice between the medical support to the NATO CBRN Task Force and Joint Medical Support (JMED). Any

[1] ☑ *Lesson Learnt*. During operations, there is a potential for multiple sources of medical advice outide of the JMed chain, which may be conflicting, being presented to Command. In order to avoid this, all medical advice including casualty care, and CBRN & outbreak response must be channelled through the Medical Advisor.

medical advice to the Joint Combined Force Commander is directed through the Medical Advisor (MEDAD) to ensure consistent advice and coherent risk communication. The MEDAD must also ensure that any decision or advice is based on appropriate specialist advice including the use of CBRN medical SMEs and reach back. During a CBRN incident, the MEDAD may chair an ad hoc Medical Advisory Group on Operations (MAGO) with membership including the representatives from the medical support organisation, RDOIT or MRIIT commanders, JAT medical advisor(s) and potentially host nation or regional medical organisations including public health, and scientific advisor (SCIAD) (see Fig 11-1).

2. Host Nation health organisations will have public health primacy for the health of the local population.

3. The Chair of the MAGO (i.e. MEDAD) should consider the invitation of other organisations for incidents which may have a wider publich health impact. They include:

a. Host Nation Public Health;

b. Regional Public Health;

c. WHO local representative; and.

d. Potentially representatives or liaisons for opposing force(s), especially for operations with an active PHEIC.

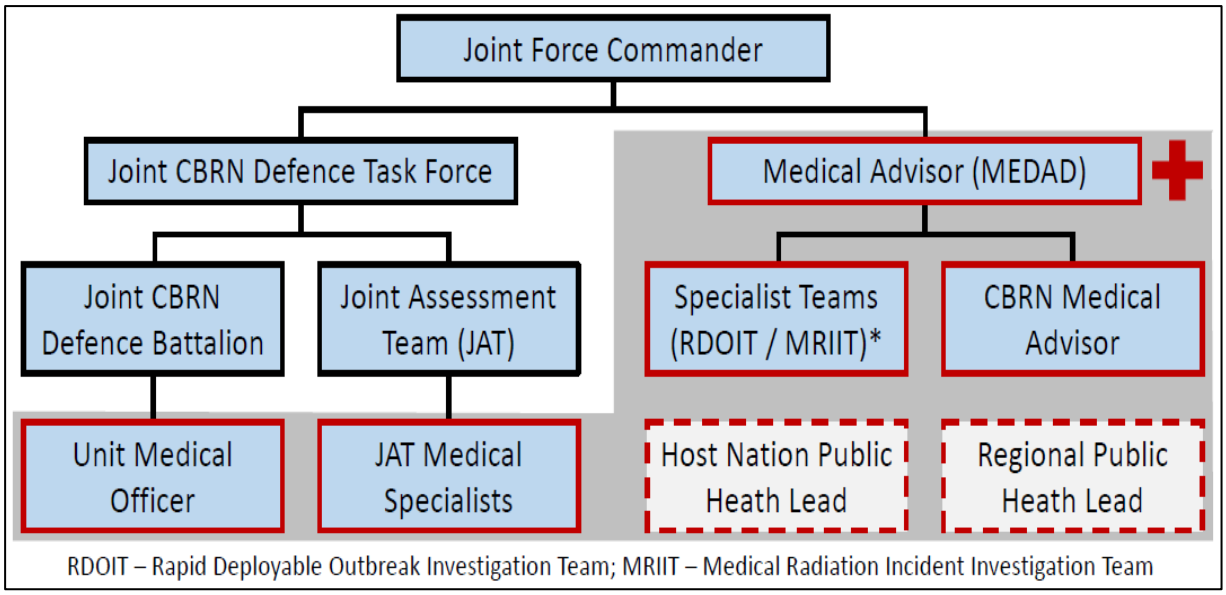

Figure 11.1: CBRN Medical Advisory Group on Operations (MAGO) (shown within grey background)**.**

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CHAPTER 12: COMMUNICATIONS

12.1. INTRODUCTION

1. The lines of communication will follow the vertical chain of command. Local communication horizontally will allow for liaison between units and agencies at the same command level such as on scene and at the headquarters. It is vital that the presence and type of CBRN agent is reported between the various medical and decontamination facilities including the CDA Commander and MTF operations room.

2. Most of the report formats for any major incident include some form of hazard assessment. Specialised reports may be specific to CBRN incidents and are more likely to be used during CBRN defensive operations between CBRN specialists. A modified conventional report may provide information that is specific to a CBRN incident but using a familiar format.

3. Due to the risks of cross-contamination, non-contact methods of information management including verbal and technological solutions should be considered. This is especially important at handover points such as the CCP, CDL and other points in the conventional medical evacuation chain.

12.2. CBRN METHANE REPORT

1. The initial major incident report will be made using the standard METHANE report as described in operation specific standard operating procedures and NATO MIMMS. Once a CBRN incident has been recognised, subsequent reports can be made using the modified CBRN METHANE report. The CBRN METHANE report is a medical report using the template from a conventional METHANE report but with minor amendments reflecting CBRN specific issue. The use of the conventional report will not significantly impair the response but the CBRN format will optimise the response and support Command decision making and risk assessment. The report consist of:

a. **M**y call sign and frequency.

b. **E**xact location, timing and wind directions.

c. **T**ype of incident (explosive, CBRN and whether deliberate, accidental or unknown)

d. **H**azards identified (C, B, R, Ex and type of agent if known, or unknown)

e. **A**ssessment (Scene and Casualty) (see Chapter 13) / **A**ccess.

f. **N**umbers of casualties and population at risk or exposed.

g. **E**mergency resources on scene and required.

2. The CBRN METHANE report may be repeated to provide an update including a revised casualty estimation and resources required.

12.3. CBRN WARNING AND REPORTING

In addition to conventional medical incident reporting systems, CBRN incidents have specific reporting requirements directed by ATP-45: *CBRN Warning and Reporting*. A CBRN 1 Report Form is to be completed.

Table 12-1: CBRN METHANE Report.

	Subject	Subject	
1	**M**y call sign	My call sign & frequency	
2	**E**xact location and wind direction	Location of incident include approximate wind direction (use smoke as indicator)	
3	**T**ype of incident	**A**: Deliberate release (Attack) **B**: Accident release (ROTA) **U**: Unknown	
4	**H**azards identified	**U**: Unknown or: **C**: Chemical - Nil (0) / Suspect (1) / Probable (2) / Confirmed (3) **B**: Biological - Nil (0) / Suspect (1) / Probable (2) / Confirmed (3) **R**: Radiological - Nil (0) / Suspect (1) / Probable (2) / Confirmed (3) **E**: Explosives - Nil (0) / Suspect (1) / Probable (2) / Confirmed (3)	
5	CBRN **A**ssessment	**A**: Scene Assessment including DIM (Detect) **B**: Casualty Assessment including signs & symptoms (Diagnose)	
6	**N**umber of casualties	**T1**: Requiring life-saving interventions **T2**: Litter (Stretcher) or Incapacitated **T3**: Ambulatory (Walking) **D**: Dead **(T4**: Expectant (if authorised))	**X**: Exposed person (well) **C**: Contamination hazard
7	**E**mergency resources	Free text: Description of resources on scene and those required (including decontamination and access to stockpiles), and treatment given.	

12.4. AT-MIST-D REPORT

1. At handover points, the time and ability to handover casualty information may be limited. It must include any residual risk from contamination or contagious disease. The AT-MIST format (see Annex 12D) is:

   a. **A**ge.

   b. **T**ime of injury or exposure.

   c. **M**echanism of injury or exposure (explosive incident, CBRN device, unknown).

   d. **I**njuries, **I**ntoxication, **I**nfection, **I**rradiation.

   e. **S**ymptoms and signs.

   f. **T**reatment (MedCM, supportive / definitive treatment & hazard management).

   g. **D**econtamination status (No contamination, residual including wound, decontaminated)

2. Any residual risk of contamination or contagious disease should also be handed over or indicated on the casualty.[1]

[1] ☑ *Lesson Learnt*. During exercises, it was noted that the casualty decontamination status may be overlooked both on receiving and handing over casualties between responders, facilities and nations. The AT-MIST report has been adapted to prompt responders in a CBRN-threat environment to consider and handover the decontamination status at AT-MIST-D.

12.5. CBRN CASUALTY REPORT FORM

1. The reporting and documentation of CBRN casualties is important. The consistency of the report is vital for both surveillance and casualty reporting through the chain of command. Medical documentation can also be used to act as an aide-memoir to prompt clinical to look for the right signs based on the reported symptoms. The CBRN Casualty Report Form provides an initial structured format for casualties presenting after a CBRN or combined incident. The form uses a standardised approach that can be used by member nations.

2. It is likely that the CBRN Casualty Medical Form will be completed either at the clean/dirty line following a verbal AT-MIST report or at the receiving MTF. Additional CBRN medical documentation can be included to support agent specific management and assessment including radiation and sepsis management.

3. Some MedCM differ between nations and this is reflected in a nation specific part of the form, while the generic approach will be the same. Annex 12A-2 provides a template for the CBRN Medical Report Form and the QR code provides a downloadable version.

12.6. NATO REQUEST FOR MEDICAL EVACUATION (NATO 9-LINER MEDEVAC)

The 9-liner is used to request medical evacuation. While this format is intended for conventional casualties, priority and triage categories are consistent for both types of incidents. Line 4 is used to request specialist equipment and can be used for decontamination equipment or MedCM, while line 9 highlights features of the pick-up zone and could be used to report CBRN specific features or exclusion zone.

12.7. INFORMATION MANAGEMENT AND COMMUNICATION

1. Historically during any major incident or military operation, communication has often been found to be inadequate or overwhelmed. The flow of information is even more likely to be compromised by a CBRN event due to operational security, contamination of paper reports including the casualty record, loss of information technology (IT) systems due to a nuclear event or speculation over the cause and implications of a CBRN-type event.

2. A well-managed flow of information with a joint communication strategy and contingency plan will help maintain situational awareness, warning and reporting systems, flow of casualty information and improve risk communication.

3. Medical planning for any CBRN incident should consider the requirements for information management and communication for medical support including:

a. Disease and health surveillance.

b. Optimisation of casualty information between forward responders and MTFs as well as across the clean / dirty line.

c. Use of computer tools to support complex casualty and incident management issues, including triage and casualty reporting.

d. Clinical investigation and reporting including the use of reach back or host nation support.

e. IT back up.

f. Warning and reporting systems with interoperability to non-medical systems.

g. Casualty registries and follow-up.

h. Outbreak investigation and epidemiology case reporting.

i. Recording of MedCM use and any adverse events.

j. Interaction with the media.

k. Strategic communication with regional and international health organisations.

l. Strategic communication with non-government organisations.

m. Medical messaging of restriction of movement requirements.

n. Risk communication to patients and exposed populations.

ANNEX 12A – CBRN MEDICAL REPORT FORMS

Identification:				(If known)
A	**Age of casualty** (adult / child (+ age))			
T	**Time of wound / exposure or time of onset of symptoms**			
M	**Mechanism of injury or type of incident**			
I	**Injuries** (including injury pattern & observed injuries)	**Intoxication** (type, route of exposure, & contamination risk)		**Infection** **Irradiation**
S	**Symptoms and signs** (including toxidromes)			**Other:**
	<C>at haem:	**C**onsciousness		
	A	**R**esp		
	B	**E**yes		
	Circ	**S**ecretions		
	D	**S**kin		
T	**Treatment given:**	**NA antidotes**	**Auto-injector**	**Other MedCM:**
			Atropine	
			Oxime	
			Benz	
D	**Decontamination status:** (no contamination; fully decontamination; wound contamination; internal hazard)			

Fig 12A-1: AT-MIST Report Form.

*(<C>at haem = catastrophic haemorrhage)

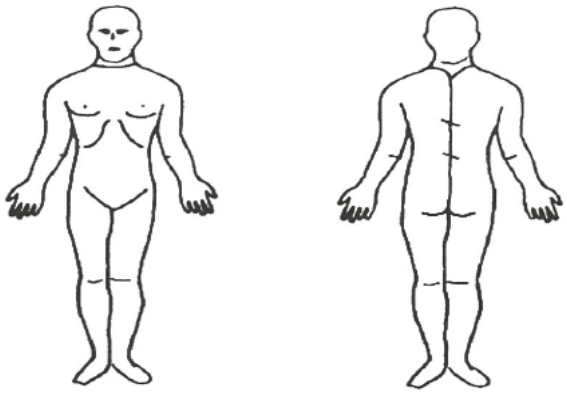
RESTRICTED – MEDICAL (when completed) CBRN Med Form 1

CBRN MEDICAL REPORT FORM

Name:		Date: / /	Sex: Male / Female	Age:	or DOB: / /
Nationality:	Rank:	Service No:	Service:		Unit:
Location:		Incident time (if overt): :	Time of symptom onset: :		Arrival time: :

Type of Incident:	❑ Chemical [suspected agent] ❑ Biological [suspected agent] ❑ Radiological ❑ Nuclear ❑ Explosive [type] ❑ Other [ ] ❑ Suspected ❑ Probable ❑ Confirmed ❑ DIM equipment used [ ] Reading [ ]
Physical Protection:	Respiratory [CBRN ❑ / Particulate ❑ / Other_____] ❑ Gloves ❑ Protective suit ❑ Other [ ]
Pre-Exposure MedCM:	❑ Chem [ ] ❑ Bio [ ] ❑ Rad [ ]

INJURIES & CONTAMINATION:



Fracture +++ Wound //// Contaminated area

CBRN QUICK LOOK

Conscious	❑ Alert ❑ Verbal ❑ Pain ❑ Unconscious ❑ Fitting
Respiratory	❑ Normal ❑ Abnormal ❑ Asymmetrical ❑ Absent /min
Eyes	❑ Pinpoint ❑ Normal ❑ Wide
Secretions	❑ Normal ❑ Secretions ❑ Dry
Skin	❑ Normal ❑ Sweaty ❑ Cyanosed ❑ Pink ❑ Purpuric rash BURNS ❑ Chemical ❑ Thermal
Other	Temp: Pulse ❑ Rad ❑ Fem ❑ Carotid ECG ❑ Sinus Rate /min ❑ Abnormal Radiation: ❑ Vomiting or ❑ Diarrhoea onset [:]

EMERGENCY MEDICAL TREATMENT AND HAZARD MANAGEMENT

INITIAL TRIAGE T	HAZARD: ❑ Gas/Vapour ❑ Liquid ❑ Dry/particulate ❑ Wound ❑ Unknown ❑ Contagious (suspected) MANAGEMENT: ❑ Removal of clothing ❑ Dry contamination ❑ Rinse ❑ Full wet contamination ❑ Isolation
Catastrophic Haemorrhage:	Site(s): [ ] [ ] [ ] [ ] ❑ CAT Applied Time:[:] ❑ Haemostatic Time:[:] ❑ FFD Site(s): [ ] [ ]
Airway:	❑ OPA / NPA Size: [ ] ❑ LMA Size: [ ] ❑ ETT Size: [at] ❑ RSI Time:[:] ❑ Surgical Airway
Antidotes / MedCMs & other therapeutics:	❑ ComboPens Number given [ ] ❑ Oxime [ ] total [ ] ❑ Atropine total [ ] ❑ Benzodiazepine [ ] total [ ] ❑ Naloxone total [ ] ❑ Amyl nitrite Dicobalt edetate ❑ 300mg ❑ 600mg & ❑ Glucose ❑ Sodium nitrite ❑ Sodium thiosulphate ANTIBIOTIC(S): [1:] dose [ ] [2:] dose [ ] [3:] dose [ ] OTHERS: ❑ Morphine total [ ] ❑ Fentanyl total [ ] ❑ Ketamine total [ ] ❑ Ondansetron dose [ ] [1:] dose [ ] [2:] dose [ ] [3:] dose [ ]
Breathing:	❑ Oxygen ❑ BVM Needle decompression ❑ L ❑ R Thoracostomy ❑ / Chest drain ❑: L ❑ R ❑
Circulation:	❑ IV/IO Site: [ ] Size: [ ] ❑ IV/IO Site: [ ] Size: [ ] ❑ CPR duration [mins] FLUIDS: ❑ Crystalloid: [ ] Volume: [ ] ❑ Blood: [ ] Volume: [ ]
Other interventions and comments:	

COLD ZONE TRIAGE CAT	T	OUTCOME	❑ Casualty Clearing Station ❑ Survivor Reception Centre ❑ RTU/Home ❑ MTF/Hospital Name: [ ] ❑ Mortuary ❑ Other: [ ]
CDL Handover Time	:	Completed by:	Initials

FOR RAD / NUC INCIDENTS: REFER TO RADIATION WORKSHEETS WITH CONTAMINATION CHARTS AND BIODOSIMETRY ASSESSMENT

RESTRICTED – MEDICAL (when completed) Version 1.2 (Jan 13)

Fig 12A-2: CBRN Medical Report Form. (Red section for nation specific antidotes)

CHAPTER 13: ASSESSMENT

13.1. INTRODUCTION

In order to optimise CBRN incident response it is necessary to make an assessment of the incident requirements. CBRN incident assessment contributes to the cycle of recognition (see Chapter 5) as well as defining resource requirements. The most obvious indicator of a CBRN incident is the presence of casualties without obvious traumatic injuries. The assessment elements are:

a. Scene (environmental) assessment – detect.

b. Casualty (clinical) assessment – diagnose.

c. Resource assessment.

13.2. SCENE ASSESSMENT

1. The scene assessment includes:

a. Visual assessment.

b. Reports of smells (see Table 13-1).

c. Detection, Identification and Monitoring (DIM, see Chapter 5).

2. Visual assessment is the first assessment made by responders in appropriate personal protective equipment. Particular attention should be made of visual CBRN combat indicators such as obvious downwind plume (including size, height, direction, colour and in some cases smell reported by personnel), dead animals or vegetation and warning indicators including hazard diamonds (Figure 13-1).

13.3. CASUALTY ASSESSMENT (4 Is)

1. Casualty assessment is required for several reasons:

a. Contribution to the cycle of recognition.

b. Identification of exposed persons that have signs of exposure (4 Is):

(1) Intoxication.

(2) Infection.

(3) Irradiation.

(4) Injuries.

c. Identification of casualties with contamination and therefore requiring decontamination.

2. The findings of the assessment will then be reported and appropriate resources assigned.

13.4. RESOURCE ASSESSMENT

The assessment of the scene for the purposes of resources is based upon resources available and demand. The initial incident reports will inform Command of the number of casualties. This assessment will inform the HQ of specific requirements. As the incident evolves addition reports using the METHANE report can be made. Resource assessment should be structured and assess:

a. Force Protection requirements.

b. Casualty numbers and breakdown.

c. Resources available on scene, including local health facilities.

d. Medical personnel required.

e. Medical equipment required.

f. MedCM required.

g. Method of casualty evacuation.

13.5. REPORTING OF ASSESSMENT RESULTS

It is vital that any positive assessment findings or any important negatives are reported through the chain of command and between MTFs. Any receiving or local MTFs should be notified of any hazard that may be responsible for the incident and clinical effects as well as any continuing contamination or contagious risk to the facility and personnel. Where detection capability is limited on scene any positive or important negative result should be reported back to the scene especially where there is continuing casualty management taking place.

Table–13-1 – Smells Associated with Some Chemical Agents.

Chemical agent	Possible smells
Chlorine	Swimming pool, household bleach
Hydrogen cyanide	Bitter almonds
Hydrogen sulphide	Bad eggs
Lewisite	Geraniums
Phosgene	Freshly mown hay
Sulphur mustard	Garlic, horseradish
Some agents that are odourless in pure form: sarin, VX	

Smells may vary due to concentration, olfactory toxicity and the presence of impurities such as solvents and precursors.

CLASS	SYMBOL	DEFINITION
1		**EXPLOSIVE** Subgroups 1.1 – 1.6 1.1 – Mass explosion hazard 1.4 – No significant hazard
2		**GASES** Subgroup 2.1 – 2.3 2.1 – Flammable 2.2 – Non-flammable 2.3 – Toxic
3		**FLAMMABLE LIQUIDS**
4		**FLAMMABLE SOLIDS** Subgroups 4.1 – 4.3 4.1 – Flammable solid 4.2 – Spontaneous combustion risk 4.3 – Release of flammable gas on contact with water
5		**OXIDISERS (5.1)** **ORGANIC PEROXIDES (5.2)**
6		**TOXIC (6.1)** **INFECTIOUS SUBSTANCES (6.2)**
7		**RADIOACTIVE SUBSTANCES**
8		**CORROSIVE SUBSTANCES** (includes chlorine, sodium hydroxide, sulphuric acid)
9		**MISCELLANEOUS** Includes asbestos, CS spray

Figure 13-1: International Hazard Warning Diamonds.

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CHAPTER 14: TRIAGE

14.1. INTRODUCTION

1. For any major incident including CBRN with casualties, there is a requirement to allocate resources on a priority basis as resources are outweighed by demand. The allocation of resources is based on the sorting of casualties so that the greatest good is provided to the greatest number of casualties. There are a number of triage methods both for conventional and CBRN triage.

2. Triage is a continuous and dynamic process used at key chokepoints either for a medical intervention or transport. Triage may be repeated if there is a delay in medical evacuation as casualties may improve or deteriorate.

3. During the initial stage of a CBRN incident with multiple casualties, a simple and generic system such as an algorithm is likely to be used. As the incident develops and the type of exposure and resource requirements are identified a more sophisticated system including the use of computer based triage tool or the development of incident specific criteria can be implemented.

14.2. TRIAGE CATEGORIES

1. CBRN incidents use the same categories as conventional incident to ensure interoperability and ensure trauma, CBRN and combined casualties have the same casualty flow priorities. The categories as summaries in Table 14-1 are[1]:

a. T1 (highest category) – immediate

b. T2 – delayed

c. T3 – minimal

d. T4 – expectant (used during mass casualty (MASCAL) incidents only)

e. Dead

Table 14-1: Guidance and Comparison on CBRN Triage Categories.

CATEGORY	TRAUMA	CHEMICAL	RADIATION*	BIOLOGICAL	COMMENTS
T1	Immediate	Severe	Severe (>2 Gy & combined with other factors e.g. trauma)	Severe sepsis or septic shock	Red label
T2	Delayed	Moderate	Moderate (>2 Gy or 0.75-2 Gy combined)	Moderate (Sepsis)	Yellow label
T3	Minimal	Mild	Mild (<0.75 Gy)	Mild (Infection)	Green label
T4	Expectant	Expectant	Expectant	Expectant	Blue label (varies)

Chemical criteria for triage will either be generic in the initial stages of triage or agent specific once the agent is identified.
*The assessment of radiation dose will be based upon multiple parametric assessment including physical dosimetry and biodosimetry such as presence and onset of prodromal symptoms and biomarkers (see Chapters 33 and 35).

[1] Depending on the response zone and nation, the term delayed is sometimes applied to T2 and T3 triage categories.

14.2.1. T1 CATEGORY

1. This is the highest priority and identifies a casualty that requires immediate life-saving intervention or continuing care. The criteria vary depending on the triage system used and can be based on physiological parameters, specific indication or the requirement or application of a specific intervention such as a tourniquet. Criteria include:

a. Catastrophic haemorrhage (or tourniquet applied).[2]

b. Respiratory rate > 30 per minute.

c. Bradycardia < 40 beats per minute.

d. Tachycardia.

e. Revised Trauma Score (RTS) of 1-10[3].

f. Seizures.

g. Airway obstruction, reversed by airway manoeuvre

14.2.2. T2 CATEGORY

1. This is a priority used to define casualties that are not T1 but are usually not walking. Mentally incapacitated casualty may in certain circumstances also be classified as T2, this is appropriate for casualty decontamination where the safest option would be stretcher decontamination.

2. Criteria include:

a. Not walking but not requiring immediate life-saving treatment.

b. Walking but incapacitated (not obeying commands, loss of vision).

c. RTS of 11.

d. CBRN or clinical features that suggest early deterioration such as biological signs of sepsis or predicted radiation dose.

3. Although life-saving interventions may not be required before or during decontamination, treatment that may prevent deterioration such as antidote administration or antibiotics would be reasonable as part of EMT, especially if there is a delay in transport and not to the detriment of T1 casualties.

14.2.3. T3 CATEGORY

This is lowest triage priority and is generally used for ambulatory (walking) casualties that are not incapacitated (i.e. obeying command and not requiring escort due to impaired vision).

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[2] ☑ *Lesson learnt*. On exercises, it has been observed that once casualties have a tourniquet in place and bleeding has stopped the triage category T2 is sometimes incorrectly applied. The application of tourniquet requires urgent reassessment at a medical facilities and the casualty should remain T1.

[3] This is a score derived from Glasgow Coma Score (GCS), respiratory rate and systolic blood pressure (BP).

14.2.4. T4 EXPECTANT CATEGORY

1. Even with additional resources, some incidents may remain uncompensated. If the supporting infrastructure is also compromised (compound incident) or extreme numbers of casualties, these incidents are often described as catastrophic or MASCAL (mass casualties)[4]. Where there are inadequate resources, the expectant (T4) triage category may be authorised by the Senior Medical Commander. Casualties that require a disproportionate amount of resources that may be futile or to the detriment of other casualties will be treated palliatively ('comfort measures'). Expectant criteria may be difficult to define for some incidents and require a consensus by senior clinicians and in some cases an ethicist or legal advisor. Examples of cases where the expected criteria may be considered:

a. Injury severity score of 75 (or 6 in any region).[5]

b. Severe head injury.

c. Extensive and severe burns.

d. Severe sepsis with multiple organ failure.

e. Combined injury (severe torso trauma and concurrent CBRN exposure).

2. In some incidents, the expectant criteria may be more definable; for severe radiation exposures > 8Gy with medical care have less than a 50% probability of survival. Treatment is also less likely to be successful in the presence of combined injury.

3. The T4 category should not be used as an alternative for announcing a death.[6]

14.3. PROPERTIES OF A TRIAGE SYSTEM

Any triage system should be:

a. Rapid – Assessment takes less than one minute.

b. Dynamic – Assessment should be repeated to identify deterioration or improvement.

c. Consistent – The same category, if applied to different casualties with the same criteria.

d. Reproducible – The same category for the same casualty but different assessor.

e. Used to prioritise for treatment.

f. Used to prioritise for transport.

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[4] Caution is advised with military and civilian definitions of mass casualties. A civilian mass casualty incident may not require the use of T4 due to the surge capacity of other MTF within the region.

[5] The injury severity score (ISS) is an anatomical scoring system for trauma and is used to assess survivability retrospectively.

[6] ☑ *Lesson Learnt*. The use of T4 by on scene responders has been seen on deployed operations and also on exercises (Exercise Clean Care 16). The latter resulted in extra resources allocated to respond to a casualty that had already died.

g. Used to prioritise for decontamination, as required. This final criterion is important as a separate triage system may be used for the choke point before decontamination and gross or liquid contamination may be an additional triage criteria used to upgrade to the category to a more urgent one.

14.4. TYPES OF TRIAGE SYSTEM

1. Triage systems vary between CBRN incident zones, types of CBRN agents and nations. However, most systems are based on algorithms, scoring systems using physiological parameters or agent specific criteria. All systems have advantages and disadvantages and they are listed in Table 14-2.

2. For certain types of CBRN agents there are specific triage criteria, each of these will be given in Parts 3-5 of this publication in the relevant chapter.

Table 14-2: Triage Systems Advantages and Disadvantages.

TRIAGE METHOD	ADVANTAGE	DISADVANTAGES
Algorithm	Easy to use Generic for all hazard including trauma Rapid No diagnostic equipment usually required	Non-specific Increased risk of over-triage
Scoring system	More specific to vital signs Graded due to scores (reduced over-triage) Minimal amendments for chemical criteria Easy to use with aide-memoir or documentation	Diagnostic equipment required if using BP Risk of cross-contamination from equipment Calculations required Not rapid (BP measurement and calculations)
Agent specific	Very specific with indications for MedCM Optimised treatment based on diagnosis, prioritisation and treatment	Requires correct diagnosis Training required Difficult to remember Comprehensive list required Documentation required as aide-memoir

14.5. RECOGNITION OF DEATH IN A CBRN ENVIRONMENT

1. The level of recognition of death depends on the responder and national legal frameworks. In a CBRN environment, death may be:

a. *Declared* by any responder based on injuries incompatible with life and stigmata of death such as rigor mortis;

b. *Diagnosed* by any trained medical personnel based on the absence of life-signed and may include diagnostic equipment such as heart monitor; or

c. *Confirmed* by any registered medical practitioner / doctor (or other regulated person depending on nation).

2. For trauma triage it is generally accepted that death is defined as respiratory arrest implying cardiac arrest. For CBRN incidents, and in particular chemical incidents the recognition of death is more difficult due to individual protective equipment reducing medical

dexterity and casualty assessment, reduced signs of life in the presence of viability, and a respiratory arrest preceding cardiac arrest, this differs from the usual model of trauma death.[7]

3. For chemical casualties where resources allow and in the absence of the confirmation of cardiac arrest, resuscitation should initially be attempted but limited to basic airway management, ventilation and immediate MedCM administration.

4. For CBRN incidents, where resources are finite an unwitnessed respiratory arrest may be the only appropriate criteria for declaring a casualty dead, if further resuscitation would be detrimental to other casualties. Where available, this may be augmented by an objective assessment of cardiac electrical activity with diagnostic equipment such as simple heart monitoring confirming the absence of electrical activity (asystole).

5. Deaths, as for conventional casualties, should be labelled as part of the recognition process. The minimum recommended information is *time*, *location* and *individual recognising death*.[8] Further details of the management of fatalities can be found in Chapter 16 and the supporting Annex 16A, as well as AMedP-7.2.

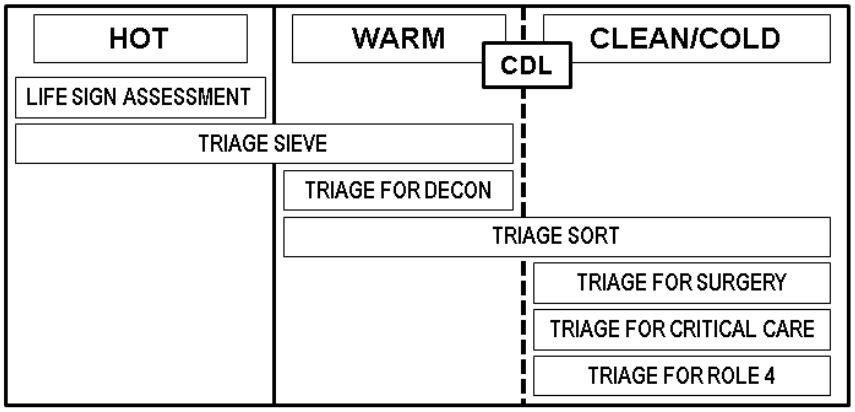

Figure 14-1: Triage Systems and CBRN Zones.

14.6. CBRN TRIAGE POINTS IN THE MEDICAL EVACUATION CHAIN

1. The method of triage system used will depend on the CBRN incident zone, accessibility to the casualty and safety factors. For NATO operations, a standardised approach with similar triage systems is recommended from point of exposure through to transport to Role 4, but especially at choke points such as decontamination, admission to intensive care and surgery.

2. The levels of triage are:

   a. Life sign assessment.

   b. Triage sieve (algorithm based) – see Figure 14-2.

   c. Triage for decontamination (algorithm based plus contamination burden).

[7] ☑ *Lesson learnt*. 25% of simulated casualties with shallow breathing were assessment as dead while responders were wearing IPE.
[8] ☑ *Lesson learnt*. The labelling of dead by the first person to triage the casualty has been a lesson identified following a number of conventional incidents and exercises.

d. Triage sort (scoring system using physiological parameters).

e. Triage for surgery (extended triage sort, clinical investigations and surgical opinion).

f. Triage for critical care (extended triage sort and end organ function).

g. Triage for Role 4 management, including replacement therapy.

3. Following a CBRN incident with multiple casualties, triage criteria will need to be defined across the medical support infrastructure and between MTF to ensure standardisation and consistency, and ensures a uniform distribution of casualties with the same medical status.

4. During an incident triage may also be applied to non-incident patients. This is because all patients use the same finite medical resources and therefore triage criteria for common care pathways such as critical care may need to reflect this[9].

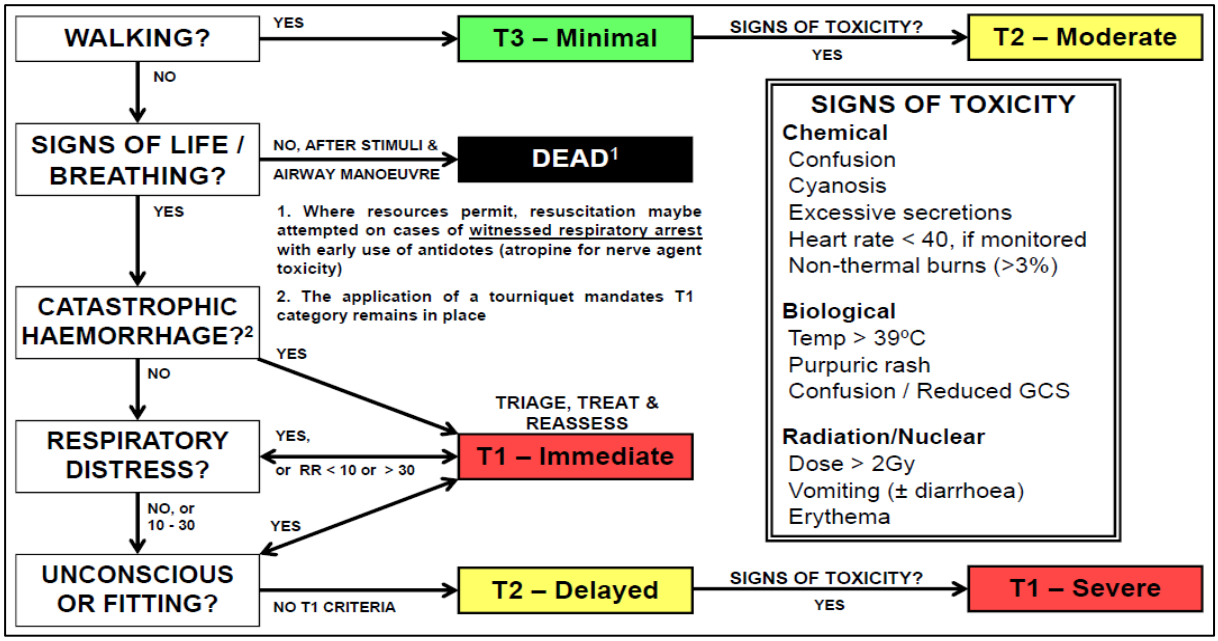

Figure 14-2: Example of a Combined CBRN Triage Sieve.[10]

[9] For critical care admissions the use of organ function scoring has been suggested such as the SOFA or APACHE scoring. These are most likely to be used for biological incidents either deliberate, endemic or emerging, including pandemic influenza.

[10] ☑ *Lesson learnt*. In austere environments with limited medical resources, the triage of a T1 casualty should be followed by initial treatment ('triage and treat') including basic life-saving interventions and excluding other life-threatening conditions, as shown in the algorithm, before moving to the next casualty.

CHAPTER 15: TRANSPORT

15.1. INTRODUCTION

1. A CBRN incident will be a challenge for transportation of casualties and in particular elements of the casualty (CASEVAC) and medical evacuation (MEDEVAC) chain. Factors include:

a. Large number of casualties requiring transport to a CDA and/or MTF.

b. Obvious presence of gross contamination and requirement to prevent further spread.

c. Short clinical timelines, similar to trauma casualties, for acute chemical and septic casualties.

d. Restricted patient access during transport due to PPE and isolation.

e. Specialist MEDEVAC equipment and training requirements.

f. Presence of contaminated personnel including casualties and responders, and equipment.

g. Finite transportation resources.

h. Decontamination requirements of the vehicle after the transportation of any contaminated casualties.

i. Restricted access to incident scene due to contamination, including downwind hazards.

j. Transportation of contagious casualties, including maintenance of negative pressure isolation.

k. Maintaining logistical support to the scene and forward assets including resupply of medical equipment and MedCM.

l. Implications of local, national and international health and travel regulations.

2. There are a number of key documents that influence the transportation of CBRN casualties. These are:

a. AJMedP-2: *Allied Joint Doctrine for Medical Evacuation*.

b. AAMedP-1.1: Chapter 7: *Management of chemically contaminated or highly infectious casualties* provides some guidance on the movement of contaminated or contagious casualties.

c. International Health Regulations (IHR) 2005.

3. MEDEVAC describes the planned movement of casualties by medical assets including the Immediate Response Team (IRT) and Blue-Light Matrix (BLM)[1]. Casualty Evacuation describes the unplanned movements of casualties without medical support or opportunistic

[1] This term is sometimes used to refer to on-site / base emergency services.

movements by medical personnel available. AJMedP-2 describes the three main categories of MEDEVAC:

a. Forward (FWD) MEDEVAC – Point of wounding (exposure) to the initial MTF, using by ground or rotary wing assets.

b. Tactical (TAC) MEDEVAC – Between MTFs within the Joint Operational Area (JOA), using ground, rotary or fixed wing assets.

c. Strategic (STRAT) MEDEVAC – JOA to home or allied nation, often using military fixed wing or civilian charter aircraft.

15.2. CBRN ZONES AND TRANSPORT

1. The same categories of MEDEVAC described above apply to a CBRN incident; the type of CBRN zone (hot, warm and clean) will restrict some of conventional transportation methods, or require COLPRO capable vehicles or for crew to wear IPE.

2. A summary of the transportation of CBRN casualties is in Figure 15-1.

15.2.1. TRANSPORTATION IN THE HOT ZONE

1. Transport in the hot zone will be limited by the risk of primary contamination of the vehicle and/or the crew, and the physical protection requirements. Walking casualties (T3) will be expected to self-extricate from the point of exposure and should go to a safe location using the 6Cs principles (see Chapter 9). The main medical transport priority within this zone is the rapid evacuation or rescue of non-ambulatory casualties to a more permissive Forward Casualty Collection Point (Fwd CCP) at the hot and warm zone interface. Medical management is limited to casualty assessment (Quick Look), first aid of non-ambulatory casualties (T1/T2) and the administration of LSI (T1 casualties only).

2. Casualty movement in this zone is most likely to be on foot, by stretcher or ground transportation with appropriate physical protection. The initial transportation of CBRN casualties will rely on CASEVAC resources by non-medical personnel. FWD MEDEVAC assets including responders should be assembled at the Fwd CCP with the following roles:

a. Deploy medical personnel forward into the hot zone as required and appropriate to do so.

b. Triage and treat casualties at the Fwd CCP.

c. MEDEVAC non-contaminated casualties direct to Role 1 MTF.

d. In extreme circumstances, MEDEVAC contaminated casualties to a CDA, Role 1 or deployed hospital with CDA. As a minimum, immediate decontamination and removal of clothing should reduce the contamination burden until full casualty decontamination can be undertaken.

e. Some casualties may be intolerant of IPE due to airway or breathing compromise, and the provision of casualty protective equipment (CPE) should be provided (see AMedP-7.2 for more details).

15.2.2. TRANSPORT IN THE WARM ZONE

1. Transport in the warm zone up to the CDL will take place after:

a. Stabilisation or administration of any LSI to T1 casualties at the Fwd CCP, CDA and Role 1 MTF.

b. IV or IO access has been established for any LSI treatment during transit such as antidotes or fluid boluses.

c. Decontamination of all stabilised T1 casualties (minimum of clothing removal) including wounds, if possible.

d. Decontamination of all T2 and T3 casualties including wounds, if possible.

e. Wounds should be decontaminated at an appropriate location with medical personnel present and ensuring haemostasis e.g. CDA. After wound decontamination, wounds should be dressed and marked as either contaminated, partially decontaminated or fully decontaminated.

2. Ground MEDEVAC of a T1 casualty may be considered before decontamination if there is no decontamination facility available or a significant delay is likely to occur. The ground asset used will be designated as a 'dirty ambulance' and may be used to ferry other contaminated casualties and medical personnel thereafter.

15.2.3. TRANSPORT BEYOND THE CDL (CLEAN ZONE).

Full casualty decontamination will allow TAC MEDEVAC by ground or air assets, but with the following constraints:

a. Casualties may still produce biological hazards due to body fluids including blood, vomit, urine and faeces.

b. Wound contamination may still be present and require surgical debridement. Wounds should be dressed and identified as a continuing hazard. If radiological shrapnel is present, any significant dose rate should be quantified and mitigated by shielding or time/distance limitations.

c. Casualties exposed to sulphur mustard may still continue to off-gas after decontamination. Casualties should be managed in well-ventilated areas or use vapour scavenging systems, where available.

d. Casualties with contagious disease will be a continuing hazard to MEDEVAC personnel and appropriate PPE must be worn.

15.3. CBRN CASUALTY PROTECTIVE EQUIPMENT

1. CBRN casualty protective equipment (CPE) is a suite of materiel to provide physical protection against CBRN agents for casualties insufficiently protected by IPE or COLPRO which permits the continued provision of casualty care in a CBRN contaminated environment or to a CBRN contaminated or contagious casualty in a clean environment. Futher details are found in Annex 15A.

15.4. AEROMEDICAL EVACUATION OF CBRN CASUALTIES

1. AAMedP-1.1[2] Chapter 7 provides guidance for the management of chemically contaminated or highly infectious casualties during AEROMED where there is a known or suspected hazard. For radiological incidents, casualties that have been solely irradiated and have not been contaminated do not require a specific casualty hazard management. For biological incidents where the suspected or confirmed agent is non-transmissible, only standard precautions that are generic to all patient interactions are required.

2. The final decision to fly an aeromed mission with potential CBRN casualties on board is made by the captain of the flight (pilot in command).

3. For some exposures with delayed effects, the CBRN presentation may only be recognised by medical personnel during MEDEVAC. In these circumstances, the following guidance is suggested:

a. Don a respirator, or if there is a particulate or biological hazard then a high specification face mask and eye protection.

b. Use single use gloves. Change the outer pair of gloves at least every 15 minutes or between patients whichever is sooner. Use of two layers allows for sacrificial layer during doffing procedure.

c. If non chemical hazard, wrap casualty in blanket to reduce aerosolisation of hazard. For chemical agents, there is a risk that a blanket may increase skin absorption and the use of a blanket should be risk assessed against the availability and use of a respirator by crew and medical personnel.

d. For casualties suspected of having a contagious illness with a droplet or airborne hazard, place a surgical or non-valved filter mask on the casualty as tolerated. Septic casualties or those with significant respiratory compromise (hypoxia) should be treated with supplemental oxygen which will restrict the use of a protective mask.

e. For ground and open cabin air assets such as helicopters, move casualties to the aft end of the compartment or cabin and maintain forward motion in order to maintain a flow of air, with open windows and doors as appropriate, to reduce cockpit and cabin concentration levels.

f. For ground and open cabin air assets such as helicopters, assume a droplet hazard is now an aerosol (airborne) hazard.

g. Notify the receiving medical facilities as soon as possible so that they can determine their CBRN response and resource requirement.

15.4.1. AEROMED OF CONTAGIOUS CASUALTIES

1. AEROMED of contagious biological casualties whether known, suspected or discovered post-flight have a number of specific considerations. During fixed wing flights within a pressurised cabin there is an increased risk of secondary transmission within the recycled air unless isolation is maintained, usually using negative pressure. For low risk agents (low

[2] AAMedP-1.1 (STANAG 3204): *Aeromedical Evacuation.*

virulence, crew immunity) standard precautions and appropriate respiratory precautions may be used.

2. Where a transmissible biological agent is suspected or known the following should be considered:

a. Number of casualties and severity.

b. Medical personnel requirement, specialisation (infection prevention and control, infectious disease, intensive care) and seniority.

c. Risk to other patients, aircrew and medical crew (consider immune status and suitability of MedCM for the tasked crew).

d. Physical protective measures such as isolation while maintaining access to the patient to safely provide clinical care.

e. Risk to planned and emergency en route and destination populations (disease suspected and population immunity).

f. International health and travel regulations.

g. Notifying nation states overflown by AEROMED.

h. Impact on overall mission and further AEROMED capability (dedication of significant and finite resources that detract from other medical mission considerations).

i. Notification, suitability, and capacity of the receiving facility.

j. Risk of contamination of the aircraft and effect of any decontamination process.

3. On notification of a communicable disease following an AEROMED, the following recommendations are suggested:

a. Number of patients suspected of being infected.

b. Flight manifesto of passengers and crew as well as stopovers.

c. Notification of the Port Authorities of any stopovers.

d. Contact tracing depending on the incubation period of the agent and the time since the flight.

e. Implementation of post-exposure prophylaxis including vaccination and antibiotics, where available.

f. Risk communication.

15.4.2. AIR TRANSPORTABLE ISOLATOR

1. The air transportable isolator (ATI) is a specialised HEPA filtered negative-pressure isolator, usually for the transportation of a single casualty. However, the use of the ATI is resource intensive requiring a large number of aeromedical personnel and equipment.

Note. For a critically ill patient, '*treat in place*' may be a safer option with the deployment of infectious disease specialists to the patient rather than a high-risk transfer of an unstable patient and risk of a death in transit.

2. During a CBRN incident or outbreak, the use of an ATI is unlikely for the mass evacuation of casualties from the JOA and the establishment of a cohort infectious disease facility is recommended. In some circumstances, the ATI may have a significant role in the support of outbreak investigation by transporting an index case to a reach back medical facility with a high level of biosafety and security with diagnostic laboratories (biosafety level 4 (BSL-4)) able to work with high risk pathogens.

3. In some circumstances, casualties who are immunocompromised such as following a significant irradiation may require reverse barrier nursing and a transportable isolator may be used in a positive-pressure mode to protect the patient from the environment.

15.5. MEDICAL EQUIPMENT TO SUPPORT CBRN MEDICAL EVACUATION

Medical equipment is mandatory for any ground or air MEDEVAC mission. MEDEVAC during a CBRN incident will have general and specific equipment requirements. Key equipment for MEDEVAC include:

a. Spare tourniquet and pressure dressings.

b. Suction.

c. Airway management equipment appropriate to CBRN zone, skills and resources.

d. Oxygen.

e. MedCM and antidotes to maintain treatment for twice the duration of the MEDEVAC mission. Drugs are recommended to be diluted and larger syringes used to improve medical dexterity.

f. Crystalloid fluids.

g. Monitoring appropriate to CBRN zone and access to the casualty.

h. Wound dressings, in case of intravenous access being compromised and bleeding.

Note: Any medical equipment will usually need to be placed inside the MEDEVAC equipment before sealing unless it has an airlock portal.

15.6. FORWARD DEPLOYED LOGISTIC MOVEMENTS

1. Logistic resources and infrastructure may be forward deployed either as pre-event preparation or as a rapid response to an incident. Forward deployment may mitigate any delay in FWD and TAC MEDEVAC. Forward assets include:

a. Medical personnel.

b. Reconnaissance or monitoring teams.

c. Medical equipment and MedCM.

d. Infrastructure such as decontamination units and cohort wards.

e. COLPRO capable MTF.

2. In some situations, resources from Role 4 or other home nation capabilities may be deployed into the JOA due to either a large number of casualties overwhelming the STRAT MEDEVAC capability or regulatory restrictions preventing the movement of casualties with infectious disease. Resources include:

a. Infectious disease cohort unit.

b. Advanced laboratory facilities with enhanced diagnostics and biosafety.

c. Deployed triage unit for radiological casualties.

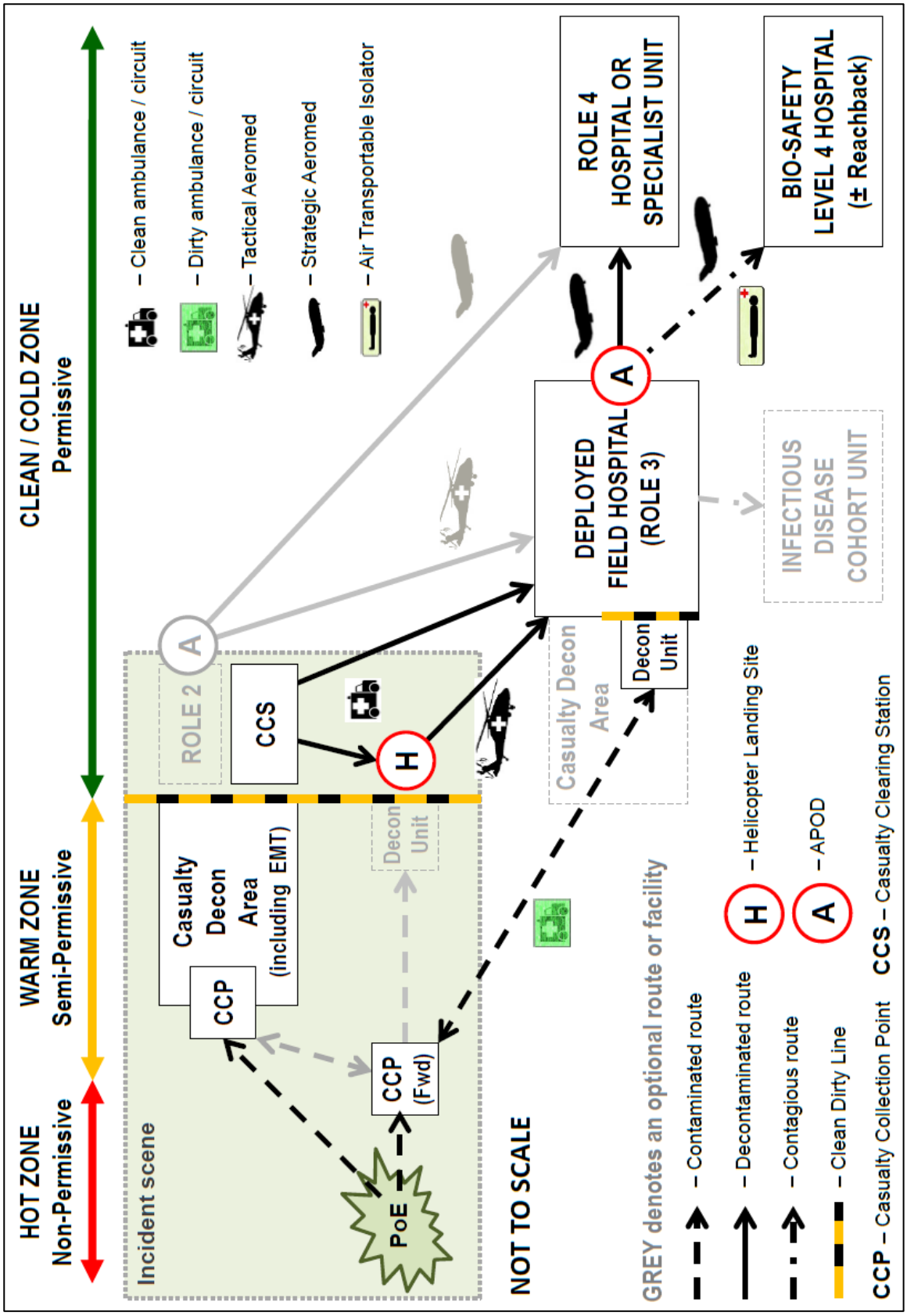

Figure 15-1: Transportation of CBRN Casualties.

ANNEX 15A – CBRN CASUALTY PROTECTIVE EQUIPMENT

15A.1. CBRN CASUALTY PROTECTIVE EQUIPMENT

1. CBRN casualty protective equipment (CPE) is a suite of materiel to provide physical protection against CBRN agents for casualties insufficiently protected by IPE or COLPRO which permits the continued provision of casualty care in a CBRN contaminated environment or to a CBRN contaminated or contagious casualty in a clean environment.

2. The main function of CBRN CPE is to either:

   a. Protect a casualty from a CBRN environment.

   b. Protect the responder and/or environment from the CBRN casualty.

   c. Protect the casualty from themselves i.e. off-gassing and carbon dioxide.

   d. A combination of the above.

3. The CBRN CPE suite includes protective equipment to support casualty care throughout the casualty evacuation chain including end of life. STRAT MEDEVAC equipment as described earlier and in other publications is used for non-CBRN medical evacuation.

15A.1.1. CBRN CASUALTY EVACUATION EQUIPMENT

1. CBRN casualty evacuation equipment provides a non-medical user the ability to provide respiratory protection for casualties for whom it is impossible or impractical to wear a respirator due to injury or illness but who require protection from inhalational CBR hazard for sufficient time to evacuate from the hazard area.

2. This equipment describes materiel for use by a non-medical user in the hot zone as part of enhanced CBRN first aid. The specific equipment capabilities may vary between nations.

3. The minimum requirement is respiratory protection with or without whole body protection. The equipment should be simple to apply over the head of a casualty with significant head or facial injury without causing further damage or pain. It should also be possible to place onto an unconscious casualty.

4. Once fitted it should be possible to continue basic first aid:

   a. Observe the casualty.

   b. Manage catastrophic haemorrhage.

   c. Perform basic airway opening procedures and removal of obstruction including suction.

   d. Administer MedCM.

5. More details are found in AMedP-7.2.

15A.1.2. CBRN WOUND DRESSING

1. CBRN wound dressing provides any user with the ability to protect wounds from CBRN agents and control haemorrhage.

2. The dressing is similar in capability and function as a standard field dressing with haemostatic control and protection of conventional wounds.

3. Following operational decontamination, some residual contamination of wounds may remain until thorough surgical debridement (removal of wound contamination and dead tissue) is undertaken.

4. The function of the CBRN wound dressing should include:

   a. Control haemorrhage.

   b. Protect responders from any secondary contamination hazard or off-gassing.

   c. Adsorb or destroy any wound contamination.

   d. Annotation to clearly identify the status of the wound: CLEAN, CHEM, BIO, RAD. If the agent and timing of exposure is known, this should also be annotated.

5. A CBRN wound dressing could also be used as IPE repair materiel.

6. More details are found in AMedP-7.2.

15A.1.3. CBRN MEDEVAC EQUIPMENT

1. CBRN medical evacuation equipment provides medical personnel with the ability to protect non-ambulatory casualties and patients for whom it is impossible or impractical to wear IPE, during transit within a CBRN environment and protects medical personnel and equipment from CBRN casualties whilst allowing the provision of emergency medical treatment.

2. CBRN MEDEVAC equipment is likely to be disposable but should allow for external decontamination after placing a patient into it. The equipment would also be medical logistic items as part of MEDEVAC capability.

3. The key capabilities include:

   a. Provide protection against chemical liquid and vapour hazards, aerosolised biological agents and radiological contamination on the operational threat list to the same standard as IPE for a minimum of 6 hours.[1]

   b. Provide containment of chemical ‘off-gas’ from a contaminated casualty

   c. Provide bio-isolation of a contagious casualty.

   d. Provide adequate air changes to mitigate any off-gassing from the casualty and maintain low CO_2 levels.

   e. Enable easy and safe insertion and removal of a casualty by handlers wearing full IPE.

   f. Enable the provision of emergency medical treatment, including observation, without breach of protective environment.

[1] This allows for loading, transport and off-loading for a 4 hour TACT MEDEVAC scenario although the specific requirements may vary between nations.

g. Enable the safe transit of a restrained casualty using in-Service stretcher, MEDEVAC platform (including rotary and fixed wing aircraft) or by using the integral properties of the system.

h. Provide filtered air at variable pressure enabling an over-pressure (positive-pressure) for CB protection and negative-pressure for containment.

i. Be man-portable by one person in packaged state and man-portable by two people unpacked.

j. Be suitably robust and resilient for operation in all climatic operations conditions.

k. Be suitable for full user and maintenance functions to be performed wearing standard and specialist IPE.

l. Allow external decontamination after a contaminated or contagious patient has been loaded into it.

15A.1.4. MOBILE ISOLATION POD

The mobile isolation pod (MIP) is a variant of CBRN MEDEVAC equipment used for FWD and TACT MEDEVAC, including rotary and fixed wing, of contagious patients. It may be CBRN MEDEVAC equipment configured for negative pressure operation and be operated within a national concept of use. The use of the MIP while possible in a CBRN incident may also be used during non-CBRN biological outbreaks where the biological agent is suspected or confirmed as being contagious with the risk of person-to-person transmission.

15A.1.5. CBRN FATALITY PROTECTIVE EQUIPMENT

This equipment type is described in Annex 16A – *Management of CBRN Fatalities*, although fatality management remains in most nations an executive and logistic capability.

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CHAPTER 16: EXPLOITATION AND RECOVERY

16.1. INTRODUCTION

1. During the response and especially as part of the recovery phase, the CBRN incident scene will need to be secured so that it can be exploited for evidence and information to inform the intelligence and forensic process. Response and recovery, both form part of the CBRN incident cycle as described in AJMedP-7 and incident recovery is a final phase in consequence management.

2. The main considerations during the incident recovery phase include:

   a. Casualty rehabilitation (see Chapter 4).

   b. Medical treatment facility and equipment hazard management.

   c. Exploitation to support intelligence and forensics.

   d. CBRN fatality management.

   e. Review and easing of restriction of movement (see Chapter 6).

   f. Risk communication especially where there is a residual hazard or risk.

   g. Medical logistics resupply.

   h. CBRN clinical waste management.

   i. Preventive (occupational) medicine support.

   j. Health surveillance (see Chapter 5 and below).

   k. After actions reporting and lesson learnt process (see below).

16.2. MEDICAL PRIORTIES FOR THE CBRN INCIDENT RECOVERY PHASE

1. Once live casualties have been cleared from the incident scene, the medical mission makes the transition from a rescue to a recovery operation. Although there may be no further primary casualties, medical services will still be required to support the recovery operation. These requirements include:

   a. The provision of advice to Command on the management and public health aspects of mass fatalities.

   b. The provision of occupational health support to responders and recovery workers.

   c. The establishment of post-incident registries and health surveillance.

2. During the recovery phase, responders should be encouraged to provide feedback in order to identify lessons and learn from them with debriefings and post-action reports. In addition, some responders may need to reflect on their own actions and the wider response as part of normal post-operational stress management. In other cases, responders and

casualties may require more formal psychological support and the recovery operation should try to identify these persons.

16.3. SCENE EXPLOITATION

Any CBRN incident especially if there are fatalities is likely to be treated as a crime scene and in some cases a war crime. From the start of the incident response, care should be taken to maintain the integrity of potential forensic evidence. Preserving the scene should not however be to the detriment of casualty care. Once the recovery operation has started, medical equipment not being used for casualty care but involved in the incident may be secured and treated as evidence; this may include personnel protective equipment, medical infrastructure and vehicles. For essential items of medical equipment and/or infrastructure such as a MTF, a risk assessment should be made by the investigating officer and a decision made by Command to release the equipment or authorise immediate replacement, as well as re-opening the MTF.

16.4. FORENSIC INVESTIGATION SUPPORT BY MEDICAL PERSONNEL

1. Medical personnel will be required to support any investigation of the incident. However, this is a complex issue and is constrained by a number of considerations including medical confidentiality, consent, Law of Armed Conflict / Geneva Convention, cultural and religious beliefs, and ethics. Potential medical-related populations and sources of evidence may include:

   a. Exposed military personnel;

   b. Exposed adult and paediatric civilians;

   c. Casualties treated in NATO MTF (military and/or civilian), but within the contraints of medical confidentiality;

   d. Fatalities; or

   e. Exposed and detained opposing forces, but within the constaints of the Laws of Armed Conflict and Geneva Convention.

2. Key areas of medical involvement include:

   a. Documentation of fatalities including timings and, in some circumstances, location and brief external examination before the body is moved.[1]

   b. Documentation of clinical samples such as blood tests that may be required for forensic investigation or confirmation of CBRN exposure such as:

      (1) Detectable agents.

      (2) Biomarkers, such as red blood cell AChE levels following nerve agent exposure.

      (3) Metabolites.

[1] The use of medical person for more invasive examinations either sampling or autopsy is on a case-by-case basis and must be within a legal, ethical and cultural framework, and with a formal agreement of the nation of the practitioner, nation of the casualty and, where required, the Host Nation. In some nations, it is illegal for non-registered medical personnel to undertake an autopsy or take post-mortem samples.

(4) Hydrolysis products.

(5) Protein and DNA adducts.

c. Documentation of medical interventions undertaken on the casualty especially if a post mortem is to take place. This allows the pathologist or medical examiner to exclude iatrogenic wounds or drug exposures.

3. Medical responders will also be required to make statements recording their actions as well as providing a narrative and scene description.[2]

16.5. MANAGEMENT OF CBRN FATALITIES

1. The management of fatalities is a Command responsibility. Medical personnel will be expected to support this task with advice and occupational medical support. CBRN incident recovery should[3]:

a. Ensure an organised multi-agency response for CBRN fatalities.

b. Identify the decedents.

c. Identify the agent causing the fatalities.

d. Ensure safe management of the fatality to minimise harm to those involved in responding to the incident.

e. Identify the best methods for transportation, storage, and disposal of the fatality so that there is little or no impact on others and the environment.

f. Ensure that the religious and cultural beliefs appropriate to the fatality are considered, where possible.

2. ATP-92: *Emergency Burial Procedures* provides guidance for the emergency temporary burial of other nations' fatalities including CBRN contaminated fatalities. However, this guidance is unlikely to be used on most NATO operations. Despite the presence of CBRN hazards, there is still likely to be a requirement for repatriation.

3. More details on the handling, transportation and final disposal of CBRN fatalities are given in Annex 16A.

16.6. PREVENTIVE (OCCUPATIONAL) MEDICINE SUPPORT

Following a CBRN incident, medical personnel will need to advise Command on Force Protection measures to be implemented to prevent or mitigate any potential exposures. This will include discussion with environmental health and occupational physicians especially if long term consequences are possible and to be avoided. These measures include:

[2] ☑ *Lesson learnt*. Following an incident it is recommended that notes or a statement are written as soon as possible after the event to support any statement being requested at a later date. In some cases, the statement or account of the incident may be required months or even years later.

[3] Baker D *et al*. Safe management of mass fatalities following chemical, biological and radiological incidents. Pre-hospital and Disaster Medicine. 2009.

a. Post-exposure prophylaxis of responders that may have been exposed during the incident. MedCM include vaccination, antibiotics or anti-radiation countermeasures.

b. PPE to limit exposure of recovery workers.

c. Monitoring of the health aspects of working in a hazardous environment and PPE including heat illness.

d. Advice and monitoring hazard management measures such as limiting working times.

e. Pre-exposure prophylaxis of recovery workers to CBRN and other occupational exposures including hepatitis A and B.

f. Recording any MedCM use and potential exposures.

16.7. HEALTH SURVEILLANCE AND REGISTERS

1. Following any potential hazard exposure, appropriate health surveillance should be started in order to:

a. Identify any delayed or long term health effects including mental health.

b. Communicate rapidly between exposed persons and health organisations.

c. If there are no observed health effects, reassure the exposed population.

d. Mitigate any delay health effects as they are identified in other members of the cohort.

e. Comply with potential health and occupational regulations.

f. Record any confirmed or potential exposure for both casualties and responders.

2. The establishment of a health register may be based upon a cohort of the population that share the same potential or confirmed exposure to a hazard substance or environment, or the same symptoms, syndromes or specific illness. Members of the register are either casualties that have been entered onto the register by the receiving MTF or other potentially exposed persons that may need to volunteer to the added to the register. Commanders may also be obliged to add personnel to the register but this will be dependent upon confidentiality issues and date protection. The recommended minimum dataset for a health register is:

a. Unique register entry number.

b. Name.

c. Date of birth.

d. Unique personal identifying number[4].

e. Date, time and duration of potential exposure.

[4] This may be a military service number, social security number or identity card number. For operations or incidents that involve multiple nations, a prefix identifying the nation should be used.

f. Location of potential exposure.

g. Any PPE worn.

h. Any pre-exposure MedCM used.

i. Any post-exposure MedCM used.

j. Any personal detection information such as local concentrations, radiation dose rate or accumulated dose.

k. Any initial (immediate or acute) symptoms or signs, using the syndromic approach.

l. Any clinical investigations taken and results where known.

m. Contact details.

3. During any casualty management, consent should be sought from the casualty for basic details, anonymised appropriately, to be used to establish the register and allow follow-up. Depending upon the circumstance this may be voluntary; while in others it will be a legislative requirement such as occupational or radiological exposures.

16.8. DEBRIEFING AND LESSONS IDENTIFIED

1. Immediate after the response, it is important to conduct an operational debrief. This is a short practical session to record issues that may be missed or forgotten after the event. Debriefing should not be focused on individual actions but is not a forum for attributing blame if shortfalls in the response are identified. The objectives for the debrief are:

a. Check that all personnel are safe and their location is known.

b. Establish a narrative of events.

c. Check all critical equipment has been returned or is being recovered.

d. Identify any key deficiencies preventing recovery to full operational capability.

e. Identify any resupply requirements.

f. Identify examples of good practice (“What went well?”).

g. Identify any areas for improvement or capability gaps (“What could be done better?”).

2. All personnel should be encouraged to maintain records and write statements. An after-action report (AAR) should be written using the same format as for conventional incidents.

3. Lessons should be identified and submitted so that they can be learnt from and any subsequent response improved upon based on best practice.

16.9. AFTERCARE OF RESPONDERS

1. As soon as possible as being stood down, all responders including medical personnel should be checked to ensure there are no injuries or acute effects to any potential hazardous exposure. In addition, all responders’ details should be logged and kept in case of health follow-up and any likely investigations into the incident and its response. Where there is a known

hazard, responders should be given advice on health features to look out for and where necessary primary or preventive health care informed.

2. Following any significant incident and especially where there may have been multiple casualties and/or CBRN exposure, stress is likely to have a number of effects on individual and this is to be expected. The management of psychological stress should be managed by the team in a supportive way that initially is independent of any medical interventions. Many post incident stress management systems use a series of escalating interventions.

a. Voluntary team debriefings.

b. Unit commander risk assessment of individual.

c. Unit level counselling.

d. Referral for formal counselling.

e. Formal medical referral and interventions.

16.10. MENTAL HEALTH SUPPORT

1. Any CBRN event is likely to have a psychological impact and described in Part One of this publication. AMedP-1.10: *Forward Mental Support* provides guidance on any post-incident event follow-up. This may be for casualties, exposed persons and responders. Psychological support may be delivered at a unit level, by an intervention team or more formal medical intervention.

2. Throughout the recovery stage, it is vital that those involved in post-incident risk communication and counselling are informed and provide a coherent message. This includes:

a. Exposure risk.

b. Initial and appropriate psychological response.

c. Direct (due to agent) and indirect (psychological response) long term effects.

ANNEX 16A – MANAGEMENT OF CBRN FATALITIES

16A.1. HAZARDS FROM CBRN CONTAMINATED OR CONTAGIOUS BODIES

Conventional mass fatalities do not themselves cause epidemics but do require appropriate management to limit disease as well as support the legal, cultural and religious process, as appropriate.[1] CBRN casualties do have some specific hazard management issues including:

a. Contamination of bodies or body bags.

b. Contamination of personal possessions.

c. Presence of transmissible biological agent and aerosolisation during putrefaction.

d. Potential infection from non-transmissible biological agents such as anthrax due to spore formation.

e. Presence and non-destruction of radioisotopes by conventional body handling methods.

f. Presence of toxic metal compounds such as arsenic.

g. Management of a body contained in a chemical resistant bag or casket.

h. Risk assessment for requirement for post-mortem examination.

i. PPE requirement for post-mortem examination.

16A.2. PRIORITIES FOR THE MANAGEMENT OF CBRN FATALITIES

1. The priorities for CBRN fatality management are:

a. Safety of personnel handing contaminated or infected bodies within safe occupational limits, including those performing any post mortem examination.

b. Identification of the decedent(s) and determination of the cause of death.

c. Collection of evidence to support the forensic process in accordance with specific national regulations.

d. Safe and dignified disposal (e.g. burial or cremation) of contaminated or infected remains. This may include the requirement for repatriation of the decedent to their home nation under media scrutiny.

e. Preventing or minimising the spread of any contamination or infectious material.

2. The general process for the management of CBRN fatalities is in Figure 16A-1 but may vary between nations.

3. The responsibilities and process for the management of CBRN fatalities remain the same as conventional fatalities with the additional requirement of reporting and mitigation of the CBRN risk. For areas of legal uncertainty, advice through the Legal Advisor (LEGAD)

[1] Pan American Health Organisation/World Health Organisation. Management of dead bodies after disasters: A field manual for first responders (2006).

should be sought in conjunction with the responsible legal authority of the nation(s) of the decedent, and the host nation.[2]

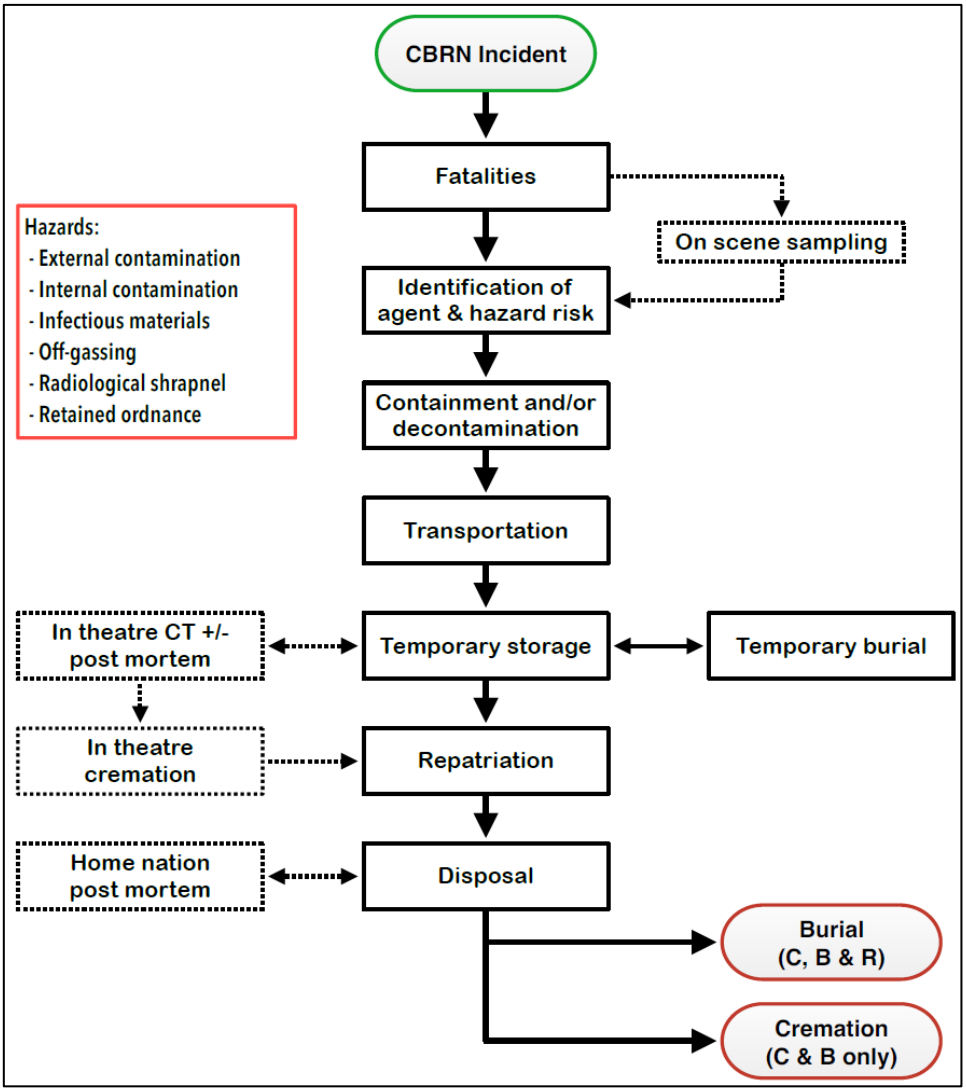


Figure 16A-1: Summary of CBRN Fatality Management.[3]

16A.3. SAFE HANDLING OF CBRN FATALITIES

1. The safe handling of decedents of a CBRN incident requires an understanding of the type of hazard. This hazards include:

   a. External contamination.

   b. Internal contamination.

[2] This depends on the Status of Forces Agreement (SOFA) between the host nation and an allied nation with military personnel deployed to that nation.
[3] On scene sampling and forensic investigations will be in accordance with specific national regulations.

c. Infectious materials including the underlying disease with high bacterial or viral load, spore formation and effluent due to early putrefaction.

d. Off-gassing.

e. Radiological shrapnel.

f. Retained ordnance.

g. Conventional hazards associated with body handling including blood borne viruses and psychological stress.

2. The management of these hazards can be in a number of ways and depends on the hazard type, number of bodies, immediate management including the requirement for a post mortem, temporary storage or burial, repatriation capability, and religious or cultural beliefs.

3. The initial safe management is focused on decontamination and containment. Considerations include:[4]

a. Identification of any hazard present.

b. External hazard labelling.

c. Removal of contaminated clothing.

d. External decontamination of the body.

e. Dose rate assessment of any radioactive shrapnel and, where possible, removal.

f. Individual safe containment (fatality protective equipment e.g. human remains bag).

g. Collective safe temporary storage (temporary or adapted mortuary).

h. Temporary burial.

16A.4. TEMPORARY HOLDING / MORTUARY FOR CBRN FATALITIES

1. The temporary holding for CBRN fatalities required a specialist holding capability to manage any CBRN hazard and surge capacity. The holding capability includes:

a. *On scene*. This time should be minimised as much as possible but may be delayed due to risk assessment, priority given to the rescue of live casualties and continuing enemy action.

b. *Body holding area*. This is a basic area to hold bodies before decontamination or any formal processing. The area should be discrete and limit any potential public or media viewing.

c. *Temporary CBRN mortuary*. This is a temporary facility to allow safe storage and processing of CBRN casualties. The processing at a temporary mortuary includes

[4] Baker D *et al*. Safe management of mass fatalities following chemical, biological and radiological incidents. Pre-hospital and Disaster Medicine. 2009.

processes associated with conventional management including fingerprinting, forensic odontology, DNA sampling and, where available, CT post mortem.

2. The temporary mortuary is a scalable capability depending upon casualty estimation or initial reported numbers. The capacity may either allow the mortuary to be co-located with a MTF or may be a standalone site, self-sustained with staff accommodation and logistic support.

3. In some circumstances a number of temporary mortuaries may be established but must report through a single casualty reporting chain and accountable to a single investigative authority.

4. A temporary mortuary should have the following facilities:

a. Secure and private.

b. Discrete parking or ambulance circuit for body movement.

c. Maintain temperature between 1 to 4°C.

d. Appropriate 'clinical' flooring.

e. Appropriate ventilation or scavenging to prevent build-up of off-gassing supported by appropriate monitoring.

f. Appropriate location of storage to prevent significant radiological exposures supported by appropriate monitoring.

g. Appropriate facility to perform full post mortem and safety package samples.

h. Drainage and waste disposal.

i. Maintain a safe distance in cases of high dose rate radiological fatalities.

j. Suitable staff working environment to support administration and accommodation.

5. The processes either on scene, at the body holding area or temporary mortuary include the following:

a. On scene:

(1) Assess CBRN risk.

(2) Assess PPE and other protective measures for recovery operations.

(3) Locating bodies and human remains.

(4) Numbering and photographing, as safe to do so.

(5) Recovery of bodies and human remains using CBRN fatality protective equipment, where available, and documenting any risks and evidential requirements.

(6) Transfer to body holding area.

b. Body handling area.

(1) Maintain log of bodies and other remains.

(2) Maintain forensic standards as well as privacy.

(3) If not already contained, place body or remains into protective equipment i.e. human remains pouch,

(4) Consider location of any external decontamination balanced against maintaining forensic integrity. This is a senior decision and can only be made after taking legal and medical advice on the risks and benefits and impact on the forensic process. Consider hair, skin and nail sampling.

(5) Document any moves and thereby the chain of custody (evidence).

c. Temporary mortuary.

(1) Reception of bodies and human remains, including numbering, tagging and photography.

(2) Radiography – plain and/or CT.

(3) If not done before, formal removal of clothing, formal external assessment, additional photography, hair, skin and nail sampling (as appropriate) and external decontamination.

(4) Finger printing.

(5) Post mortem examination – subject to risk assessment.

(6) Odontology.

(7) Re-packaging – containment.

(8) Reconstruction and embalming – see above re: embalming risks.

(9) Operational disposal considerations: temporary / war burial, in-theatre cremation or repatriation.

16A.5. CBRN FATALITY PROTECTIVE EQUIPMENT

1. For the management of bodies following a CBRN incident, the use of chemical resistant bags may reduce potential exposure. It is unlikely that for mass CBRN casualties, repatriation back to the home nation would be allowed immediately or without appropriate hazard management of the bodies and compliance with travel and aviation regulations. Initial transportation is therefore likely to a temporary storage / mortuary area, or temporary burial subject to legal advice on the requirement for a post mortem examination.

2. The requirements for fatality protective equipment such as a human remains pouch for deaths in a CBRN environment will depend on the specific type of incident and agent involved. The generic properties of the pouch include:

a. Containment of any hazard (chemical, biological or radiological). This may require triple containers including leak-proof and potentially hermetically-sealed. Zips should be on the top.

b. Conforms to transportation regulations including air transport. This may require the use of zinc-lined coffins in order to allow security screening and provide a gas-tight seal.

c. Allowing diagnostic and security imaging including computed tomography (CT). This may be for the purposes of security screening to exclude ordnance as well as supporting any post-mortem examination.[5]

16A.6. REQUIRMENT FOR A POST MORTEM EXAMINATION

1. The requirement for post mortem examination depends on the certainty of the cause of death given the known fact of the incident. The presence of a CBRN hazard and risk to the pathologist should be considered. Post mortem investigations are not limited to an invasive and potentially hazardous procedure. Cause of death investigations include:

a. On scene information including detection, identification and monitoring information.

b. A confirmed diagnosis before death including laboratory confirmation.

c. CT post mortem – useful in the presence of trauma and to exclude retained ordnance.

d. External visual examination and photography.

e. Extremal sampling including hair, skin and nails.

f. Invasive post mortem sampling including blood and urine.

2. *Any risk or exposures to the pathologist are within occupational regulatory limits*. The only exception may be the removal of radiological shrapnel as a dose reduction intervention but only after a formal dose reduction justification has been made and authorised by the chain of command, and practitioner(s) consent given.

3. Specific hazard considerations are described below. The final decision on the requirement for a post mortem will be made by the decedent's national legal authority usually the coroner and in some cases in discussion with the next of kin.

16A.7. REPATRIATION OF CBRN FATALITIES

1. While Emergency War Burial guidance describes the temporary burial of casualties in the operational theatre, *it is highly likely that public opinion and political direction will require the repatriation of the dead despite a residual CBRN hazard.*

2. Repatriation is most likely using strategic airframes although other means such as ground and sea exist. Ground and sea repatriation may be options if there are mass fatalities or over-flying restrictions. However, they may be more costly both financially and emotionally for family having to wait for the return of loved one, while also not mitigating all risks.

3. Specific hazard considerations are described below.

16A.8. FINAL DISPOSITION OF CBRN FATALITIES

1. There are two main options for the disposal of bodies within the JOA, whether permanent or temporary.

[5] CT may provide vital information to support or exclude a likely cause of death in a CBRN casualty especially where there may be combined injuries. This will inform the ultimate decision to conduct a full post-mortem either in theatre or home nation.

a. *Burial*. The first option is burial and this is the most likely option for mass casualties. The advantage of burial is the ability to use mass graves especially for incidents of mass fatalities, significant risk of contamination or biological agent infection. The disadvantage of burial is the potential contamination of groundwater as the body decomposes, and potential accessibility of biological agents by further use.[6]

b. *Cremation*. The second option is cremation using an industry standard facility of >600°C. The advantage of this method is the destruction of all biological agents and most chemical agents although the toxic metals and radioisotopes will not be destroyed. Crematoria are not usually deployable but may be available locally within the host nation. Disadvantages of crematoria are a slow turnover and the destruction of future forensic evidence.[7] Improvised crematoria and pyres must not be used due to incomplete destruction and potential aerosolisation of hazardous material.

2. Specific hazard considerations are described below.

16A.9. AGENT SPECIFIC CONSIDERATIONS – CHEMICAL

1. After a chemical incident, the major residual hazard is external and can be removed. Most military chemical agents, especially if vapour, will evaporate or bind and be deactivated once absorbed by the body. Some persistent agents such as sulphur mustard may continue to be a hazard especially if absorbed as a concentrated liquid. Off-gassing may occur if there remains a reservoir of a volatile agent in subcutaneous tissues. Residual amounts may also remain in the nails and hair.

2. *Industrial incidents*. Following a major industrial accident, gross contamination and saturation by a toxic industrial chemical may be possible and present a significant residual hazard.

3. *Decontamination and containment*. Any clothing should be removed and the victim decontaminated externally. The body should be stored in a refrigerated unit and any collective storage area should be filtered or well-ventilated depending on the location of the mortuary.

4. *Transportation*. Ground transport is possible using appropriate chemical-resistant human remains bags in both liquid and vapour form. Air transportation must comply with international health and aviation regulations.

5. *Post mortem considerations*. Following a chemical incident, on scene assessment may have identified the causative agent especially if sampling is carried out during the event or a residue or breakdown product is left. In some cases, the body may be the only evidence especially if death is not immediate or in cases of poisoning. A full post-mortem may be required in order to assess the action of the agent on tissues and organs. In some cases, toxicology can be carried out on blood and other biological media taken during treatment or immediately post mortem. Limited sampling of the body and the transport of these samples to a reach back facility may provide sufficient information to identify the cause of death without the requirement for a hazardous full post mortem examination. This is important if there remains an off-gassing hazard although this can be mitigated by the use of PPE during the procedure but with additional risks due to PPE use. A post-mortem may still be required if

[6] For some biological agents, the use of 10% bleach has been recommended for the body and surrounding area with a minimum contact time of 10 minutes.
[7] Baker D *et al* (2009). Prehospital and disaster medicine.

there is uncertainty as to the actual cause of death either due to a combined injury or other circumstances.

6. *Disposal*. Both cremation and burial are options.

a. *Cremation*. The cremation process will destroy all chemical agents. However, toxic elements such as arsenic may still be present in the residue, although the quantity is insufficient for there to be a significant concentration in the crematorium exhaust.

b. *Burial*. Chemical agents will breakdown following burial due to hydrolysis and oxidation. Toxic elements will also remain following burial and where there is concern, any ground water should be restricted from entry by use of a metal casket.

16A.10. AGENT SPECIFIC CONSIDERATIONS – BIOLOGICAL

1. Following a biological incident, the risk depends on the type of biological hazard – live biological agent or toxin. A toxin will be managed in the same way as a chemical agent.

2. *Body handling*. In general, most bodies can be handled in the same way as other dead bodies with the exception for agents requiring strict precautions such as viral haemorrhagic fevers. In such cases, especially where there already is a diagnosis, a post mortem is not recommended.

3. *Spore formation*. Although anthrax is non-transmissible, following death the bacteria will go into a refractory state as spores. Anthrax spores present a persistent external hazard.

4. *Transportation*. Ground transport is possible using appropriate specialised human remains bags. Air transportation will have to comply with international health and aviation regulations. The body will be required to be sealed in three layers. This may include a gas-tight, hermitically sealed container with zinc or lead lining. Post mortem diagnostic samples will be treated as per other diagnostic biological samples in accordance with IATA regulations for the air transportation of dangerous goods, in this case Class B infectious materials. This requires triple packaging.

5. *Post mortem considerations*. In many biological cases, a laboratory diagnosis will have already been made before the time of death. A full post-mortem is not recommended in cases of high-risk infections (BSL 3-4) including viral haemorrhagic fevers. For biological fatalities, a post-mortem may still be required if there is uncertainty to the actual cause of death either due to a combined injury or other circumstances.

6. *Disposal*. Both cremation and burial are possible.

a. *Cremation*. All biological agents including spores will be destroyed by the cremation process, although any exhaust should go through an appropriate filter.

b. *Burial*. Burial is the most likely option during an outbreak due to numbers. While mass burials are required, mass graves should be avoided due to cultural and religious beliefs.[8] Following burial, most live biological agents are usually non-viable within weeks

[8] A lesson learnt from the West African Ebola Crisis is that a policy of ‘safe and dignified’ burials made a significant contribution to fatality hazard management and community engagement, thereby reducing further cases in those managing both patients and bodies. Simple marked individual graves are more time consuming than mass graves but the social benefit cannot be under-estimated.

with the exception of spore forming agents such as *B.anthracis*. Genetic material may still be recoverable for many years.

c. *Embalming*. Where there is a significant infection hazard, embalming should not be performed due to the infection risk and requirement for waste management of blood run-off.

16A.11. AGENT SPECIFIC CONSIDERATIONS – RADIOLOGICAL

1. Following a radiological incident, the risk of any contamination or fragment can be quantified. Contamination can be assessed by use of an appropriate contamination monitor and mitigated by removal of clothing and external decontamination. Any significant dose rate can be monitored and appropriate protective measures applied by use of time, distance and shielding.

2. *Irradiation only*. Anybody that has been irradiated only and not contaminated is not a radiological hazard to recovery and mortuary personnel.

3. *Dose rate restrictions*. The following rules apply for radiation fatality management:

a. During recovery operations, radiation protection dose limits will be based on occupational exposure levels and not emergency levels. Any deviation from these limits requires a formal justification, legal advice and command authorisation.

b. A body with a dose rate of 1 Gy (100 millirad) per hour at 25mm (one inch) from the skin should be placed in a controlled area. This should ideally be in a refrigeration unit with a minimum of ten metres (30 feet) and dose rate monitoring.

c. Where there is a high dose rate, any viewing of the body should be restricted.

d. Early burial should be considered within a religious and cultural context.

4. *Radiological fragments*. Where possible, any high dose rate fragment should be removed from the body. However the decision will be based on a dose reduction calculation compared to leaving *in situ*.

5. *Transportation*. Any ground transport is possible using appropriate specialised human remains bags. Air transportation will have to comply with international health and aviation regulations and dose rate reading will be required as part of the consignment documentation. Any high dose rate fragments must be removed.

6. *Post mortem considerations*. Unless there is a pure irradiation exposure with consistent acute radiation syndrome (ARS) history, a post mortem may be required due to other possible causes of death e.g. trauma following detonation of a dirty bomb, or sepsis and haemorrhage as part of ARS. Where there is a significant radiation risk to the pathologist and a probable cause, the legal authority of the decedent's nation may waiver a formal post mortem.

7. *Disposal*. Cremation is not permitted for fatalities with radiological contamination. If the contamination and/or fragments are removed, this can be reassessed but subject to authorisation by the national environmental regulatory body. Any burial will require additional environmental considerations to prevent ingress and contamination of ground water. This may be mitigated by use of metal caskets and a concrete vault. Where the dose rate exceeds twice background, burial in the ground is preferred to interment above the ground as is custom in

some cultures. A discrete radiation warning label should also be left in case of exhumation or future movement.

CHAPTER 17: OPERATIONAL EPIDEMIOLOGY

17.1. INTRODUCTION

1. For slowly evolving scenarios, such as biological incidents, chronic chemical or acute radiological exposures, an epidemiological approach is required to establish cause and identify appropriate control measures. Epidemiology is the study of the factors determining and influencing the frequency and distribution of disease, injury, and other health-related events and their causes in a defined human population for the purpose of establishing programs to prevent and control their development and spread. In the context of CBRN agents, there are the additional requirements of determining any deliberate intention possibly with a background of endemic disease.

2. For any outbreak or cohort of unexplained presentations, there must be three elements: *agent*, *host* and *environment*. Descriptive epidemiology studies the relationship between the three elements to help determine a causal link so that the relationship may be disrupted. Breaking the relationship may be by vaccination of the host, eradication of the agent or ensuring the agent and host are in different environments. Operational epidemiology provides a link between the descriptive (inform) components of CBRN protection (detect and information management) and the application of effective control (protective) measures (physical protection, MedCM and hazard management). This is summarised in the Figure 17-1 below.

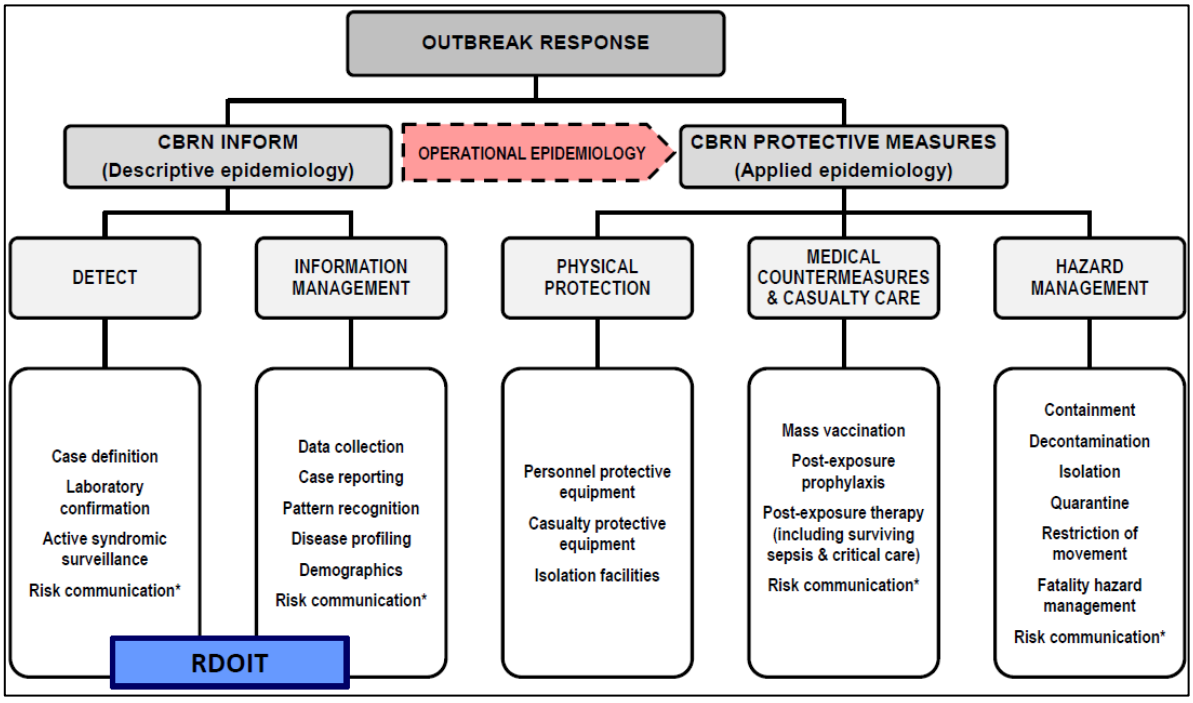


Figure 17-1: Operational Epidemiology including the Investigation Role of RDOIT.

3. The goals of the management of an outbreak or an incident with unknown cause are:

   a. Identify the source of the disease.

   b. Identify cases, treat and report.

   c. Identify control measures to prevent further cases.

d. Reducing the basic reproduction number or reproductive ratio (R_0) to less than one (see below).

17.2. OUTBREAK RECOGNITION

1. Unlike explosive and most chemical incidents with immediate or acute onset effects, biological and covert radiological incidents may not be obvious. Recognition of an event may be based on an individual diagnosis by an astute clinician or by the pattern of disease. Even if an outbreak or spike in disease is detected, the unnatural origins could be missed and deliberate intent undiscovered.

2. The recognition of a slowly evolving CBRN incident may come from a number of sources and include clinicians, infection prevention and control staff, laboratory staff and pharmacists. Veterinaries will have an important role in the identification of human diseases that involve animal vectors or suspect an unnatural cause for the human disease by the absence of the disease in the animal population. Irrespective of any malicious or deliberate intent, all medical personnel should be aware of significant diseases that present a secondary hazard to themselves including endemic disease and other occupational hazards

3. Mechanisms involved in the recognition of a slowly evolving event include:

a. Clinical diagnosis.

b. Laboratory diagnosis by microscopy with confirmatory testing (biochemistry and agglutination), serology and molecular biology.

c. Toxicology testing.

d. Forensics including post mortem and gross pathology findings.

e. Public health reporting.

f. Health surveillance.

g. Veterinary reporting of animal borne human diseases.

4. As well as specific cases, medical personnel should be aware of features that determine the epidemiological pattern of an outbreak. These include:

a. Specific agents.

b. Occurrence (incidence or prevalence) of endemic diseases.

c. Reservoir such as animal hosts.

d. Method of transmission including any vectors.

e. Transmissibility (person to person) i.e. contagious.

f. Incubation period (or latency period if toxin).

g. Susceptible population.

h. Abandoned toxic industrial hazards.

5. *Basic Reproduction Number or Reproductive Ratio (R_0)*. The severity of the effect of transmissibility is demonstrated by the initial R_0. This is the number of secondary cases a contagious patient will cause on average in an uninfected population over the infective period. R_0 is a function of the infectivity of the agent, agent stability, and its mode of transmission (e.g. contact compared to airborne) and the length of the infectivity period. Subsequent ratios will also be a function of the effective protective measures. A non-transmissible agent will have an R_0 of zero. For an outbreak to be sustained R_0 must be greater than one. Examples of R_0 include Ebola (2014) R_0 = 1.5-2.5; influenza (1918) R_0 = 2-3; smallpox R_0 = 5-7 and measles R_0 = 12-18.

17.3. PRINCIPLES OF OUTBREAK INVESTIGATION

1. During the early stages of an incident, the investigation including initial outbreak confirmation may have to be conducted by unit medical personnel. Significant outbreaks will require additional resources including public health, communicable disease specialists, and even a RDOIT with reach-back facilities. If radiation is suspected then a MRIIT may be deployed. Operational epidemiology will also be supported by other information from other information sources including environmental health surveys and animal health information.

2. The principles of outbreak investigation are:

   a. Verify the initial diagnosis; this may be a probable or confirmed diagnosis.

   b. Confirm there is an outbreak based upon current and background rates.

   c. Establish a case definition for reporting and generation of epidemiological curves.

   d. Collect case information on person, place, time, and activities.

   e. Develop the hypothesis.

   f. Implement control measures.

   g. Evaluate control measures.

   h. Collect data and evaluate the hypothesis (analytical epidemiology).

   i. Formulate conclusions.

   j. Communicate findings and risks.

3. It is important to ensure the validity of the data collected including correct timings, avoiding duplication of information and standardisation starting with an objective and clear case definition. Examples of potential errors include the use of the date of case presentation, laboratory confirmation date or date of death rather than onset date, and double reporting of clinical case and laboratory confirmation as different cases.

4. With regards to an epidemiological confirmed case this will be based on a positive case definition plus a positive laboratory test. In the context of a CBRN incident and investigation, a confirmed case requires positive laboratory results by two independent laboratories and/or methods in the presence of a positive control.

17.3.1. CASE DEFINITION

The case definition may initially be quite broad and include both clinical and laboratory criteria. Criteria should be objective and this is especially important for clinical criteria such as symptoms and signs. As more information is collected, more specific criteria including laboratory testing may be identified and the case definition refined. However, altering of the case definition should be limited as subsequent data interpretation is more complex.

17.3.2. CASE REPORTING

Once cases have been identified, addition information should be collected including:

a. Date of onset of illness.

b. Symptoms and signs, including those that are not part of the case definition.

c. Travel history.

d. Occupational history.

e. MedCM taken including full vaccination history.

f. Unwell contacts.

g. Contact with animals.

h. Treatment received.

17.4. EPIDEMIOLOGICAL CURVES

1. The presentation of cases in the form of an epidemiological curve will provide investigators with information about the characteristics of the causative agent, risk of transmission and potential method(s) of release. The curve as shown in Figure 17-2 consists of 3 main parts:

a. Incubation (latency) period.

b. Primary case peak.

c. Second case peak(s).

2. *Incubation (latency) period*. For most toxins, the *latency period* may be relatively short and are usually measured in hours. In general, live agents have a longer *incubation period* as an infection is established by live agent reproduction and overcoming the host's immune system.

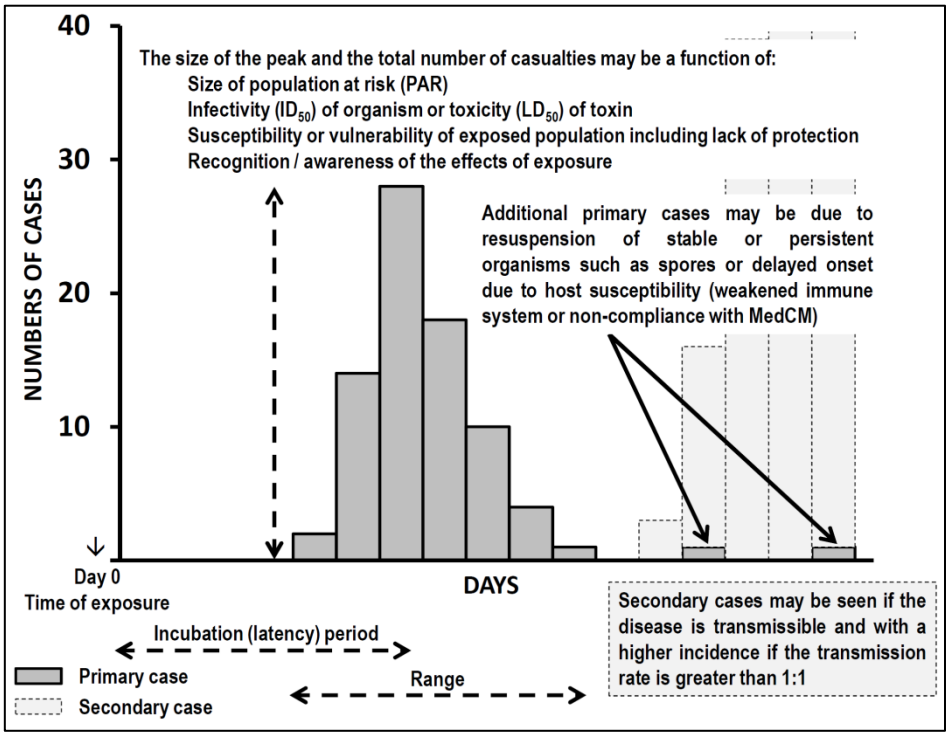


Figure 17-2: Factors Influencing the Epidemiological Curve.

3. *Primary case peak*. This is the initial peak usually starting with an *index case*. For some transmissible outbreaks, the first peak may be a single case followed by a larger secondary peak and in rare cases the index case may not be identified. The peak is around the mean incubation period and the variation is described as the range either side of the peak. While the height and slope of the peak indicates casualty surge, the size and area under the curve indicates the total number of cases. The total number of cases is a function of a number of factors including:

a. The total size of the exposed population (population at risk).

b. The infectivity of the agent (ID_{50}) or the toxicity of the toxin (LD_{50}), the smaller the value the greater the effect an agent will have on the same population.

c. The susceptibility of the exposed population (host) to the agent. This is a predisposing factor that may include lack of exposure to the agent before and no immunity, or a reduced immunity due to extremes of age. Population susceptibility may be an important factor for civilian populations. This may also be a reason of late presentation (outliers) that may appear to be secondary cases but are due to the primary exposure and a prolonged incubation period. Delayed primary cases may also be due to reintroduction of a stable or persistent agent such as anthrax spores back into the environment.

d. The vulnerabilities of the exposed populations (host). These are variable factors that alter the likelihood (plausibility) or consequence of an exposure. Examples of vulnerabilities include the lack of physical protection, non-vaccination and non-compliance with MedCM.

e. Increased awareness or recognition of the effects of exposure. While this factor does not affect the absolute number of cases, an increased reporting rate may be seen and even a surge due to casualties, clinicians and laboratories having increased knowledge of the effects of the exposure and actively looking and reporting cases.

f. Continuous exposure to the agent. This factor gives a specific epidemiological curve which is a plateau and with a sustained presentation of new cases. An example may be a contaminated food or water source. An endemic disease is a condition which has a sustained annual incidence although there maybe seasonal variation, and is said to be prevalent.

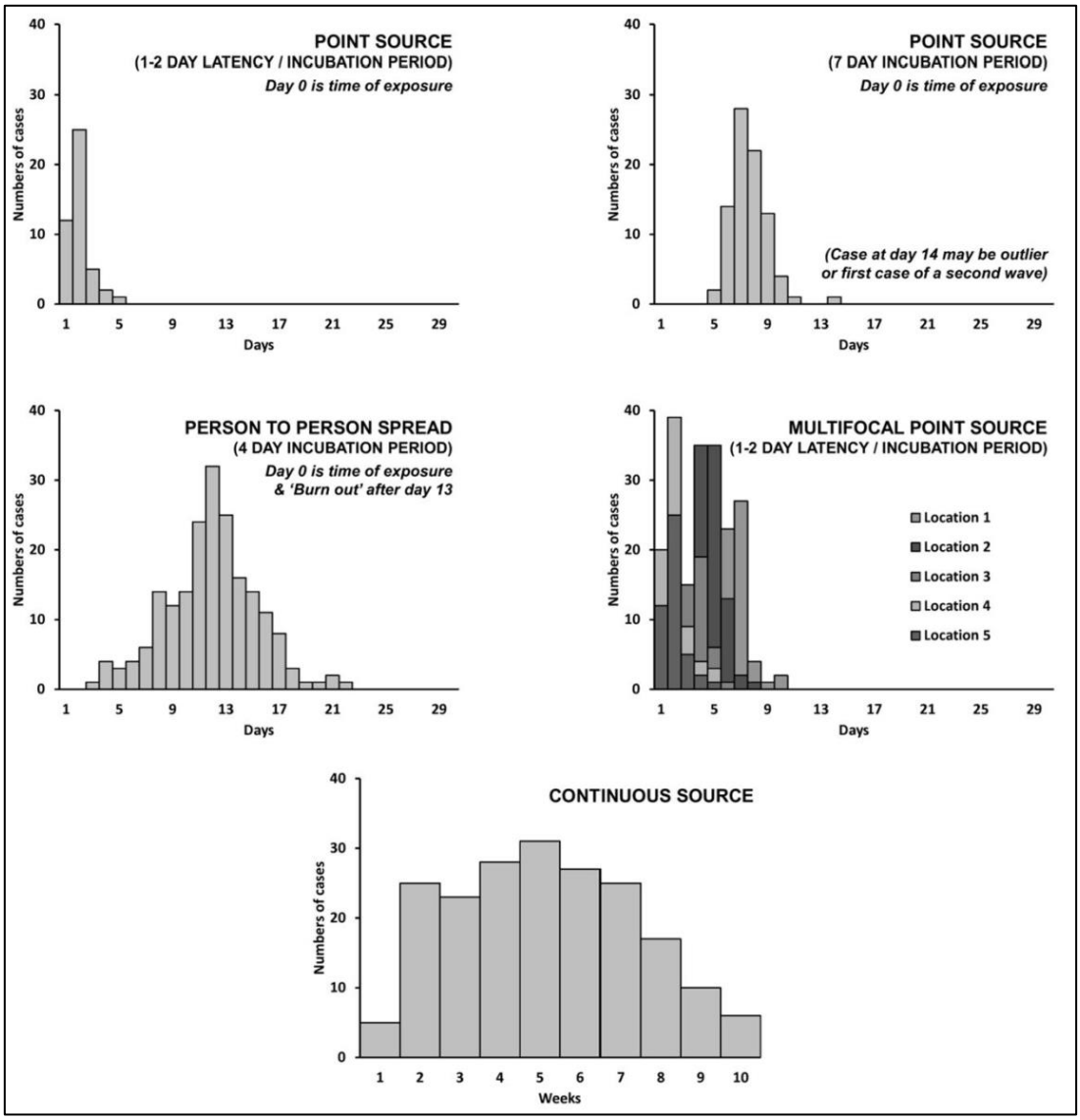


Figure 17-3: Epidemiological Curves.

4. *Secondary case peak(s)*. If there is person to person spread, further peaks following an incubation period(s) will be seen. The size of the secondary peaks depends on the factors described above for the primary peak as well as the R_0. For $R_0>1$, the secondary peak will be larger until effective control measures have been introduced or the disease has 'burnt out' due

to no further hosts being available (herd immunity) or high virulence/lethality with long incubation period preventing further transmission.

5. There are three types of epidemiological curves and examples are shown in Figure 17-3. The three curves are:

a. Point source.

b. Continuous source.

c. Person to person spread.

Note: Early recognition of the person to person pattern of disease is vital for the early implementation of control measures including mass vaccination and RoM.

17.5. INDICATORS FOR A DELIBERATE RELEASE

1. There are a number of indicators that a disease, outbreak or incident may be deliberate. These can be categorised into:

a. Event/incident indicators.

(1) A highly unusual event with large numbers of casualties.

(2) Non-endemic disease including: suspicion of smallpox-like illness, plague, tularaemia or inhalational anthrax.

(3) Uncommon disease, geographical, time or age.

(4) Event/incident includes a mass gathering event or significant persons such as military or political persons.

b. Alert and surveillance signals.

(1) Medical intelligence.

(2) Overt threat.

(3) Direct evidence.

(4) Sudden unexplained death in a previously well person.

(5) Multi-focal outbreaks without a direct link.

c. Clinical findings.

(1) Higher morbidity or mortality than expected.

(2) Unusual route of exposure.

(3) Unusual disease manifestation.

(4) Cluster of 2 or more cases of the following: neurological syndrome, respiratory syndrome, acute fulminating septicaemia or shock, fulminant hepatitis or hepatic failure, or multi-organ failure.

d. Laboratory findings.

(1) Laboratory confirmed case/cluster of specific agent with no known risk factors for natural infection.

(2) Same strain/characteristics of agent but in temporally or spatial distinct sources.

(3) Confirmed atypical, genetically engineered or antiquated strain of an agent.

e. Epidemiological findings.

(1) Point source outbreak.

(2) Downwind plume pattern.

(3) Lower attack rates in protected individuals (vaccination, IPE, MedCM).

(4) Reverse or simultaneous spread, human disease preceding or at the same time as animal disease.

17.6. NATO SPECIALIST INVESTIGATION AND RESPONSE TEAMS

As part of a NATO CBRN capability, specialist response teams may be pre-deployed or rapidly deployed to support the investigation of an outbreak or possible CBRN event. These teams may be medical or CBRN specialists. They include:

a. RDOIT (see AMedP-7.7).

b. MRIIT (see AMedP-7.4).

c. SIBCRA (see AEP-66).

d. Environmental health monitoring teams.

e. Deployable diagnostic laboratories.

2. The activation of a team will be initiated upon request by the Theatre Commander or higher-level authority through the chain of command, following medical advice. Command guidance on activation or access to specialist teams will be provided in AMedP-7.6.

3. In addition to deployable teams, NATO has a network of reach back laboratories as well as providing expert advice. This will be accessed either through the medical or CBRN chain, or via the scientific advisor (SCIAD).

17.7. INTERNATIONAL HEALTH REGULATIONS

1. Annex 5 of the IHR 2005 provides guidance on the reporting of PHEIC. While many of the likely diseases may be of natural origins, the regulations also consider unknown and unnatural disease outbreaks. The criteria for reporting any PHEIC include:

a. Any case of the following diseases:

(1) Smallpox

(2) Poliomyelitis due to wild-type poliovirus

(3) Human influenza caused by a new subtype

(4) Severe acute respiratory syndrome (SARS)

b. Any event involving the following diseases and after following the algorithm in Figure 17-4, because they have demonstrated the ability to cause serious public health impact or to spread rapidly internationally:

(1) Cholera.

(2) Pneumonic plague.

(3) Yellow fever.

(4) Viral haemorrhagic fevers (Ebola, Lassa, and Marburg).

(5) West Nile fever.

(6) Other diseases that are of special national or regional concern, e.g. dengue fever, Rift Valley fever, and meningococcal disease OR

c. Any event of potential international public health concern, including those of unknown causes or involving other events or diseases not listed above after following the algorithm in Annex 17A.

2. For the algorithm in Annex 17A each question has further supporting questions:

a. Is the public health impact of the event serious?

(1) Is the number of cases and/or number of deaths for this type of event large for the given place, time or population?

(2) Has the event the potential to have a high public health impact?

(3) Is external assistance needed to detect, investigate, respond and control the current event, or prevent new cases?

b. Is the event unusual or unexpected?

(1) Is the event unusual?

(2) Is the event unexpected from a public health perspective?

c. Is there a significant risk of international spread?

(1) Is there evidence of an epidemiological link to similar events in other States?

(2) Is there any factor that should alert us to the potential for cross border movement of the agent, vehicle or host?

d. Is there a significant risk of international travel or trade restrictions?

(1) Have similar events in the past resulted in international restriction on trade and/or travel?

(2) Is the source suspected or known to be a food product, water or any other goods that might be contaminated that has been exported/imported to/from other States?

(3) Has the event occurred in association with an international gathering or in an area of intense international tourism?

(4) Has the event caused requests for more information by foreign officials or international media?

3. Any reporting of a PHEIC under the IHR 2005 must be made through the strategic chain of command with notice given to each appropriate member nations' defence medical service and civil health service.

17.8. NON-BIOLOGICAL CAUSES FOR SLOWLY EVOLVING INCIDENTS

1. While slowly evolving events are most likely to be due to a biological agent either live or toxin, some scenarios may mimic a biological incident either by the delayed onset or associated pattern of disease or syndrome. In some cases there may be additional clues such as detection or intelligence, but in others the diagnosis is clinical. Chapter 7 provides guidance on the investigation of unusual illnesses and potential clues to the cause from initial clinical investigations.

2. Deliberate exposures that may mimic a biological incident include:

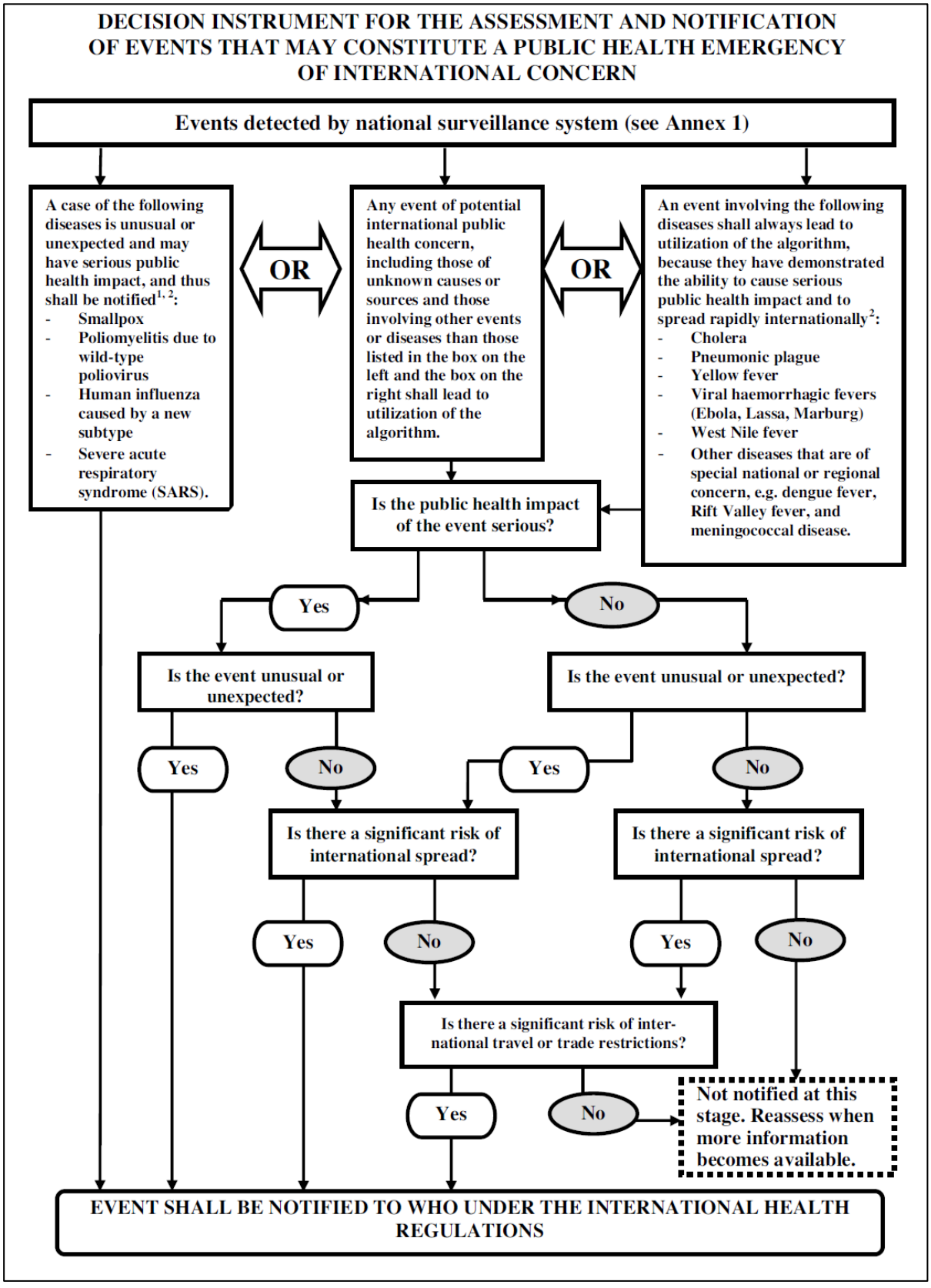
a. *Acute high dose radiological exposure (>2 Gy)*. Initial symptoms due to the prodromal phase of acute radiation syndrome will include nausea, vomiting and, for very high doses (>LD_{50}), diarrhoea.[1] The gastrointestinal syndrome will rapidly resolve and will mimic an acute gastroenteritis possible due to an enterotoxin. Later cases of the manifest illness following the latent phase will present as a neutropenic sepsis or appear to be a haemorrhagic biological syndrome due to a radiation-induced fall in leucocytes and coagulopathy. Cytogenetics will demonstrate radiological damage to the DNA (see Part 5).

b. *Heavy metal toxicity*. Heavy metal compounds that may be used deliberately include arsenic, thallium, mercury and lead. Some of these chemical may cause unusual symptoms and signs that include peripheral neuropathies, haematological disturbances, renal toxicity, pulmonary effects especially if inhaled, encephalopathies, transient pyrexias and in severe cases multi-organ failure. Some of these cases may look like severe sepsis but without an obvious source. It should be noted that some biological toxins, such as ricin, will also appear as multi-organ failure. Toxicological testing using urine or in some cases hair will demonstrate elevated heavy metal levels.

c. *Low level chemical exposure*. Exposures to non-lethal doses of nerve agents, cyanides and pulmonary agents may cause delayed onset or chronic effects. Low dose cyanide may be mistaken for a respiratory sepsis with raised respiratory rate and transient lactic acidosis. Phosgene-induced pulmonary oedema may also initially look like a respiratory biological syndrome especially due to the delayed onset and potential undetected exposure. For nerve agents, reduced red cell AChE levels may be demonstrated.

[1] Lethal dose for radiation exposures are often caveated with a time also i.e. $LD_{50/60}$ – over a 60 day period.

ANNEX 17A – PUBLIC HEALTH EMERGENCY DECISION TOOL



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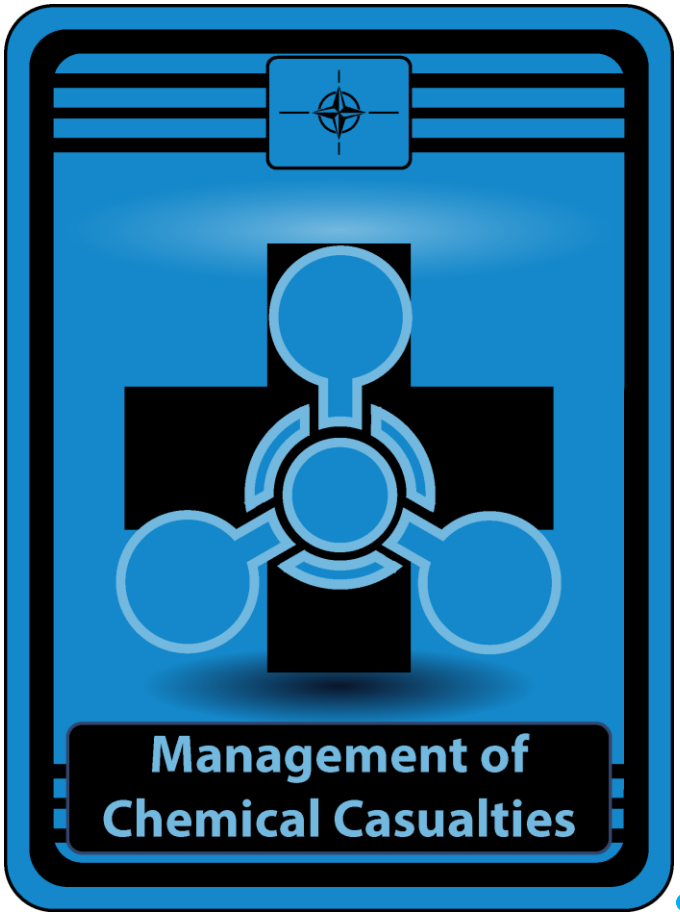
AMedP-7.1 PART 3: MANAGEMENT OF THE CHEMICAL CASUALTIES



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CHAPTER 18: INTRODUCTION TO CHEMICAL AGENTS

18.1. INTRODUCTION

1. The effectiveness of chemical agents as tactical weapons was clearly demonstrated in World War I and in the Iran-Iraq conflict. They can equally affect both forward and rear areas. In addition, CW agents could be used against military or civilian targets in terrorist actions, as demonstrated in the Tokyo subway sarin incident of 1995. In 1997 the Chemical Weapons Convention (CWC) came into force. This convention prohibits the development, stock pile and use of CW agents. Despite this convention the possibility of use of CW against NATO forces cannot be ruled out by nation states, insurgents and terrorists. CW exposure may also occur accidentally due to damaged legacy device or a demilitarisation accident.

2. Some of the characteristics shared with other CBRN agents are described in Chapter 2. The effective use of any chemical agent is dependent on its physical, chemical and toxicological properties as well as meteorological conditions at the time of and after the release.

18.2. TYPES OF CHEMICAL AGENTS

Chemical agents may be classified based on the severity of the effects, persistency, chemical group or similar mechanism or site of action.

18.2.1. SEVERITY

As described in Chapter 2, the severity of the effects of chemical agents can be classified as *lethal*, *damaging* and *incapacitating*. In addition, the effects of some MedCM may be significant and *iatrogenic* is also included as a fourth category.

18.2.2. CHEMICAL AGENT CLASSES

1. Chemical agents can be grouped into a number of classes based upon a similar toxicology and antidotes, site of action, type of presentation or legal status. Some traditional CBRN chemical classes based upon WW1 descriptions are listed in italics, however in some cases, the descriptions describe incorrect mechanisms i.e. cyanides ('blood agents') do not directly affect the blood. The classes are:

a. Nerve agents (organophosphorus) – see Chapter 19.

b. Vesicants (*'blistering agents'*) – see Chapter 20.

c. Pulmonary agents (*'choking agent'*) – see Chapter 21.

d. Cyanides (*'blood agents'*) – see Chapter 22.

e. Incapacitating agents – see Chapter 23.

f. Riot control agents (RCA) – see Chapter 24.

g. Pharmaceutical based agents (PBA) – see Chapter 24.

h. Military smokes and incendiaries – see Chapter 25.

2. *Toxic Industrial Chemical (TIC)*. TIC are often considered in a class of their own in isolation of the traditional chemical agent classes. However many TIC, including chlorine, have

been used on the battlefield. They are not banned by the Chemical Warfare Convention due to their civil industrial use. Some other TIC are operationally important as they may also be precursors used in the manufacture of chemical agents or breakdown products, or mimic the effects.

18.3. PHYSICAL CHARACTERISTICS OF CHEMICAL AGENTS

The physical properties affect the agent's state in the environment. They also influence the route of exposure (absorption) and method of weaponisation as well as persistency and the requirement for decontamination. Chemical agents cover a wide range of physical properties. Under ambient conditions, their physical state may be gaseous, liquid or solid. Their vapour pressures vary from high to negligible and their vapour densities vary from slightly lighter than air to considerably heavier. The range of odours varies from none to highly pungent or characteristic. Agents may be soluble or insoluble in water and this has a direct impact of their effect on the moist mucosa and early signs and symptoms. In the following chapters the physical properties of various agents are given in tables in the appropriate chapter. These may give an indication of the behaviour of the agents in the field with regard to routes of exposure e.g. inhalational or skin, effectiveness, persistency and potential methods of casualty decontamination.

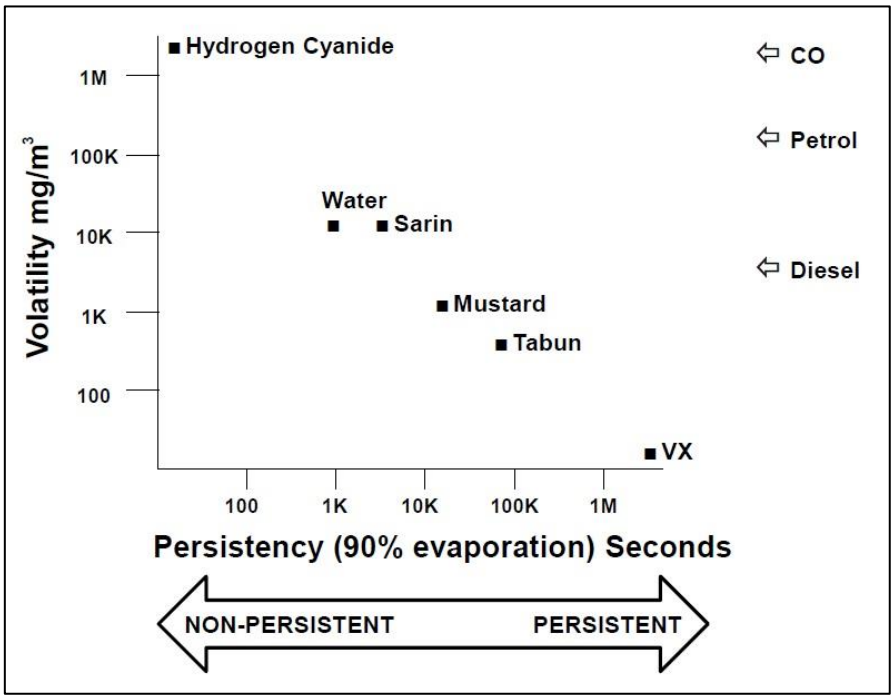

Figure 18-1: Persistency Comparison Chart with Illustrative Examples to Compare Decontamination Requirements.

18.3.1. PERSISTENCY

The duration that a chemical agent remains a hazard is called its persistency. Agents with a high boiling point and low vapour pressure tend to be persistent while agents with a low boiling point and high vapour pressure tend to be non-persistent or volatile. Persistency is defined as the length of time for which the agent will present an inhalation or contact hazard and in some literature water is used as a reference with a persistency factor of one. Agents with a factor less than one are more volatile and will evaporate quicker. Non-persistent agents disperse

rapidly after release and present an immediate, short duration and usually inhalational hazard. Persistent agents continue to present a hazard for considerable periods after delivery by remaining as a contact hazard or by evaporating over a longer period or in a warmer temperature (such as daytime or spring time) to produce a hazard by inhalation. Non-persistent agents may be made persistent by thickening. Figure 18-1 provides some examples of persistent and non-persistent agents.

18.3.2. METEOROLOGICAL FACTORS

The following meteorological factors will influence the duration of persistence of chemical agents:

a. *Winds*. The effect of wind is to disperse agents rapidly in open country. However, dangerous concentrations may remain longer in woods, trenches, dug-outs and built-up areas.

b. *Temperature*. High temperatures decrease the persistency of agents and cause higher vapour concentrations. Low temperatures increase the persistency of agents. Some agents may freeze thus reducing the immediate vapour hazard while remaining a potential contact hazard. There is a danger of carrying such frozen agents on clothing and equipment into a warm building with the subsequent risk of toxic vapour being given off.

c. *Rain*. Rain disposes, dilutes and promotes hydrolysis of agents. This reduces their effectiveness but does not make them impossible to use.

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

18.4. TOXICOLOGICAL PROPERTIES

It should be realised that not all individuals and species react in the same way to a given amount of agent. Also, toxicological studies estimate the biological effects of potential agents by different routes of exposure. The physical properties of such materials may affect the toxicological studies since the response of the biological system concerned may vary depending on the physical state of the material. Studies of the mode of action are related to the development of MedCM and physical protection. The terminology used in this manual is as follows:

a. *Dose*. The dose is the quantity of the compound received by the subject. This may either be expressed as an absolute dose based on a standard 70kg adult or as a relative dose based on weight i.e. mg/kg.

b. *Lethal Dose 50 (LD_{50})*. The LD_{50} is the dose which statistically would kill 50% of the exposed population. A list of agents and their comparative LD_{50} is in Table 18-1.

c. *Incapacitating Dose 50 (ID_{50})*. The ID_{50} is the dose which statistically would incapacitate 50% of the exposed population.

d. *Concentration Time (Ct)*. The Ct is a measure of exposure to a vapour or aerosol. The concentration in the air and the duration of exposure govern the dose received, as does the rate of respiration. It is assumed that, when the product of concentration and

time is constant, so is the biological effect over a limited range of concentration and time. For very short or long exposures the biological effect may vary. Concentration is expressed as $mg.m^{-3}$ and time as minutes, so that the concentration time (Ct) is expressed as $mg.min.m^{-3}$.

e. *Lethal Concentration Time 50 (LCt_{50})*. The LCt_{50} is the Ct which statistically would kill 50% of the exposed population. A list of agents and their comparative LCt_{50} is in Table 18-1.

f. *Incapacitating Concentration Time 50 (LCt_{50})*. The Ict_{50} is the Ct which statistically would incapacitate 50% of the exposed population.

g. *Therapeutic (safety) index*. This is the comparison or ratio of the dose to cause a therapeutic or effect (effective dose in the case of non-therapeutics) and the dose to cause adverse or lethal effects (see Chapter 24).

Table 18-1: Comparison of LD_{50} and LCt_{50} including Some Toxins for Comparison.[1]

AGENT	LD_{50} (mg)	LCt_{50} ($mg.min.m^{-3}$)
Botulinum toxin	0.0008	-
Chlorine (CL)	-	13500
Cyanogen chloride (CK)	-	4700
Hydrogen cyanide (AC)	7000 (skin)	2600
Phosgene (CG)	-	1500
Ricin	0.34 (mg/kg)	-
Sarin (GB)	1700 (skin)	33
Soman (GD)	350 (skin)	33
Sulphur mustard (HD)	1400 (skin)	1000 (inhalation) 5-10,000 (skin)*
Tabun (GA)	1500 (skin)	70
VX	3 (skin)	12

*LCt_{50} reduces with rising ambient temperature (i.e. >29°C)

18.5. MEDICAL COUNTERMEASURES FOR CHEMICAL CASUALTIES

1. The medical management of chemical casualties follows the same principles described in Part 1 of this AP. Treatment is focused on the initial management of T1 casualties. For chemical casualties, the early use of antidotes is important as a LSI. Early use of antidotes such as oximes (see Chapter 19) may reduce other medical logistic requirements including other antidotes such as atropine. Casualty decontamination as part of casualty hazard management is usually concurrent. The early removal of clothing and external contamination for lethal liquid/solid agents that are absorbed through the skin is also considered a LSI.

2. The use of chemical MedCM depends on a number of factors including the lethality of the agent, side effects of the MedCM, onset, certainty of diagnosis such as clinical investigations and efficacy.

[1] Based upon a 70kg person and 2 minutes vapour exposure time.

a. *Pre-exposure prophylaxis*. There are very few pre-exposure prophylaxis MedCM for chemical agents. Therefore, protection is provided by a deployed detection capability and physical protection such as IPE.

b. *Pre-treatment*. This type of MedCM is used to enhance post exposure MedCM such as antidotes. This type of MedCM will be specific to a class of chemical agent and an example is pyridostigmine for the pre-treatment of nerve agent exposure where ageing and reduced therapy efficacy is a concern (see Chapter 19).

c. *Post-exposure prophylaxis*. This type of MedCM has some limitations as the window of opportunity can be very short for acute intoxications such as inhaled nerve agent and cyanides. In addition, the side effects should be less than the effects of the agent and should not interfere with the assessment of any exposed person. Post-exposure prophylaxis is therefore likely to be limited to a detected exposure to a delayed onset chemical agent with limited side-effects.

d. *Immediate therapy*. This type of MedCM can be divided into symptomatic and agent specific therapy, however, there will be some overlap. This differentiation will be important for those developing first aid and treatment guidelines. Examples of each type of therapy are:

(1) Symptomatic treatment: Bronchodilators (for bronchoconstriction), atropine (for bradycardia & secretions), benzodiazepine (for seizures) and oxygen (for cyanosis).

(2) Agent specific: Oximes and atropine (for nerve agents), naloxone (for opiates), cyanide antidotes, and oxygen (for cyanide and CO).

e. *Continuing medical therapy*. Some MedCM will also be used in either the same or different forumulations for management within the medical chain. The agent-specific MedCM and continuing medical treatment along the casualty chain should ideally be interoperable and synergistic, while adverse drug interactions must be avoided.

Note. The issue of MedCM interoperability requires allied nations to declare deployed MedCM to each others' medical organisations.

18.6. DRUG INTERACTIONS WITH CHEMICAL AGENTS AND MEDCM

1. During any CBRN defensive operation, and especially where MedCM are used, medical personnel should be aware and anticipate potential drug interactions. The interactions may occur in a number of ways and either be:

a. *Synergistic* with an increased effect on an organ or system.

b. *Antagonistic* with a decreased or opposing effect on an organ or system.

c. *New effect.*

2. The interactions are either due to *pharmacodynamics*, the alteration of drug action on the target organ or system, or *pharmacokinetics,* the altered absorption, distribution, metabolism or elimination of the drug, MedCM or chemical agent. In the case of chemical agents, the most likely cause is due to altered pharmacodynamics due to the potent action of both the agents and some of the prescribed medications, in particular those used for anaesthesia.

3. The interactions described (Table 18-2) may be either theoretical or based on animal studies. In some cases, the experimental observations may differ from theoretical hypothesis of action.

Table 18-2: Potential Drug, MedCM and Chemical Agent Interactions.

Drug	Nerve agents
Succinylcholine	Inhibition of the acetylcholinesterases, including pseudo-acetylcholinesterase may prolong the effect of the drug. There may also be an increase in parasympathetic side effects such as bradycardia. The period of relaxation may be prolonged; a significant disadvantage for this drug during a rapid sequence induction (RSI). *A non-depolarising agent such as rocuronium should be considered although an increased dose may be required.*
Non-depolarising muscle relaxants	Non-depolarising muscle relaxants would expect to have competitive pharmacodynamics with excess ACh competing for the ACh receptor sites due to the effect of NAs. The period of paralysis may be less predictable. Use of clinical assessment including a peripheral nerve stimulator is recommended. In the case of rocuronium, an antidote (Sugammadex) is available in some nations.
Thiopentone	Thiopentone is an induction agent, sometimes used in trauma. It is also used as one of the induction drugs of choice in status epilepticus. It may cause cardiovascular depression and this may restrict its use in trauma. Thiopentone may also precipitate bronchospasm, especially in the presence of cholinergics.
Ketamine	Ketamine is widely used in pre-hospital scenarios due to its dissociative anaesthesia effects. In many nations, it is the drug of choice for the induction of anaesthesia for trauma and asthma as it has positive cardiovascular effects and acts as a bronchodilator. Research results are mixed with both positive and negative findings potentially in the presence of other anaesthetic agents. It would theoretically be the drug of choice in NA poisoning as it may reduce bronchospasm and be neuroprotective; it is also associated with increased airway secretions that could be reduced by atropine.
Propofol	Propofol appears to have protective properties in animal models with an increase in the LD50 required for the same toxic effects. Caution should however be exercised in the presence of trauma due to propofol's cardiovascular depressant effects.
Etomidate	There is no experimental data on the effect of etomidate in the presence of NA. Etomidate is often the drug of choice in the induction of anaesthesia in trauma patients due to its relative cardiovascular stability although adrenal corticosteroid suppression is documented.
Volatile agents	Inhalational agents are generally thought to have no significant interactions alone in NA casualties. These agents are used to maintain anaesthesia during animal research. Newer volatile agents may need further investigation. **Note**: Volatile agents may activate some chemical agent monitors.
Drug	**Sulphur mustard (HD)**
Succinylcholine	Succinylcholine appears to cause prolonged apnoea in mildly toxic animal models. Sulphur mustard has weak cholinergic properties. Its use in HD poisoning is not recommended and a non-depolarising agent such as rocuronium is advised.
Drug	**Pyridostigmine (PYR)**
There is concern that prolonged pyridostigmine exposure may cause down regulation of acetylcholine receptors as a result of excess acetylcholine. Caution should be applied in the interpretation of animal data using a short exposure time to PYR.	
Succinylcholine	PYR will inhibit plasma acetylcholinesterase, this may prolong the effects of succinylcholine. An increase in Ach may enhance the effect of succinylcholine and so a reduced dose may be required. In the 1991 Gulf War, there was no evidence to suggest that there was a prolonged apnoea as a result of PYR. Because of two potential interactions, non-depolarising muscle relaxants should be considered.
Non-depolarising muscle relaxants	Non-depolarising muscle relaxants may be antagonised by PYR (PYR acting similar to neostigmine) by increasing Ach levels. This has been seen in animal models. Larger doses of agents may be required and there is a theoretical risk of a reduction in the length of action. This effect can be monitored by a peripheral nerve stimulator, with appropriate titration of relaxant. An increase in the requirement of vecuronium was not noticed during the 1991 Gulf War.
Ketamine	There is no experimental evidence to suggest any specific interaction. However, both PYR and ketamine may increase airway secretions and this may precipitate respiratory problems.
Antimuscarinics	PYR will increase upper airway secretions. Antimuscarinics, such as atropine, may be required in higher doses to antagonise this effect.

CHAPTER 19: NERVE AGENTS

19.1. INTRODUCTION

The nerve agents (NA) are a group of particularly toxic organophosphorus esters classed as chemical agents. They were first developed in the late 1930s and were used by Iraq and the Aum Shinrikyo sect in Matsumoto and Tokyo. Recent use has been confirmed in 2013 in the suburbs of Damascus, Syria. Although chemically related to the organophosphorus insecticides, their clinical presentation and medical management may differ due to toxicity and different routes of exposure. The principle nerve agents known to have been weaponised are tabun (GA), sarin (GB), soman (GD), cyclosarin (GF) and the V-agents such as VX and VR. In addition, organophosphorus pesticides are toxic industrial chemicals that are still commonplace in some regions of the world and may be used opportunistically.

19.2. PHYSICAL AND CHEMICAL PROPERTIES

The “G” agents tend to be non-persistent, whereas the “V” agents are persistent. Some agents may be thickened with various substances in order to increase their persistence, and therefore the total amount penetrating intact skin. It can be seen that at room temperature GB is a comparatively volatile liquid and therefore non-persistent. GD is also significantly volatile, as is GA though to a lesser extent. VX is a relatively non-volatile liquid and therefore persistent. In the pure state nerve agents are colourless and mobile liquids. In an impure state, nerve agents may be encountered as yellowish to brown liquids. Some nerve agents such as GD have a faint fruity odour. In general, nerve agents are moderately soluble in water with slow hydrolysis, highly soluble in lipids and rapidly inactivated by strong alkalis and chlorinating compounds.

19.3. ROUTES OF EXPOSURE

Nerve agents may be absorbed through any body surface. When dispersed as a spray or an aerosol, agents can be absorbed through the skin, eyes and respiratory tract. When dispersed as a vapour, the vapour is primarily absorbed through the respiratory tract with a local effect on the respiratory tree; it also has a local effect on the eye. If enough agent is absorbed, local effects are followed by generalised systemic effects. The rapidity with which effects occur is directly related to the amount of agent absorbed in a given period of time and the route of absorption. The respiratory tract is the most rapid route for systemic absorption. After dermal exposure, there will be a latency period before local and systemic effects will appear.

19.4. MECHANISM OF ACTION

The effects of the nerve agents are mainly due to their ability to inhibit the enzyme acetylcholinesterase (AChE) throughout the body. Since the normal function of this enzyme is to hydrolyse acetylcholine wherever it is released, such inhibition results in the accumulation of excessive concentrations of acetylcholine at its various sites of action. Acetylcholine interacts with both muscarinic and nicotinic receptors resulting in different medical effects during acetylcholine overload. These sites are summarised in Table 19-1 and include:

a. *Parasympathetic*. The accumulation of acetylcholine at the endings of the parasympathetic nerves to the smooth muscle of the iris, ciliary body, bronchial tree, gastrointestinal tract and bladder; to the salivary glands and secretory glands of the gastrointestinal tract and respiratory tract; and to the cardiac muscle via the vagus nerve (muscarinic).

b. *Motor end plate (neuromuscular junction)*. The accumulation of acetylcholine at the endings of motor nerves to voluntary muscles (nicotinic).

c. *Autonomic ganglia*. The accumulation of excessive acetylcholine at some autonomic ganglia resulting in nicotinic signs and symptoms leading to increase parasympathetic tone and some limited sympathetic effects.

d. *Sweat glands*. The accumulation of excessive acetylcholine at the endings of sympathetic nerves to the sweat glands (muscarinic).

e. *Central nervous system*. The accumulation of excessive acetylcholine in the brain and spinal cord results in characteristic central nervous system symptoms.

Table 19-1: Medical Effects of Nerve Agent intoxication.

Mechanism	Site of Action	Signs and symptoms *
Muscarinic following local absorption (depending on dose)	Pupils	Miosis – marked, usually maximal (pin-pointed), sometimes unequal.
	Ciliary body	Frontal headache, eye pain on focusing, blurring of vision
	Nasal mucous membranes	Rhinorrhoea, hyperaemia
	Bronchial tree	Tightness of chest, bronchoconstriction, increased secretion, cough
	Gastrointestinal	Occasional nausea and vomiting
Muscarinic following systemic absorption (depending on dose)	Bronchial tree	Tightness in chest, with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, dyspnoea, pain in chest, increased bronchial secretion, cough, cyanosis
	Gastrointestinal	Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness with "heartburn" and eructation, diarrhoea, tenesmus, involuntary defecation
	Sweat glands	Increased sweating
	Salivary glands	Increased salivation
	Lachrymal glands	Increased lachrymation
	Heart	Bradycardia **
	Pupils	Miosis, occasionally unequal, later maximal miosis (pin-point)
	Ciliary body	Blurring of vision, headache
	Bladder	Frequency, involuntary micturition
Nicotinic following local or systemic absorption	Striated muscle	Easy fatigue, mild weakness, muscular twitching, fasciculations, cramps, generalised weakness/flaccid paralysis (including muscles of respiration) with dyspnoea and cyanosis
	Sympathetic ganglia	Pallor, transitory elevation of blood pressure followed by hypertension
Central nervous system (muscarinic and nicotinic effects)		Acute effects: Generalised weakness, depression of respiratory and circulatory centres with dyspnoea, cyanosis and hypertension; convulsions, loss of consciousness and coma
		Delayed effects: Giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headaches, tremor, withdrawal and depression, bursts of slow waves of elevated voltage in EEG (especially on hyperventilation), drowsiness, difficulty concentrating, slowness of recall, confusion, slurred speech, ataxia, memory and learning impairments

* These are the common sign and symptoms – presentations may vary depending on site of action.

** Cardiovascular – The heart rate may be decreased because of stimulation by the vagus nerve, but it is often increased because of other factors such as stress, hypoxia and adrenergic stimulation due to ganglionic stimulation. Thus, the heart rate may be high, low or in the normal range. Bradyarrhythmias such as first, second or third degree heart block may occur. The blood pressure may be elevated due to increased sympathetic vascular tone.

19.5. MEDICAL EFFECTS

The order in which signs and symptoms appear and their relative severity depends on the dose and route of exposure. The route of exposure often being determined by whether the agent is a liquid or vapour. The signs and symptoms following exposure to nerve agents are given in Table 19-1. The local effects of vapour and liquid exposure are described followed by a description of the systemic effects which occur after significant absorption of agent via any route (Table 19-2).

19.5.1. LETHAL EFFECTS

In the absence of treatment, death is caused by asphyxia resulting from airway obstruction, paralysis of the muscles of respiration and central depression of respiration. Airway obstruction is due to pharyngeal muscular collapse, upper airway and bronchial secretions, bronchial constriction and occasionally laryngospasm and paralysis of the respiratory muscles. Respiration is shallow, laboured, and rapid and the casualty may gasp. Cyanosis increases. Finally, respiration becomes slow and then ceases. Unconsciousness ensues. The blood pressure (which may have been transitorily elevated) falls. Cardiac rhythm may become irregular and death may ensue. If assisted ventilation is initiated and airway secretions are removed by postural drainage and suction and diminished by the administration of atropine, the individual may survive several lethal doses of a nerve agent albeit with a potential for severe irreversible brain damage. However, if the exposure has been overwhelming, amounting to many times the lethal dose, death may occur despite treatment as a result of respiratory arrest and cardiac arrhythmia. When overwhelming doses of the agent are absorbed quickly, death occurs rapidly without orderly progression of symptoms.

19.5.2. CUMULATIVE EFFECTS

Repeated exposure to concentrations of a nerve agent insufficient to produce symptoms following a single exposure may result symptoms over time. Continued exposure, possibly over several days, may be followed by increasingly severe effects. After symptomatic exposure, increased susceptibility to cholinergic effects may persist for up to 3 months (the body synthesises AChE at varying rates). During this period the effects of repeated exposures to any cholinergic agent are cumulative.

19.5.3. LONG TERM EFFECTS

Minor EEG changes have been noted more than a year after a clinically relevant, symptomatic nerve agent exposure when averaged EEGs in a group of people who had been exposed to a nerve agent were compared to a control group. Since changes could not be identified in individual EEGs, the clinical relevance is unknown. Neuropsychiatric changes have been noted in individuals for weeks to months after insecticide poisoning including poor concentration, memory problems and sleep disturbance. Organophosphorus-induced delayed neuropathy (OPIDN) has been reported after poisoning, although it has not been reported in humans exposed to nerve agents. OPIDN has been produced in animals only at doses of nerve agents so high that survival would be unlikely. Clinical signs or symptoms of the intermediate syndrome, including weakness and pain, has not been reported in humans after nerve agent exposure, nor has it been produced in animals by nerve agent administration, but might occur if the cholinergic crisis is prolonged.

Table 19-2: Local and Systemic Effects of Nerve Agent in Liquid and Vapour Form.

Nerve agent	Types of effects	Route of absorption	Description of effects	When effects appear after exposure*	Duration of effects after	
					Mild exposure	Severe exposure
Vapour	Local	Lungs	Rhinorrhoea, nasal hyperaemia, tightness in chest, wheezing	One to several minutes	A few hours	1 – 2 days
Vapour	Local	Eyes	Miosis, conjunctival hyperaemia, eye pain, frontal headache	One to several minutes	Miosis 24 hours	2 –3 days
Vapour	Systemic	Lungs	Muscarinic, nicotinic and central nervous system effects	Less than one minute to a few minutes after moderate or severe exposure	Several hours to a day	Acute effects 2 – 3 days. CNS effects days to weeks
Liquid	Local	Eyes	Same as vapour effects	Instantly	Similar to effects of vapour	
Liquid	Local	Ingestion	Gastrointestinal	About 30 minutes after ingestion	Several hours to a day	2 – 5 days
Liquid	Local	Skin	Local fasciculation at site	3 minutes – 2 hours	3 days	5 days
Liquid	Systemic	Lungs	Same as for vapour	Several minutes		1 – 5 days
Liquid	Systemic	Eyes	Same as for vapour	Several minutes		2 – 4 days
Liquid	Systemic	Skin	Generalised fasciculation and sweating	15 minutes – 2 hours		2 – 5 days
Liquid	Systemic	Ingestion	Early gastrointestinal symptoms	15 minutes – 2 hours		3-5 days

19.6. NERVE AGENT PRE-TREATMENT

The binding of a nerve agent to AChE is essentially irreversible in the absence of an enzyme reactivator such as an oxime (see below). Through *ageing* the binding of the agent to the AChE becomes absolutely irreversible and reactivators become ineffective. The time frame between the binding of an agent and its ageing depends on the agent. The window for the efficacy of an oxime to reactivate the inhibited AChE (enzyme-reactivation) may be very short for some nerve agents e.g. Soman (GD) is 2 minutes. These difficulties have been mitigated, in part, by the use of carbamates (reversible AChE binding) as pre-treatment (treatment enhancers).

Note. The function of pre-treatment and pre-exposure prophylaxis are different. Pre-treatment is the administration of drugs in advance of poisoning in order to enhance the efficacy of post-exposure treatment i.e. pre-treatment MedCM may not have any preventative properties before post-exposure therapy is given.

19.6.1. CARBAMATES

1. *Mechanism of action*. Current pre-treatment uses carbamate anticholinesterases e.g. pyridostigmine, by virtue of their capacity to bind to cholinesterases reversibly and preventing the organophosphorus (OP) binding to the enzyme. *Carbamates are intended to be used in combination with post-exposure immediate therapy i.e. nerve agent antidote auto-injectors.* The term reversible is used here comparatively: the carbamate-AChE complex breaks down

more rapidly than the organophosphorus-AChE complexes as pyridostigmine is in equilibrium and present in a free and bound form. In comparison the aged organophosphorus-AChE complex is stable. When carbamates are used as pre-treatments, carbamoylation of AChE prevents phosphorylation, but later the carbamate-AChE complex dissociates, freeing active enzyme. Current pre-treatment regimes using pyridostigmine are designed to give between 20 - 40% binding of peripheral AChE. This allows the carbamate to protect a significant proportion of the AChE against nerve agent binding. There are no significant acute cholinergic signs and symptoms associated with this level of inhibition as the inhibition of the enzyme is slower than that due to nerve agent and there is negligible inhibition of central AChE. In conjunction with immediate therapy, good protection against lethality is obtained within 2 hours of the first dose, but is enhanced by continued intake of pyridostigmine pre-treatment, according to the accepted dosage regimes. Pre-treatment must be in a formulation that can be used by non-medical personnel.

2. *Cessation of pre-treatment*. Pyridostigmine is only intended for short term use usually 14-28 days although this varies between nations and is also dependent on the nerve agent threat state. Pre-treatment must be stopped upon developing symptoms of nerve agent poisoning following a chemical warfare attack and post-exposure therapy started. Pyridostigmine is not a prophylaxis or immediate therapy, and it should not be taken after nerve agent exposure because it will block all the AChE and as a result may well enhance the effects of nerve agent poisoning. It is ineffective unless standard therapy is also used in the appropriate manner.

3. *Side effects***.** The recognised side effects of pyridostigmine use include gastrointestinal symptoms including increased flatus, loose stools, abdominal cramps and nausea. Other reported effects are urinary urgency, headache, rhinorrhoea, diaphoresis and tingling of the extremities. However, several studies have concluded that these side effects are tolerable by most and lead to negligible degradation of military performance. Symptoms due to pyridostigmine may be ameliorated by taking the tablets with food and/or combined with anticholinergic drugs such as hyoscine. Pyridostigmine pre-treatment may be discontinued on medical advice in less than 0.1% of individuals, generally because of intolerable nausea and diarrhoea. When taken in excess of the recommended dosage, symptoms of carbamate poisoning will occur including diarrhoea, gastrointestinal cramps, tight chest, nausea, rhinorrhoea, headache and miosis.

Note: *Contra-indications: Due to the possibility of bronchospasm, personnel with reversible airway disease such as asthma should not take carbamates and this may result in the loss of these personnel to the generated force.*

4. *Compliance*. Good compliance is required if optimal protection is to be obtained. The importance of pyridostigmine pre-treatment should therefore be stressed during training and issuing of the MedCM to personnel. Medical officers must be aware of potential drug interactions and contraindications when dealing with personnel on pre-treatment. This is of particular relevance with respect to some muscle relaxants used in anaesthesia. Pyridostigmine has been used in clinical situations for up to 50 years. At this time there is no evidence of long term side effects.

5. *National policies use of pre-treatment*. The national policy for the use and formulation of pre-treatment varies. It cannot be assumed that casualties from an allied nation will have received pre-treatment. Pre-treatment is only likely to be deployed where there is a significant threat to exposure to a nerve agent with a short ageing time.

19.7. CASUALTY ASSESSMENT AND CLINICAL INVESTIGATIONS

A summary of the management of nerve agent casualties is given in the factsheet at the end of this chapter, although specific regimens will vary between nations.

Table 19-3: Likely Signs and Symptoms and Approximate Red Blood Cell Acetylcholinesterase (RBC-AChE) Depression Following GB vapour Exposure

Short term Ct ($mg.min.m^{-3}$)	RBC-AChE inhibition approx)	Symptoms and signs*	
		Vapour exposure (including local eye and respiratory effects)	Systemic features (excluding local eye and respiratory effects)
<2	<5%	Incipient miosis (miosis produced at Ct=2, t=30 min), slight headache	Nil
5	20% ± 10%	Increased miosis, headache, eye pain, rhinorrhoea, conjunctival injection, tightness in chest	Tightness in chest
5-15	20-50% ±10%	Eye signs maximal, Bronchospasm and all the effects already described	Symptoms beginning to appear. Bronchospasm
15	50% ±10%	Bronchospasm and all the effects already described	Wheezing, salivation, eye effects, nausea, vomiting. (Local sweating and fasciculation in liquid contamination of the skin).
40	80% ± 10%	Symptoms and signs as for systemic	Weakness, defecation, urination. Paralysis, convulsions
100	100%	Respiratory failure, death	Respiratory failure, death

* All symptoms and signs will be subject to considerable inter-subject variation especially following mild vapour exposures. >80% depression is associated with life-threatening conditions and death. RBC-AChE is a surrogate marker for central AChE activitiy.

19.7.1 SYMPTOMS

1. Nerve agent poisoning may be identified from the characteristic signs and symptoms. If exposure to vapour has occurred, the pupils will be very small, usually pin-pointed. If exposure has been cutaneous or has followed ingestion of a nerve agent in contaminated food or water, the pupils may be normal or, in the presence of severe systemic symptoms, slightly to moderately reduced in size. In this event, the other manifestations of nerve agent poisoning must be relied on to establish the diagnosis. No other known chemical agent produces muscular twitching and fasciculations, rapidly developing pin-point pupils, or the characteristic train of muscarinic, nicotinic and central nervous system manifestations. A summary is in Table 19-1.

2. *Triage*. Triage criteria are found in the nerve agent factsheet.

3. *Clinical Investigations*. While measurement of tissue AChE levels is difficult, red blood cell AChE depression may used as a surrogate to confirm diagnosis and monitor treatment and further oxime therapy. Near-patient and laboratory testing is now available. Table 19-3 gives a summary of likely clinical effects and associated AChE depression.

Note. Caution should be used in the interpretation of RBC-AChE for clinical decision making and further treatment. This is due to a reported wide individual variation and limited correlation. Treatment should therefore be guided by clinical endpoints rather than biochemical surrogate markers. RBC-AChE remains an important forensic investigation in order to confirm exposure to nerve agent or other organophosphorus compound.

4. *Differential Diagnosis*. The effects caused by a mild vapour exposure, namely rhinorrhoea and tightness in the chest, may easily be confused with an upper respiratory illness, an allergy or, in the context of an explosive incident, blast. Miosis, if present, will help to distinguish these, but the eyes must be examined in very dim light to detect this and may be confused with opiates. Similarly, GI symptoms from other illnesses may be confused with those from nerve agent effects, and in this instance there will be no useful physical signs. History of possible exposure will be helpful, and laboratory evidence (decreased RBC-AChE activity), if available, will be useful to distinguish the two. The diagnosis is easier in the severely intoxicated patient. The combination of miosis, excessive secretions and generalised muscular fasciculations in a gasping, cyanotic and convulsing patient is diagnostic.

19.8. NERVE AGENT TREATMENT

1. Nerve agent casualty management consists of pre-treatment, diagnosis and treatment with a combination of pharmacological, basic airway, breathing and other general supportive measures, as guided by the condition of the casualty. The treatment of life-threatening nerve agent poisoning is largely based on clinical experience of OP pesticide poisoning. However, the life-threatening effects of acute nerve agent poisoning occur much more rapidly than those produced by OP pesticides with the exception of percutaneous nerve agent absorption. OP pesticide poisoning may also be combined with other constituents such as solvents.

2. Following pre-treatment, successful post-exposure treatment relies on the rapid recognition of effects and timely administration of the appropriate therapeutic measures. Urgent skin decontamination and continued respiratory protection must be applied alongside the administration of auto-injectors for self and buddy aid, all of which must be maintained during rapid evacuation to medical care. Triage priorities must be reviewed frequently, with subsequent delivery of appropriate drug therapy and continued respiratory support, where necessary, administered in a timely and confident manner if emergency medical management is to be successful and the ultimate prognosis favourable. Field medical management may have to be conducted initially in a contaminated environment, requiring practised procedures to avoid cross-contamination and unnecessary exposure of the already compromised casualty.

3. *Casualty decontamination*. The method of decontamination will depend on the physical properties of the agent (persistent or non-persistent) and type of exposure (direct contact with a liquid or to a vapour). If the uniform is contaminated, it should be removed as soon as possible. In the case of thickened mustard, the bulk of the agent may have to be scraped off with a knife or similar object.

4. *Nerve agent treatment*. The main principles of immediate therapy for nerve agent poisoning are based on early drug therapy in combination where necessary with measures to support respiration and provide general supportive care. The pharmacological treatment of nerve agent poisoning involves the use of:

a. Anticholinergics (antimuscarinics) to antagonise the muscarinic effects.[1]

b. Oximes to reactivate inhibited enzyme and thereby correct both muscarinic and nicotinic effects.

c. Anticonvulsants to prevent seizure activity and protect against subsequent CNS damage.

[1] Anti-nicotinic antagonists are also under investigation as potential nerve agent MedCM.

19.8.1. ANTICHOLINERGICS

1. Atropine is the most widely used anticholinergic for nerve agent intoxication, although scopolamine (hyoscine) can also be used. Immediate treatment with atropine in cases of systemic nerve agent poisoning is essential, and the absolute requirement will depend on both the severity of effects and whether pre-treatment has been used.

2. *Mechanism of action – antimuscarinic*. Atropine and scopolamine (hyoscine) act by blocking the effects of acetylcholine at muscarinic receptors and so produce relief from many of the parasympathetic symptoms including excessive secretions, bronchospasm and bradycardia. Some therapeutic effects are also produced within the central nervous system. At high doses (> 6 mg) atropine may reduce central respiratory depression, and if administered early (5-10 minutes after poisoning), it may have some anticonvulsant action. There is no direct effect at the nicotinic acetylcholine receptors at the neuromuscular junction although the action of the weakened diaphragm may be enhanced by the reversal of the parasympathetic effects on the respiratory tract such as bronchospasm and secretions.

3. *Signs of treatment efficacy*. Signs of successful atropinisation include decreased bronchospasm, reduced airways resistance, the drying up of bronchial and salivary secretions, reduced sweating and a stabilisation in the heart rate to approximately 90 beats per minute. The effect of atropine in drying bronchial secretions may make the removal of mucus more difficult so suction is likely to be necessary. After emergency medical treatment, atropinisation may be required to be maintained for 24 hours or longer either by intramuscular injection, repeated intravenous boluses or infusion of atropine adjusted as required or until AChE reactivation has taken effect. The reversal of miosis should not be used as an end-point as the nerve agent may have a local effect on the eye.

4. *Dose regime (atropine)*. The dose of atropine given depends on the user (non-medical or medical), route of administration and severity of the casualty. The following routes most likely to be used are:

a. *Intramuscular auto-injection*. For first aid auto-injector therapy by non-medical personnel, doses are usually small (2mg) and given regularly every 15 minutes until signs of successful atropinisation are noted or up to a dose of 6mg. This prevents high doses being given inadvertently if the recognition of nerve agent is incorrect. With enhanced chemical first aid training, some personnel may be authorised to give a severe case (T1) all three auto-injectors at the same time and thereby delivering 6mg as an initial dose. This must only be for cases where nerve agent poisoning is a definite diagnosis based on the syndrome and ideally supported by nerve agent detection.

b. *Intravenous / intraosseous route*. The intravenous (or intraosseous) routes is preferred in cases of severe poisoning. Both achieve rapid circulation times although the intraosseous route may require flushing after being given. Higher doses may be given by medical personnel and starting doses vary between nations ranging from 2mg to 10mg, either as a bolus or an infusion. These route allow rapid action and allows for titrating the dose to effect monitoring the reversal of parasympathetic effect such as secretions (bronchorrhoea), bronchospasm and bradycardia – the ‘3 Bs’. Some guidelines recommend avoiding the intravenous route until significant hypoxia is corrected, as in such cases intravenous administration of atropine may precipitate life-threatening ventricular arrhythmias. Paradoxically, aggressive atropine therapy may be the only way to reverse hypoxia. In the Iran-Iraq War, larger atropine doses were used in the absence of oxime therapy (20-200mg) and can be implied for cases of oxime-

insensitiviety.[2] Early and effective oxime therapy may significantly reduce atropine requirements and help to preserve stockpiles.

c. *Serial doubling*. One atropine dosing regimen suggested for the management of OP poisoning is serial doubling. This starts with an initial starting dose but after no action the dose is then rapidly increased to reach full atropinisation e.g. 2mg + 2mg+ 2mg + 4mg + 8mg + 16mg.[3] Higher doses may be expected for OP poisoning because of a large reservoir of pesticide remaining in the gastrointestinal tract causing a prolonged pharmacokinetic profile compared to the bolus effect of inhaled nerve agent vapour. This dosing regimen may be applicable to percutaneous nerve agent or where oxime therapy appears to be ineffective.

5. *Complications*. Atropine, especially in the presence of hypoxia, may render the myocardium more susceptible to arrhythmias. Correction of hypoxia, and ECG monitoring if available, is recommended during atropinisation. Atropine overdosage may produce euphoria, hallucinations, anxiety, and delirium. Close observation is necessary and sedation of casualties may be required. Urinary retention may necessitate catheterisation. By the reduction in sweating, atropine increases the risk of heat stress.

6. *Atropine for eye effects*. Atropine given systemically has comparatively little effect on nerve agent induced miosis. The reversal of miosis should not be used as a clinical end-point for atropinisation in the case of nerve agent vapour exposure. The local application of cycloplegic/ mydriatics (such as cyclopentolate, homatropine, tropicamine) to the eye reduces both the degree of miosis, eye pain and associated nausea, vomiting and headache. Patients should be warned of the effect on accommodation and discomfort due to bright light.

7. *Other anticholinergics*. Other anticholinergic drugs are considered to be beneficial in the treatment of nerve agent poisoning, based on experimental data. Anticholinergics with pronounced central antimuscarinic effects (e.g. scopolamine, benactyzine, biperiden, trihexyphenidyl) have the potential to suppress seizure activity without further anticonvulsant therapy, if administered early after poisoning. Drug combinations with atropine (e.g. benactyzine and atropine) have been introduced by some nations in specific clinical situations, but caution must be exercised when using these regimes due different pharmacokinetics and risk of over-atropinisation. Glycopyrrolate, a quaternary amine, is an antimuscarinic that does not cross the blood-brain barrier but will have a peripheral action and has a longer half-life than atropine. Some anticholinergics have also been suggested for use in combination with carbamates for pre-treatment.

19.8.2. ACETYLCHOLINESTERASE REACTIVATORS – OXIMES

1. Oximes are available in auto-injectors for self/buddy/first aid and injectable form for medical administration. The three most common oximes are listed are:

a. Pralidoxime (as chloride (2-PAM), methylsulphate or mesilate (P2S) salts).

b. Obidoxime (chloride (toxogonin)).

[2] In severe cases *previously receiving carbamate pre-treatment*, it is estimated that between 30-50 mg of atropine in total may be required to achieve atropinisation.

[3] Eddleston M *et al*. Management of acute organophosphorus pesticide poisoning. Lancet. 2008 Feb 16; 371(9612): 597–607.

c. HI-6 (dichloride or dimethanesulfonate (DMS)).

2. *Mode of action*. Oximes react chemically with the NA-AChE complex to remove the NA (enzyme reactivation). While atropine blocks the cholinergic effects of nerve agent poisoning at muscarinic sites (antimuscarinic), it has little effect upon the nicotinic effects at skeletal neuromuscular junctions and the autonomic ganglia. The reactivation of AChE will reverse the effects of the nerve agent, if given in a timely manner, and mitigate the effects of nerve agents at these sites and at muscarinic sites.

3. *Efficacy and choice of oxime*. The administration of atropine and oximes are synergistic in their effects. The relative potency of the different oximes in reactivating inhibited AChE varies according to the specific nerve agent. A rough guide to relative reactivation capacity (based on *in vitro* data) is presented in Table 19-4.

Table 19-4: In Vitro Comparison of Oxime Effectiveness.[4]

	Pralidoxime	Obidoxime	HI-6
Tabun (GA)	**-**	**+/-**	**-**
Sarin (GB)	**+**	**+**	**+**
Soman (GD)	**-**	**-**	**-**
Cyclosarin (GF)	**+/-**	**+/-**	**+**
VX	**+**	**+**	**+**
Key: + effective	- ineffective	+/- partially effective	
AGEING TIMES			
GA 46 hours	**GB** 5 hours	**GD** 2 minutes	**VX** > 48 hours

4. *Immediate therapy*. In the field, immediate post-exposure therapy consisting of atropine, oxime and benzodiazepine, if available, will be given by intramuscular injection from an auto-injector device on the appearance of the first significant signs of nerve agent poisoning. Following the use of the auto-injector, casualties if they remain symptomatic will require a therapeutic dose of oxime (and atropine titrated to effect) to reactivate the inhibited enzyme. The therapeutic dose should be given as soon as possible and this may be a combination of auto-injector and intravenous (or intraosseous) injection. Where there is doubt as to the dose given by auto-injector, the full dose should be given by medical personnel by the most effective route and under observation.

5. *Dosing*. The dosing schedules of the currently available oximes for intravenous use (based on data derived from human cases of organophosphorus insecticide poisoning) are shown in Table 19-5. Under field conditions, similar doses can also be given by intramuscular injection.

Note: *Rapid bolus intravenous (or intraosseous) injections of oximes should be avoided due to possible respiratory arrest. A slower infusion over 5-10 minutes is recommended and manufacturers' guidance should be followed.*

6. *Monitoring oxime therapy*. Oxime reactivation and efficacy can be monitored by a reduction in atropine use, return of muscle (nicotinic) function and increased RBC-AChE level, where AChE monitoring point of care testing is available. The monitoring of oxime therapy is

[4] ✍ Consensus from previous edition AMedP-6(C) and recommendation from HFM-253.

vital in order to confirm the efficacy of the chosen oxime, identify therapy failure including potential oxime-insensitivity and/or ageing of the nerve agent – AChE complex.

Table 19-5: Current Oxime Dosing.

Oxime	Route of administration	Initial dose	Continuing dose
Pralidoxime chloride (2-PAM)	Individual auto-injector dose Total adult loading dose	600mg 2g (30mg/kg)	Repeat initial dose every 10-12 hours as required
Pralidoxime mesilate (P2S)	Individual auto-injector dose Total adult loading dose	500mg 2g (30mg/kg)	Repeat initial dose every 10-12 hours as required
Obidoxime chloride	Individual auto-injector dose Total adult loading dose	220mg 250mg	750mg/day as continuous infusion

7. Failure of oxime therapy. Failure of oxime therapy is possible due to poor oxime efficacy for the nerve agent used e.g. pralidoxime and GA exposure, or ageing in the case of GD exposure. As well as nerve agent detection it is vital that the nerve agent is identified to enable the correct oxime to be used if there is a choice. Signs of oxime therapy failure include a continuing requirement of high dose atropine, nicotinic stimulation including fasciculation, continuing voluntary muscle paralysis and respiratory failure. Further management will require the use of an alternative oxime, or if none is available or ageing occurred increased atropine dosing using serial doubling should be started.

8. *Continuing oxime therapy*. It is generally accepted that nerve agent is rapidly cleared from the blood or hydrolysed following an acute exposure. However prolonged absorption following percutaneous exposure could result in the persistence of clinical relevant amounts of nerve agent in the body. Continued oxime therapy may therefore be required, either given at intervals or as a continuous infusion, supported by AChE monitoring. As a result of the ageing of the inhibited AChE, it is suggested that in the absence of clinical or monitored improvement, administration of oxime for periods in excess of 24-48 hours is unlikely to achieve further reactivation. For percutaneous poisoning, prolonged oxime treatment may be beneficial until nerve agent absorption and distribution has ceased.

9. *Side Effects*. The rapid intravenous injection of pralidoxime can produce drowsiness, headache, visual disturbance, nausea, dizziness, tachycardia, increased blood pressure, hyperventilation and muscular weakness. Obidoxime produces hypotension, a menthol-like sensation, a warm feeling in the face and hepatic dysfunction has been observed. On intramuscular injection, it can produce a dull pain at the site of infection; after multiple dosing.

19.8.3. ANTICONVULSANT THERAPY

1. In the absence of anticonvulsant therapy, irreversible brain damage may result; this is exacerbated by periods of hypoxia. Atropine protects only partially against seizure activity and the resulting brain damage in severe poisoning; other anticholinergics vary in this ability. Early administration of anticonvulsants is vital for the saving of life and reducing long term brain damage and disability. Experimental evidence shows that the early administration (5-10 minutes) of a benzodiazepine antagonises the seizure activity of nerve agent. The addition of this class of drugs to the basic treatment regimens greatly improves morbidity and mortality, in addition to its anti-seizure effect. Currently diazepam (or its prodrug avizafone in auto-injectors) is the most widely fielded and should be administered as a 5-10 mg dose initially and further doses should be given frequently enough to control seizures. This may require additional injections at intervals ranging from a few minutes to several hours. Other

benzodiazepines such as midazolam and lorazepam may available and can be also be effectively used.

2. The management of status epilepticus will be challenging. Large doses of benzodiazepines may be required and result in further airway and ventilatory compromise. Advanced airway management and critical care may be required once in hospital and resources allow. Further treatment may require formal muscle paralysis and ventilation, while the brain is monitored for further seizures by electroencephalogram monitoring, if available, or non-specific signs such as hypercapnia and hyperpyrexia. Other agents with potential anticonvulsant properties, especially in refractory epilepsy include ketamine. Cardiotoxic anticonvulsants such as phenytoin should be used with caution due the potential interactions.

19.8.4. BIOSCAVENGERS

Bioscavengers are a relatively new concept using biological proteins such as butyrylcholinesterase (BChE). The proteins act as an alternative target site for nerve agent to bind to and become deactivated.

19.9. CASUALTY DECONTAMINATION

The importance of early skin decontamination cannot be over-emphasised. Decontamination of the skin should be accomplished immediately after exposure to liquid agent if it is to be fully effective. However, a low degree of protection is provided by late decontamination. Liquid agent may be removed by an adsorbent, such as fullers' earth, or chemically inactivated by the use of active decontaminants such as Reactive Skin Decontamination Lotion (RSDL). Decontamination personnel should use a respirator and full protective equipment whilst decontamination is performed. If battle dress is contaminated, it should be removed as soon as possible. Once a casualty has been decontaminated or the agent is fully absorbed, no further risk of contamination exists. The casualty's body fluids, urine or faeces do not present a CW hazard.

19.10. ATROPINE TOXICITY

If atropine is administered in the absence of nerve agent poisoning, the following effects may be noted: dryness of the mouth and pharynx, decreased sweating, slight flushing and tachycardia, urinary retention, dilated pupils, mild drowsiness, impaired memory and recall and blurring of near vision. This can be remembered as the saying "*Mad as a hatter, blind as a bat, dry as a bone, red as a beet, hot as hell*". After one 2 mg injection, these symptoms should not interfere with ordinary activity except in the occasional person, in hot environments or at high work rates especially if wearing IPE. Mydriasis may cause problems with focusing and the aiming of weapon systems. Higher doses, or repeated doses, will produce more marked symptoms which may become totally incapacitating, particularly in warm environments or high work rates. The effects of atropine are fairly prolonged, lasting 3 to 5 hours after one or two injections of 2 mg and 12 to 24 hours after significant over-atropinisation. Management of an atropine overdose is by:

a. *Supportive management*. This consists of simple measures such as removing any personal weapons, reassuring the casualty and reducing external stimulation, prevent and treat any heat illness and avoid physical restraint. If unwell, including severe confusion, heat stroke and chest pain, seek medical advice. The antidote for simple anticholinergic intoxication is physostigmine.

b. *Physostigmine*. The traditional antidote for atropine overdose is physostigmine 1-2mg intravenous injection, repeated as required. This is a carbamate anticholinesterase, like pyridostigmine, but it crosses the blood brain barrier. The administration of physostigmine is also for use in the treatment of BZ intoxication (incapacitation), as detailed in Chapter 23 (Incapacitants). However, as physostigmine acts by inhibiting anticholinesterase, it must be used with caution in treating over-atropinisation following nerve agent poisoning, where the levels of enzyme may be reduced below normal. In such cases, the risk of repeated cholinergic effects is greater. Its use should therefore be reserved only for those cases with severe symptoms of atropine overdose, refractive to general supportive measures.

NERVE AGENTS

Tabun (GA)	Sarin (GB)	Soman (GD)	Cyclosarin (GF)	V-agents incl. VX
Colourless to brown liquid. Fruity smell.	Colourless liquid. Odourless.	Colourless liquid. Fruity, camphor.	Colourless liquid. Odourless.	Colourless or straw liquid. Odourless.

MECHANISM OF ACTION

Inhibition of the enzyme acetylcholinesterase that breaks down the neurotransmitter acetylcholine. This results in over stimulation of the following parts of the nervous system:
Parasympathetic: Miosis, secretions (tears, bronchorrhoea, salivation), vomiting, incontinence, bradycardia.
Central nervous system: Confusion, coma, seizures and central respiratory failure.
Sympathetic ganglia: Tachycardia, hypertension. *Sweat glands*: Sweating.
Neuromuscular junction: Fasciculation (systemic and local), depolarising paralysis, respiratory failure.

QUICK LOOK (CRESS)

Conscious	Respiration	Eyes	Secretions	Skin	Other
Convulsions Unconscious	Increased → Reduced / Stopped	**Pinpoint pupils**	**Increased** Vomiting	Sweating	Bradycardia

CASUALTY MANAGEMENT

SELF / FIRST AID

Remove from scene
Immediate decontamination
Clear secretions / vomit (suction airway, if equipment available)
Nerve Agent Antidote as auto-injector or equivalent
Place in recovery (semi-prone) position

CASUALTY DECONTAMINATION

GB – Vapour hazard
GA, GD, GF – Liquid/Vapour hazard
VX – Liquid hazard
Some agents may be thickened and require debulking before formal decontamination.

TRIAGE CATEGORIES

T1 (Severe)	T2 (Moderate)	T3 (Mild)
Unconscious, convulsions, respiratory distress, respiratory paralysis / arrest, very slow heart rate < 40, cyanosis.	Not walking. Excessive secretions, confusion, not obeying commands, wheezing, vomiting, diarrhoea.	Walking. Pinpoint pupils, dimmed vision, eye pain.

EMERGENCY MEDICAL TREATMENT

Supportive management:
Airway: Airway manoeuvres, suction
Breathing: Ventilatory support, oxygen, reverse secretions and bronchospasm,
Circulation: Reverse bradycardia.
Disability: Treat convulsions.

MedCM / Antidotes:
(Pre-treatment: Pyridostigmine*)*
Antimuscarinic: Atropine titrated to effect (see 3B below)
Atropine 2mg IM every 5-15 minutes
Atropine 2-10mg IV/IO over 5 minutes; titrated to effect
Oxime: Pralidoxime, obidoxime or HI-6 (auto-injector, IV, IO)
Anticonvulsant: Benzodiazepines (auto-injector, IV, IO)

Clinical Investigations:	Red blood cell acetylcholinesterase level (limited correlation with clinical severity) Blood and urine toxicology analysis

ADDITIONAL INFORMATION

For skin exposure – localised muscle twitching, delayed pinpoint pupils
DUMBELS (**d**iaphoresis (sweating), **u**rination, **m**iosis (pinpoint pupils), **b**radycardia, **e**mesis (vomiting), **l**achrymation (tears), **s**weating)
Treatment: High doses of atropine are expected to be reduced by the early use of an effective oxime. Where the oxime does not show efficacy due to ageing or poor NA selectivity, an alternative oxime should be considered and/or the atropine dose should be increased by serial doubling titrated to the clinical endpoints reversal of the 3B's (bradycardia, bronchospasm, bronchorrhoea (secretions)).

ATROPINE OVERDOSE / BZ (ANTI-MUSCARINICS)

Atropine	3-Quinuclidinyl benzilate (BZ)

MECHANISM OF ACTION

Both atropine and BZ are peripherally and centrally acting anti-cholinergic (anti-muscarinic) antagonists. They cause:

Parasympathetic: Blurred vision, dilated pupils, dry mucosa, urinary retention, and tachycardia.
Central nervous system: Confusion, delirium & coma.
Sympathetic ganglia: Dry skin and risk of heat illness.

Hazard: Atropine (ingestion, injectable), BZ (solid aerosol).

QUICK LOOK (CRESS)

Conscious	Respiration	Eyes	Secretions	Skin	Other
Agitated / Confused	Increased	Large pupils / Blurred vision	Dry mouth / Feeling of thirst	Flushed / Dry skin	**Agitated / Confused**

Differential diagnosis: Heat stroke, acute stress reaction, mass psychogenic illness.

CASUALTY MANAGEMENT

SELF / FIRST AID

Remove any weapons from casualty
Remove from scene, if chemical agent
Immediate decontamination, if chemical agent
Reassure casualty, & avoid physical restraint due to risk of heat illness
Manage in a cool, calm and sheltered environment

CASUALTY DECONTAMINATION

Decontamination not required for atropine.
Remove clothing, avoid inhaling any re-aerosolised particulates and rinse.

TRIAGE CATEGORIES

T1 (Severe)	T2 (Moderate)	T3 (Mild)
Unconscious, extreme agitation or violent behaviour, convulsions, chest pain. Temp > 40°C.	Not walking. Confusion, not obeying command, hallucinations. Temp > 39°C.	Walking. Dry mouth, blurred vision, mild agitation.

EMERGENCY MEDICAL TREATMENT

Supportive management:
Consider sedation – benzodiazepines.
Prevent and treat heat illness.
Manage any urinary retention.

MedCM / Antidotes:
Physostigmine injection (1-2mg intravenous injection, repeated as required).

Clinical Investigations:	Urinalysis.

ADDITIONAL INFORMATION

Symptoms may continue for several hours.

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CHAPTER 20: VESICANTS (BLISTERING AGENTS)

20.1. INTRODUCTION

1. Blister or vesicant agents are likely to be used both to produce casualties and to force opposing troops to wear full IPE thus degrading fighting efficiency. The effect of vesicant ranges from mild incapacitation through the systemic toxicity and death. They can be thickened in order to enhance persistency and contaminate terrain, ships, aircraft, vehicles or equipment. Additionally, the loading of sulphur mustard on particulate matter is known as dusty mustard.

2. The vesicant agents include:

   a. Sulphur mustard (H) (HD refers to distilled mustard).

   b. Nitrogen mustard (HN).

   c. Arsenical vesicants such as Lewisite (L).

   d. Halogenated oximes, such as phosgene oxime (CX).[1]

3. Vesicants cause chemical burns with skin and any other part of the body that they come into contact with. All of the mustards are alkylating agent and some of the nitrogen mustards have been used as chemotherapeutic agents. As well as the local contact effects, vesicants may also have systemic effects either due to direct effects as in the case of the arsenicals and to some degree the mustards, or indirectly through secondary mechanisms such as SIRS and ARDS.

4. In addition there are a number of toxic industrial chemical that can cause chemical burns that may be used opportunistically or may mimic the effects of vesicants.

20.2. PHYSICAL AND CHEMICAL PROPERTIES

1. Most of the vesicants are persistent agents and remain in the environment for some time and up to several months. The hazard from the agent (liquid or vapour) will also be determined by the environmental conditions such as ambient temperature and humidity. Each type of vesicant will be described in its relevant section.

2. Understanding the physicial and chemical properties for this group of agent is vital in order to understand the best risk reduction and decontamination methods. Mustard agent that comes into contact will bind to tissues within minutes. For contact hazard, 80% is thought to evaporate while 20% is absorbed. The situation is complicated by the fact that mustard compounds do not cause any immediate skin discomfort and are therefore initially asymptomatic. Lewisite and phosgene oxime however cause immediate pain, providing an indication for immediate decontamination.

20.3. ROUTES OF EXPOSURE

While the agent is in a liquid state, the main target organ will be skin. Once the agent starts to evaporate and becomes airborne the routes of exposure and organs exposed include:

[1] Although phosgene oxime is a corrosive and an urticant (producing wheals) rather than a vesicant. It is usually group with the true vesicants.

a. Skin.

b. Eyes.

c. Upper airway.

d. Lungs – associated with more severe cases (and with warmer climates).

e. Gastrointestinal tract (less likely).

20.4. MUSTARD AGENTS

1. Sulphur mustard was used extensively in World War I, during the Iran / Iraq War and used against the Kurdish population in Iraq in 1985. Protection against these agents can only be achieved by a full protective ensemble. The respirator alone will protect against eye and lung damage, but does not give sufficient full body protection. Extensive, slow healing skin lesions and other effects will place a heavy burden on the medical services.

2. Sulphur mustard (bis (2-chloroethyl) sulphide) is the best known of these agents. Synthesised in 1822, its vesicant properties were discovered in the middle of the nineteenth century. As a chemical agent it was used for the first time in 1917 near Ypres, Belgium leading to the French name (Yperite). It is also known by the name"LOST" in German. In the US the symbol HD has been given to the distilled product. In 1935 it was discovered that the vesicant properties remained when the sulphur atom was substituted by a nitrogen atom leading to the nitrogen mustard series.

3. *Physical Properties*. Sulphur mustard is denser than water, but small droplets remain on the water surface and present a special hazard in contaminated areas. Spreading can also occur when decontaminating the skin with aqueous solutions. This effect can be minimised by the use of adsorbents such as fullers' earth (FE), emulsifying or reactive decontamination agents and rinsing with copious amounts of water. The mustards are able to penetrate most tissues they come into contact with and many other materials such as woods, leather and rubber. Due to their physical properties, mustards are very persistent in cold and temperate climates. In warmer climates the persistence of mustards is reduced but the hazard from vapour increases with an increase in respiratory cases. It is possible to increase the persistency by thickening mustard with finely powdered material. These thickened mustards are more difficult to remove by decontaminating processes, and highlight the importance of IPE.

4. *Chemical properties*. Mustards are instable in normal water and inactivated by hydrolysis although mixing is required for this to be achieved. Alkalinity and higher temperatures increase the rate of hydrolysis. In running water the contact surfaces are frequently changed and persistency is only a few days; in stagnant water, the persistency can be several months while salt water increases persistency.

5. *Detection*. Mustard agents can be detected by a variety of means including vapour ionisation and immunological assays. Single and three colour detector papers will detect liquid agent and monitoring devices for vapour hazard and water testing kits are also available.

6. *Clinical investigation*. Clinical samples including blister fluid, blood, tissue and urine may be used to detect either mustard, protein and DNA adducts, and hydrolysis products (e.g. thioglycol) for some time after exposure.

Note. Some vapour detectors if used in hospital may misinterpret volatile anaesthetic agents as nerve agent or sulphur mustard and should be interpreted in the clinical context.

20.4.1. MECHANISM OF ACTION

1. The exact mechanism of action is not fully understood, however several mechanisms are thought to be in action. Sulphur and nitrogen mustards are alkylating agents and affect a very wide range of biologically important molecules. One important molecular interaction is with DNA leading to the formation of cross-links leading to cell death or mutation, similar to the effects of ionising radiation. Alkylation of ribonucleic acid (RNA), proteins, cellular membrane components, and cross-links between DNA, and proteins / enzymes can be the cause of cellular damage, necrosis and programmed cell death (apoptosis) (see Figure 20.1).

2. The main mechanisms of action are:

   a. Cytotoxic; and

   b. Mutagenic.

3. There may be other effects due to reactions with cellular membranes or critical enzymes. Due to the resemblance to ionising radiation, the mustards are sometimes referred to as ‘radiomimetic compounds’. As with radiation the cells exposed to the greatest dose with the highest turnover appear to be more affected including the basal epidermal cells, and those of the haematopoietic system and mucosa.

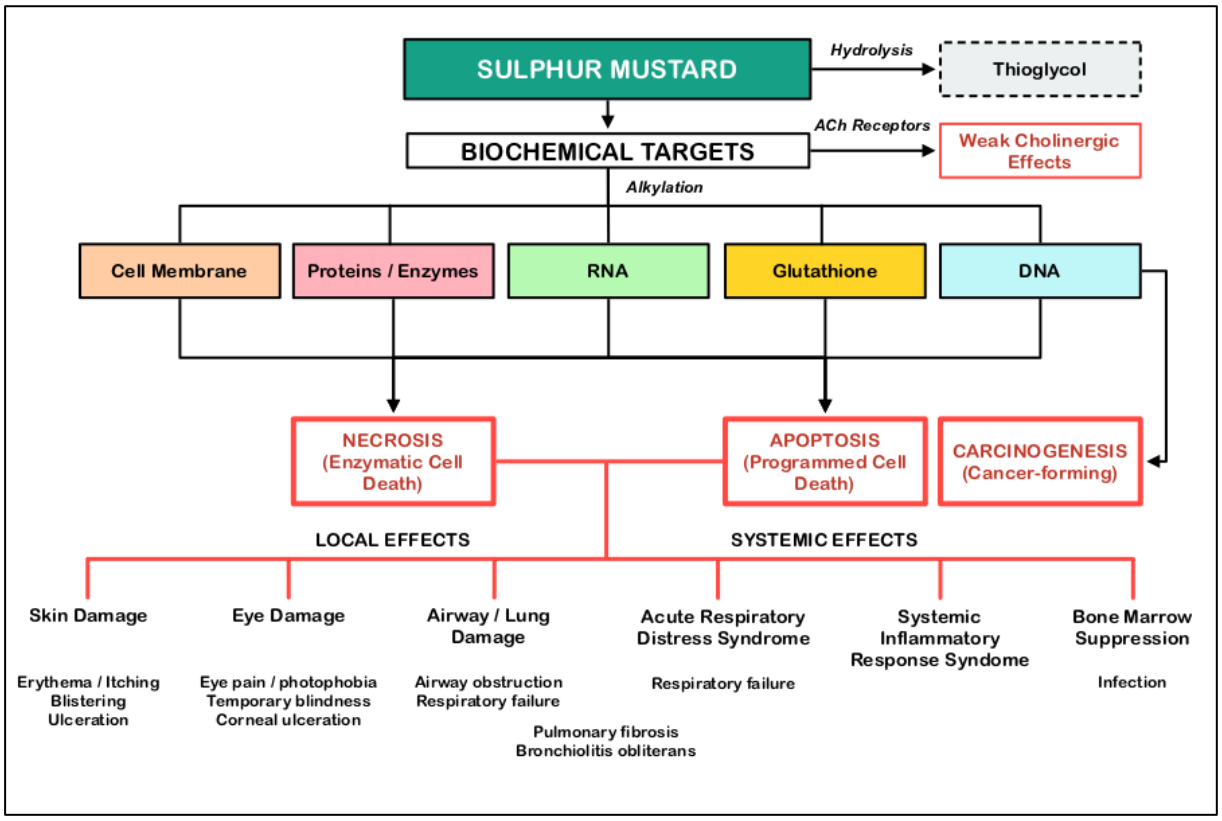

Figure 20-1 - Mechanisms of Action for Sulphur Mustard.

20.4.2. MEDICAL EFFECTS

1. *Skin*. The hallmark of sulphur mustard exposure is a latency period of 6-24 hours post exposure before blister formation. The duration of this period and the severity of the lesions are dependent upon the level and type of exposure, environmental temperature and probably on the individual susceptibility. High temperature, moist, thin or delicate and occluded skin are associated with more severe lesions and shorter latent periods for a given dose. The sensitivity of the skin depends on its thickness and upon the density of sweat and sebaceous glands. Apart from mucous membranes the most sensitive areas are the face, axilla, genitalia, neck, skin between the fingers and the nail beds. The palm of the hand, sole of the foot and the skin of the scalp are very resistant. Some people are markedly more sensitive to sulphur mustard than others. Burns may be the result of either vapour or liquid exposure. The sequence of skin changes normally seen is as follows:

a. *Erythema (6-12 hours post exposure).* Generalised redness is seen initially. Itching is also common and may be intense (this sequence is reminiscent of that seen in sunburn).

b. *Blistering (12-24 hours post exposure).* Erythema is followed by the development of numerous small vesicles which may coalesce to form larger blisters. Blisters at points of flexure, anterior aspects of elbows and posterior aspects of knees can seriously restrict movement. Sulphur mustard blisters are delicate and may be easily ruptured by contact with bed linen, bandages or during transport of casualties. Crops of new blisters may appear as late as the second week post exposure.

Note. Blister fluid is not a vesicant and does not produce secondary blistering or form a chemical hazard to medical personnel although it remains a biological hazard.

c. *Deep burning with pigmentation and scarring.* This is particularly likely to occur on the eyelids, penis and scrotum since the epidermis in these sites is particularly thin, naturally moist and often occluded. Lesions tend to be more painful and some patients complain of very severe pain. Healing of skin lesions is slow. The areas which were markedly erythematous may darken and become hyperpigmented. Brownish-purple to black discolouration of some areas may occur. These changes tend to disappear over a period of several weeks with desquamation leading to the appearance of areas of hypopigmentation. The appearance of such areas alongside those of hyperpigmentation may be striking.

2. If only a small dose is applied to the skin the effect is limited to erythema and after several days the colour changes from red to brown. The itch diminishes progressively and the epidermis desquamates. At higher doses, the blistering can go on for several days before reaching its maximum. They are often more than 1 cm^2 and may be very large forming bulla. Their domes, which are thin and yellowish, contain a relatively clear or slightly yellow liquid. The blisters are fragile and usually rupture spontaneously giving way to a suppurating and necrotic wound. The necrosis of the epidermal cells may be extended to the underlying tissues, especially to the dermis. The damaged tissues are covered with necrotic debris and are extremely susceptible to infection.

3. The systemic fluid derangement seen as a consequence of these injuries is appreciably less than for thermal burns, and, therefore the overall outcome is better for equally large affected areas.

4. *Eyes*. The eyes are more susceptible to sulphur mustard than either the respiratory tract or the skin. Mild effects may follow exposure durations of about 1 hour to concentrations barely perceptible by odour or monitoring. This exposure does not affect the respiratory tract significantly. A latency period is 4 to 12 hours, after which there is lachrymation and a sensation of grit in the eyes. The conjunctivae and the lids become red and oedematous. Heavy exposure irritates the eyes after 1 to 3 hours and produces severe lesions. Sulphur mustard burns of the eyes may be divided as follows:

a. Mild conjunctivitis (75% of cases in WW1) with recovery in 1-2 weeks.

b. Severe conjunctivitis with minimal corneal involvement (15% of cases in WW1). Blepharospasm, oedema of the lids and conjunctivae occur, as may an orange-peel roughening of the cornea. Recovery takes 2-5 weeks.

c. Mild corneal involvement (10% of cases in WW1). Areas of corneal erosion stain green with fluorescein dyes. Superficial corneal scarring and vascularisation occurs as does an iritis. Temporary relapses occur and convalescence may take 2-3 months. Hospital care or close ambulatory monitoring should be considered for casualties of this type.

d. Severe corneal involvement (about 0.1% of WW1 sulphur mustard casualties). Ischaemic necrosis of the conjunctivae may be seen. Dense corneal opacification with deep ulceration and vascularisation occurs. Convalescence may take several months and patients are predisposed to late relapses even after many years. Late relapses have a bad prognosis and are refractory to therapy.

5. *Airway and Lungs*. Sulphur mustard attacks all the mucous membranes of the respiratory tract. After an average latent period of 4 to 8 hours (range 2 to 48 hours depending on dose), it irritates and congests the mucous membranes of the nasal cavity and the throat, as well as the epithelium of the trachea and large bronchi. Symptoms start with rhinorrhoea, burning pain in the throat and hoarseness of the voice. This pain may make the patient reluctant to cough. A dry cough gives way to copious expectoration. The vocal cords often become damaged, resulting in aphonia. Airway secretions and fragments of necrotic epithelium may obstruct the airways; rales and reduced air entry can be detected by auscultation. There is pronounced dyspnoea. The damaged lower airways become infected easily, predisposing to bronchopneumonia after approximately 48 hours. If the inhaled dose has been sufficiently high the victim dies in a few days, either from pulmonary oedema or mechanical asphyxia due to fragments of necrotic tissue obstructing the trachea or bronchi, or from superimposed bacterial infection, facilitated by an impaired immune response. Significant BSA of burns may also lead to secondary injury through SIRS and a resulting ARDS.

6. *Gastrointestinal tract*. Ingestion of contaminated food or water may cause destruction of mucous membranes although this route is the least likely. The perforation of the gastrointestinal tract with subsequent mediastinitis or peritonitis may even occur in the case of severe ingestion. Symptoms include nausea, vomiting, pain, diarrhoea and prostration. These features may make casualties reluctant to eat. Vomit and faeces may be bloodstained. Hypovolaemic shock may occur from the loss of fluids and electrolytes from prolonged vomiting and diarrhoea.

7. *Systemic effects*. Systemically absorbed sulphur mustards by any route, including severe skin exposure, may cause signs similar to those of irradiation and chemotherapy, such as headache, gastrointestinal pain, nausea, vomiting, leucopenia and anaemia. As with

traumatic insults, a large surface area of chemical burns may cause a SIRS and associated ARDS. Both will require hospital monitoring and the later may require critical care and possible long term ventilation, although treatment will be similar to ARDS due to conventional causes.

8. *Immunosuppression*. Mustard agents may cause a general depletion of all elements of the bone marrow. The cells of the granulocyte series and megakaryocytes appear more susceptible to damage than those of the erythropoietic system. A reactive leucocytosis may occur during the first three days, followed by a decrease in the peripheral white cell count (10 days post exposure). The development of a severe leucopenia or an aplastic anaemia is associated with a poor prognosis.

9. *Cholinergic effects*. Absorption of high doses may result in CNS excitation leading to convulsions, followed by CNS depression. Weak cholinergic effects have also be suggested following sulphur mustard exposure.

10. *Cardiac effects*. Cardiac irregularities may occur with atrio-ventricular block and cardiac arrest may follow. Hypotension, refractory to standard resuscitation, has been described on a number of occasions following massive exposure. These rare hypotensive cases have a poor prognosis although the underlying mechanism is not universally accepted.

11. *Overall prognosis*. Sulphur mustard agents are associated with high casualty rates and a significant demand on medical resources. However, overall the fatality is still relatively low with an estimated fatality rate during WW1 of 3-4% compared to a higher conventional fatality rate in a pre-antibiotic era. However, long term effects including pruritus, dry skin, corneal abnormalities and chronic lung disorders such as bronchiolitis obliterans are described.

Note. Exposures in warmer climates may be associated with higher fatality rates or morbity due to higher vapour exposures and respiratory effects.

20.4.3. FIRST AID

First aid consists of:

a. Immediate decontamination of any liquid contamination, as tissue binding and absorption is extremely rapid.

b. Immediate removal, ideally upwind, from the contaminated area as the hazard is persistent.

c. Remove any clothing that may still have any agent on it.

d. During the early stages, there may be no significant skin lesions. However care should be taken not to rub erythematous areas or expose the patient to vibration such as vehicle movement as this may cause a sheering effect and increase blistering (Nikolsky's sign).

e. Blisters should either be left untouched until assessed by medical personnel or dressed where there has been trauma or there is a risk of the blister breaking.

f. Supportive management of any airway problems and seek urgent medical help.

g. Support any breathing difficulty (refer to pulmonary agent first aid).

h. There is no immediate therapy MedCM for first aid providers.

20.4.4. EMERGENCY MEDICAL TREATMENT

1. EMT in the early stages following exposure is unlikely to be required unless casualties are presenting late. Resources should be focused on casualty decontamination and the monitoring for any airway or breathing problems as well as managing any life-threatening trauma. Life-threating conditions related to sulphur mustard exposure may include airway compromise, bronchospasm as well as respiratory and circulatory compromise.

2. Symptomatic treatment may be also required for severe skin and eye pain.

20.4.5. CASUALTY DECONTAMINATION

Exposure to mustard may not be noticed immediately because of the lack of immediate effects and latent period. This may result in delayed decontamination or failure to decontaminate at all and tissue binding occurs within minutes. Specific considerations are:

a. *Decontamination of mucous membranes and eyes*. The substances used for skin decontamination are generally too strong or irritating to be used on mucous membranes and the eyes. In this case the affected tissues should be flushed immediately with water from the water bottle (canteen). The eyes can be flushed with copious amounts of water, or, if available, isotonic sodium bicarbonate (1.26%) or saline (0.9%).

b. *Decontamination of the skin*. Each service person is given the means for immediate decontamination of the skin. The method is usually using physical removal by adsorption or a combination of adsorption and chemical inactivation. Physical adsorption by powders is highly effectively for immediate decontamination. Chemical inactivation may also be achieved by reactive decontaminants. The addition of detergent may make water more effective, but liquid mustard should not be decontaminated with water alone, except for the eyes, as this may spread the agent and increase skin exposure, local effects and systemic absorption.

Note. While chlorine deactivates sulphur mustard at high concentrations, these concentrations are contraindicated for skin.

c. *Additional procedures*. Early decontamination is vital and if within 2 minutes of contact may prevent or greatly reduce clinical effects of sulphur mustard exposure. However, some protection may be provided by late decontamination. Chemical inactivation using chlorination is effective against sulphur mustard but only at levels that are incompatible with skin.

d. *Thickened agents*. In the case of thickened mustard, where the usual procedure is inadequate, the bulk of the agent may have to be scraped off with a knife or similar object. This may be followed by wetting the surface with a cloth drenched in an organic solvent, e.g., petrol (unleaded gasoline) and subsequent application of the usual decontaminating procedure. If water is available in abundant amounts these procedures should be followed by copious rinsing. If the uniform is contaminated it should be removed as soon as possible.

e. *Skin cooling*. Research has shown that skin cooling can reduce the severity of the skin lesions. However, this requires the skin temperature to be reduced to 18°C for several hours and may not be tolerated or achieved operational and for mass casualties. However, this may be achievable for exposed limbs.

20.4.6. TREATMENT OF SKIN LESIONS

1. Due to the lack of definitive treatment, the general principles of medical management are:

   a. Symptomatic relief.

   b. Blister management.

   c. Infection prevention.

   d. Healing promotion.

2. *Symptomatic relief.* It is important to ensure that no remaining contamination is present before commencing treatment. The skin turns red and itches intensely. This itching can be diminished by local applications of cooling preparations, e.g., calamine lotion, corticosteroid preparations or silver sulphadiazine cream. Severe erythema around the genitalia may become quite painful and associated weeping and maceration may occur. Often, treatment with exposure of the damaged area is desirable but care must be taken to prevent secondary infection. Analgesics should be given as required.

3. *Blister management and infection prevention*. Infection is the most important complicating factor in the healing of sulphur mustard burns. Under aseptic conditions, de-roofing of the blisters is preferred enabling the evaluation of the wound bed. However, in the event of a mass casualty incident blisters may be left covered to prevent secondary infections until resources allow definitive treatment. Routine wound inspection aids in the early detection and start of appropriate therapy for any infections.

4. *Healing promotion*. The regeneration of these tissues is very slow, taking from several weeks to several months, much longer than the time required for the restoration of skin destroyed by physical means or by caustic compounds.[2] Healing may result in scarring and fragile skin which may be easily damaged by trauma. Severe long term problems have been reported. More rapid healing has been demonstrated following laser ablation of necrotic tissue, and skin grafting may be required following a period of observation.

20.4.7. TREATMENT OF EYE EFFECTS

1. The main principles for the management of the sulphur mustard eye effects are:

   a. Symptomatic relief.

   b. Infection prevention.

   c. Healing promotion.

   d. Psychological support.

2. *Symptomatic relief*. The effects of mustard on the eyes are very painful. Use of local analgesics may increase corneal damage and are not recommended. Systemic analgesics may therefore be required. The use of cycloplegics can provide significant symptomatic relief

[2] In a review of casualties from the Iran-Iraq conflict, it appeared that the healing process and the final outcome were more dependent on the severity of the initial lesion than on the treatment applied. Overall, superficial lesions heal in 14-21 days while deep lesions may be expected to heal in up to 60 days.

due to ciliary muscle relaxation and pupil dilation. However, care should be taken to prevent sudden exposure to bright lights including sunshine due to the lack of a pupillary response. Topical eye ointment may also provide relief as well as being part of ongoing treatment and prevents the eyelids from sticking together. Do not cover the eyes with a bandage, but if necessary protect them with dark or opaque goggles especially in bright light, and dusty or windy conditions.

3. *Infection prevention*. Secondary infection is a serious complication and increases the amount of corneal scarring. In order to prevent infection, treat with an appropriate broad-spectrum topical antibiotics. When the lesion proves more serious (e.g. blistering of the eyelids, blepharospasm) continuing antibiotic application will be required usually as an ointment (i.e. chloramphenicol eye ointment) and also provides symptom relief and promotes healing.

4. *Healing promotion*. If the eyelids can be separated without too much pain, examine the cornea for possible lesions with fluorescein followed by lavage. Uptake of fluorescein as a green spot indicates a lesion, which, when severe should be referred or discussed with an ophthalmologist as soon as possible. Patients with corneal lesions should receive mydriatics to prevent adhesions between the iris and cornea. In the case of secretions accumulating, the eyes may be irrigated gently with a sterile saline solution and antibiotic ointment may be reapplied.

5. *Psychological support*. Special mention should be made of the psychological effects of eye lesions, even of a mild degree. Severe injuries will cause oedema of the lids, photophobia and blepharospasm which obstruct vision. This will alarm patients and to reassure them, the lids may be gently forced open to assure them that they are not blind.

6. *Prognosis*. Most eye lesions are resolved within 14 days of exposure.

20.4.8. TREATMENT OF RESPIRATORY TRACT LESIONS

1. The main principles for the management of the sulphur mustard respiratory tract effects are:

   a. Symptomatic relief.

   b. Infection prevention and management.

   c. Airway management.

   d. Ventilatory support.

2. *Symptomatic relief*. For mild respiratory tract injury, including hoarseness and sore throat, no specific treatment is required other than symptomatic. A persistent cough may also require symptomatic relief including bronchodilators and cough suppressants, and consideration of anti-reflux treatment. Cough suppressents, such as codeine or equivalent, should however be used with caution. Upper airway symptoms from laryngitis and tracheitis may be managed symptomatically with topical medication, steam or humidified air / nebulisers.

3. *Infection prevention and management*. Following a suspected moderate or severe respiratory tract injury, admission to hospital is recommended and the patient monitored for any airway or respiratory compromise including infection. If a bacterial pneumonia is suspected, isolation of the specific organisms followed by antibiotic sensitivity assays should be performed. Appropriate antibiotic therapy should be based on clinical features, suspected

pathogens as well as taking into consideration any additional features such as immunosuppression.

4. *Airway management*. In cases of severe inhalational exposure, effects on the airway should be suspected. Sulphur mustard can cause pseudomembrane formation that may require repetitive broncho-alveolar lavage. Early symptoms may require further investigation including visualisation of the respiratory tract and where there is a high suspection of airway compromise pre-emptive airway management that includes intubation or surgical airway.[3] Due to further swelling, any airway equipment such as an endotracheal tude should remain uncut.

5. *Ventilatory support*. Follow a severe skin or inhalational sulphur mustard exposure, respiratory compromise should be anticipated. This may be due to the direct or indirect effects on the respiratory tract and lungs due to pulmonary oedema and ARDS. Chest imaging as well as other assessment of respiratory function should be considered. In deteriorating cares, ventilatior support will be required and ventilatory strategies are described in Chapter 7.

6. *Prognosis*. The time course for complete recovery is difficult to define and some of the complications are described below.

20.4.9. TREATMENT OF SYSTEMIC EFFECTS

Every effort should be made to maintain adequate metabolic status and to replace loss of fluids and electrolytes. Infection should be treated promptly and vigorously. The use of colony stimulating factors should be considered to shorten the duration of leucopenia.

20.4.10. LONG TERM EFFECTS AND FOLLOW UP

1. The long term effects of sulphur mustard may be divided into three groups:

   a. Personnel exposed to mustard agents may experience prolonged psychological manifestations including chronic depression, loss of libido and anxiety. This may be a direct effect of the agent or a reactive disorder associated with PTSD.

   b. Local effects of sulphur mustard exposure may include:

      (1) Visual impairment (permanent blindness is extremely rare).

      (2) Scarring of the skin with hyper/hypopigmentation.

      (3) Chronic obstructive airways disease, including bronchiolitis obliterans, chronic bronchitis, emphysema and reactive airways disease.

      (4) Bronchial stenosis.

      (5) Gastrointestinal stenosis with dyspepsia after ingestion of agent.

      (6) Increased sensitivity to sulphur mustard.

   c. Sulphur mustard is a known carcinogen. A follow up study of American soldiers exposed to sulphur mustard during WW1 revealed an increased incidence of lung cancer (and chronic bronchitis) as compared with soldiers who had sustained other

[3] A surgical airway should be considered early before severe ulceration and pseudomembrane formation due to the risk of false tracts and failure.

injuries. A study of British workers involved in the production of sulphur mustard during WW2 revealed no increase in deaths due to cancer amongst those who had died since 1945, but an increase in the prevalence of laryngeal carcinoma amongst those still alive. As some solid tumours take 20 years or more to develop, recent uses of mustard may not have disclosed their long term sequelae yet.

20.5. ARSENICALS – LEWISITE

1. The arsines possessing the $–AsCl_2$ group have both local vesicant and lethal systemic properties. Lewisite (2-chlorovinyl-dichloroarsine) is the best known but also includes ethyldichlorarsine. Lewisite was initially developed in the US during the final stages of WW1, but was not used on the battlefield.

2. *Physical properties.* In a pure form, Lewisite is a colourless and odourless liquid, but usually contains small amounts of impurities that give it a brownish colour and an odour resembling geranium oil. It is heavier than sulphur mustard, poorly soluble in water but soluble in organic solvents.

3. *Chemical properties.* Lewsite in contact with water is hydrolysed at an appreciable rate, forming an oxide that is also a vesicant. In contact with strong alkalis Lewisite is decomposed to non-vesicant products. Oxidizing agents (e.g., hypochlorite, peroxide and nitric oxide) oxidise Lewisite to 2-chloroethenylarsonic acid which is physiologically inactive.

4. *Detection*. The detection of Lewisite is facilitated by the fact that it forms coloured products with many reagents. Colorimetric gas detector tubes are available which react with organic arsenicals.

20.5.1. MECHANISM OF ACTION

Due to its physical and chemical properties, Lewisite can easily penetrate the skin, where it exerts its vesicant action. It can spread through the whole body and act as an arsenical poison. It has been shown that Lewisite inhibits a great number of enzymes rich in SH-groups. Lipoic acid is an essential part of the pyruvate dehydrogenase system, acting as a co-enzyme in the formation of acetyl-CoA from pyruvate. Lewisite is thought to interact with lipoic acid to form a cyclic compound, thereby interfering with energy production within the cells. Inhibition of the pyruvate dehydrogenase system is a property common to all trivalent arsenic compounds causing multiple systemic effects.

20.5.2. MEDICAL EFFECTS

1. *Eyes*. Liquid arsenical vesicants cause severe damage to the eye. On contact, pain and blepharospasm occurs instantly. Oedema of the conjunctivae and lids follow rapidly and close the eye within an hour. Inflammation of the iris usually is evident by this time. After a few hours, the oedema of the lids begins to subside, while haziness of the cornea develops and iritis increases. The corneal injury, which varies with the severity of the exposure, may heal without residual effects, induce pannus formation or progress to massive necrosis. The iritis may subside without permanent impairment of vision, if the exposure was mild. After heavy exposure, hypopyon may ensue, terminating in necrosis, depigmentation of the iris and synechia formation. Liquid arsenical vesicants instantly produce a grey scarring of the cornea, like an acid burn, at the point of contact. Necrosis and separation of both bulbar and palpebral conjunctivae may follow very heavy exposure. All injured eyes are susceptible to secondary infection. Mild conjunctivitis due to arsenical vesicants heals in a few days without specific treatment. Severe exposure may cause permanent injury or blindness.

2. *Skin*. Liquid arsenical vesicants produce more severe lesions of the skin than liquid mustard. There is full thickness injury to the skin and burns may penetrate to connective tissue and muscle and cause greater vascular damage and more severe inflammatory reaction than in sulphur mustard burns. In large, deep, arsenical vesicant burns, there may be considerable necrosis of tissue and gangrene. Despite the overall severity of these skin lesions the spontaneous rate of healing is considerably faster than that of comparable sulphur mustard burns. Exposure of the skin is followed shortly by erythema, then by blistering which tends to cover the entire area of erythema. The surrounding halo of erythema is less noticeable than with sulphur mustard blisters, although the two are often indistinguishable. The yellowish blister fluid is slightly more opaque due to the presence of more inflammatory cells.

Note. It has been shown that blister fluid contains hydrolysis products which may present a further vesicant risk to the patient if this blister fluid remains in contact with normal skin. Standard clinical protective measures should prevent injury to health care providers when dealing with these patients.

3. *Pain*. Stinging pain may be felt in 10 to 20 seconds after contact with liquid arsenical vesicants. The pain increases in severity with penetration and in a few minutes becomes a deep, aching pain. Pain on contact with liquid arsenical vesicants usually gives sufficient warning so that decontamination may be begun promptly and deep burns thus avoided in conscious victims. After about 5 minutes of contact, there appears a grey area of dead epithelium resembling that seen in corrosive burns. Erythema is like that caused by sulphur mustard but is accompanied by more pain. Itching and irritation persist for only about 24 hours whether or not a blister develops. Blisters are often well developed in 12 hours and are painful at first, in contrast to the relatively painless sulphur mustard blister. After 48 to 72 hours, the pain lessens.

4. *Respiratory tract*. The vapours of arsenical vesicants are so irritating to the respiratory tract that conscious casualties will immediately put on a respirator to avoid the vapour. No severe respiratory injuries are likely to occur except among the wounded who cannot put on their respirator and those without respiratory protection. The respiratory lesions are similar to those produced by sulphur mustard except that in the most severe cases, pulmonary oedema may be accompanied by pleural effusion.

5. *Systemic effects*. Liquid arsenical vesicants on the skin, as well as inhaled vapour, are absorbed and may cause systemic poisoning. A manifestation of this is a change in capillary permeability, which permits loss of sufficient fluid from the bloodstream to cause haemoconcentration, shock and death. In non-fatal cases, haemolysis has occurred with a resultant haemolytic anaemia. The excretion of oxidised products into the bile by the liver produces focal necrosis of that organ, necrosis of the mucosa of the biliary passages with peri-biliary haemorrhages and some injury of the intestinal mucosa. Acute systemic poisoning from large skin burns causes pulmonary oedema, diarrhoea, restlessness, weakness, subnormal temperature and hypotension. Some symptoms associated with arsenic poisoning, such as nephritis with proteinuria and neuropathy may occur.

20.5.3. FIRST AID

First aid is the same as for sulphur mustard agent although decontamination of skin will be precipitated by immediate symptoms. There is no immediate systemic therapy for first aid providers. Topical eye ointment should ideally be applied as soon as possible to reduce the effects of exposure. Because of the shorter latent period, airway and breathing symptoms including hoarse voice, difficulty in swallowing and shortness of breath may present more early.

20.5.4. TRIAGE

The triage of Lewisite casualties include criteria for early antidote treatment or serious local effects to the eyes as well as the supportive criteria used for sulphur mustard.

20.5.5. EMERGENCY MEDICAL TREATMENT

1. EMT in the early stages following arsenical exposure may require treatment due to the short onset and potentially lethal effects. Systemic effects may include airway, breathing and circulatory complications requiring fluid replacement, early advanced airway management and respiratory support at hospital due to pulmonary effects.

2. The skin lesions may be very painful and analgesia may allow more rapid decontamination and medical evacuation. Symptomatic treatment may be required for eye pain including the application of dimercaprol eye ointment (see below), where available.

3. Casualties with early systemic features associated with local effects including hypotension (unresponsive to fluid resuscitation) or peripheral oedema should be considered for early chelation therapy. In the pre-hospital environment, this may require intramuscular dimercaprol (see below).

20.5.6. CASUALTY DECONTAMINATION

Casualty decontamination follows the same principles as for sulphur mustard with the exception of the availability of topical antidote for the eyes (see below).

20.5.7. TREATMENT OF LEWISITE EXPOSURE

1. *Supportive therapy*. The systemic effects of Lewisite will cause severe and life-threatening organ dysfunction and failure. These include hypotension due to distributive shock and fluid loss and respiratory failure due to local and systemic effects. The airway may also be compromised if Lewisite was inhaled. Maintenance of metabolic status and replacement of fluids and electrolytes is important, particularly in the case of hypovolaemic shock complicating severe exposure. The specific haematological hepatic and renal effects arising from systemic poisoning by arsenical compounds such as Lewisite may require specialist and, possibly intensive, medical management. Renal failure should be anticipated and will require renal replacement therapy, this is unlikely to be immediately available in a deployed field hospital and may require STRAT MEDEVAC or a deployed renal capability. The following indications are given as a guide for the use of systemic antidote treatment:

   a. Cough with dyspnoea and frothy sputum, which may be blood tinged and other signs of pulmonary oedema.

   b. Skin burn the size of the palm of the hand or larger, caused by a liquid arsenical blister agent which was not decontaminated within the first 15 minutes.

   c. Skin contamination by a liquid arsenical vesicant covering 5% or more of the body surface, in which there is evidence of immediate skin damage (grey or dead-white blanching of the skin), or in which erythema develops over the area within 30 minutes.

2. *Antidote therapy*. The toxicity of Lewisite and other arsenicals is due to the heavy metal arsenic. The chemical properties of this element can be exploited and elimination from the body can be achieved by the use of chelating agents. Chelating agents available include:

a. Dimercaprol (also known as British Anti-Lewisite (BAL)).

b. Dimercaptosuccinic acid (DMSA) (also known as Succimer, 'Chemet').

c. 2,3-Dimercapto-1-propanesulfonic acid (DMPS) (also known as Unithiol, DIMAVAL).

3. While dimercaprol is the traditional antidote in many nations. The latter two antidotes have fewer side effects than BAL without loss in efficacy. DMSA is available as an oral preparation. DMPS is available in injectable and oral formulations. Where BAL may be given during the initial stages of therapy, the patient should then be converted to oral therapy.

Note. DMPS and BAL should not be used for acute arsine poisoning.

20.5.8. DIMERCAPROL (BAL)

1. *Mechanism of action*. Dimercaprol is a yellow viscous liquid usually in peanut oil. It can combine with arsenic forming a water soluble complex that can be excreted. With arsenicals, the complex formed possesses a pentagon with two carbon atoms, two sulphur atoms and one arsenic atom at the corners. This is the same mechanism by which Lewisite blocks two adjacent SH groups of pyruvate dehydrogenase system. The therapeutic action of dimercaprol can thus be explained by the law of mass action. Dimercaprol provides a great number of adjacent SH groups that displace the arsenic bound to the target enzymes. The enzymes are reactivated and can resume their normal biological activity.

Note: Dimercaprol is formulated in peanut oil and as such should not be given to patients with nut allergies.

2. *Dosage*. The usual dosage of BAL is as follows. 3 mg/kg intramuscularly repeated every 4 hours for 2 days and then every 12 hours for 7-10 days. Administration must be by deep intramuscular injection with special attention being given to aseptic technique. In the most severe and life-threatening cases, an initial dose of 5mg/kg could be considered but with a higher association with side effects.[4]

3. *Side effects*. BAL injections are painful and may result in tissue necrosis at the injection site. Dimercaprol when given by injection may produce significant reactions especially above 3mg/kg. The effects usually last less than one hour and include:

a. Increased systolic and diastolic pressure.

b. Tachycardia.

c. Nausea and vomiting.

d. Headache.

e. Burning sensation of lips.

f. Feeling of constriction of the chest.

g. Conjunctivitis.

[4] Dosing regimens will vary between nations. Early transfer to other intravenous agents or oral medication (DMSA) is recommended once the patient is able to swallow and gastrointestinal effects have settled.

h. Lachrymation.

i. Rhinorrhoea.

j. Sweating.

k. Anxiety and unrest.

4. *Dimercaprol eye ointment*. Dimercaprol eye ointment may diminish the effects of Lewisite if applied within 2-5 minutes of exposure. In severe cases, the systemic use of morphine may be necessary for control of pain. When the conjunctival oedema subsides enough to permit ophthalmic examination, the cornea should be stained with fluorescein to detect erosions and the iris should be examined for iritis. Mydriatics should be instilled in all cases with corneal erosions, iritis or with marked photophobia or painful miosis. Antibiotics may be used to combat infection. Sterile petroleum jelly applied to the lid margins will help prevent their sticking together. Irrigations of the eye should be copious, employing isotonic solutions. Occlusive dressings or pressure on the globe must be avoided.

5. *Skin treatment*. Dimercaprol ointment may be applied to skin exposed to Lewisite before actual blistering has begun, but application after this time may be of little use. BAL ointment is spread on the skin in a thin film and allowed to remain at least 5 minutes. Occasionally, BAL ointment causes stinging, itching or urticarial wheals. This condition lasts about an hour and should not cause alarm. Mild dermatitis may occur if BAL ointment is frequently applied on the same area of skin; hence, this property precludes its use as a protective ointment. Dimercaprol is chemically incompatible with silver sulphadiazine and the two should not be used together.

20.5.9. DIMERCAPTOSUCCINIC ACID (DMSA / SUCCIMER)

1. *Mechanism of action*. DMSA is an oral chelating agent and analogue of dimercaprol and is usually used for lead chelation especially in children.

Note: The use for arsenic poisoning has not been confirmed due to lack of data.

2. *Dosage*. The administration is based on a daily dose of 10mg/kg (or 500mg) usually administered as 100mg tablets given every eight hours for the first 5 days followed by every twelve hours for two weeks.

3. *Side effects*. Side effects include nausea, vomiting, decreased appetite, diarrhoea, metallic taste in the mouth, drowsiness, dizziness, watering eyes, or headache as well as allergic reaction, skin rash and mouth sores.

20.5.10. 2.3-DIMERCAPTO-1-PROPANESULFONIC ACID (DMPS / UNITHIOL)

DMPS is a chelating agent available in intravenous and oral form. It is a water soluble analogue of BAL and similar pharmacokinetics and side effects profile as DMSA. There are a number of dosing regimens and national guidelines should be consulted.

20.5.11. PROGNOSIS

The long term effects of exposure to Lewisite are unknown but systemic effects can be extrapolated from acute arsenic poisoning. Even if the patient survives the acute effects, the prognosis must be guarded for several weeks and may need critical care and organ support. The treatment of the erythema, blisters and denuded areas is identical with that for similar sulphur mustard lesions. A severe full thickness burn involving a large surface area is similar

to a thermal injury and must be managed by intravenous fluid replacement to correct potential hypovolaemic shock. Morphine and splinting of the affected parts may be necessary to relieve pain. Lewisite burns may be deeper and more severe than those seen with sulphur mustard and may require long term plastic surgery follow up. Eye injuries may lead to permanent blindness and urgent ophthalmology follow-up is highly recommended.

20.6. HALOGENATED OXIMES – PHOSGENE OXIME

1. The urticarial properties of the halogenated oximes were discovered long before WW2. The most commonly known in this series is phosgene oxime (CX) (CCl_2-NOH).

Note: Phosgene oxime should not be confused with either the pulmonary agent phosgene or the oxime group of AChE-reactivators.

2. *Physical properties*. Phosgene oxime is a white crystalline powder. It melts between 39-40°C, and boils at 129°C. By the addition of certain compounds it is possible to liquefy phosgene oxime at room temperature. It is fairly soluble in water and in organic solvents. In aqueous solution phosgene oxime is hydrolysed fairly rapidly, especially in the presence of alkali. It has a high vapour pressure, and its odour is very unpleasant and irritating. Even as a dry solid, phosgene oxime decomposes spontaneously and has to be stored at low temperatures.

3. *Mechanism of action.* The exact mode of action is not known.

4. *Clinical effects*. The characteristic signs and symptoms of phosgene oxime exposure may suggest its use. There is no clinical experience with casualties arising from this agent and hence accurate prognosis is uncertain.

a. *Low concentration exposure*. Phosgene oxime severely irritates the eyes and respiratory tract possible due to interaction with moist tissues.

b. *High concentration exposure*:

(1) *Skin*. Skin effects are seen with high concentrations. The action on the skin is immediate: phosgene oxime provokes irritation resembling that caused by a stinging nettle. A few milligrams cause intense pain which radiates from the point of application, within a minute the affected area turns white and is surrounded by a zone of erythema which resembles a wagon wheel in appearance. In 1 hour, the area becomes swollen and within 24 hours the lesion turns yellow and blisters appear. Some days later the area shows desquamation with necrosis of the skin followed by crust formation and a purulent discharge or necrotising wound.

(2) *Eyes*. At high concentrations, phosgene oxime causes corneal lesions and blindness.

(3) *Lungs*. Phosgene oxime may also act as a pulmonary agent.

c. Systemic toxicity has been described from parenteral absorption.

20.6.1. CASUALTY DECONTAMINATION

Chemical inactivation using alkalis is effective, whereas chlorination is ineffective against phosgene oxime. The eyes should be flushed immediately using water or isotonic sodium

bicarbonate solution if available. Physical removal of the agent should be carried out as soon as possible.

20.6.2. GENERAL TREATMENT

There is no antidote available. Treat as any other ulcerated necrotic skin lesion (e.g., thermal burn) with due consideration of other supportive measures as well as anti-urticarials. Systemic analgesia may be required. Pulmonary oedema should be treated appropriately.

SULPHUR MUSTARD AGENTS

Yellow to brown liquid. Smell of garlic or horseradish.

MECHANISM OF ACTION

Sulphur mustard and the nitrogen mustards are alkylating blistering agents causing damage to DNA and other biological molecules resulting in cell damage and death. The effects of sulphur mustard is most prominent at the point of exposure with rapid absorption. Symptoms are delayed (12-24 hours) but the onset time is related to the concentration, duration of exposure and type of skin and moisture.

Progression of symptoms:

Eye irritation (3-6 hours) ▸ Skin erythema (6-24 hours) ▸ Blistering (12-48 hours)

QUICK LOOK (CRESS)

Conscious	Respiration	Eyes	Secretions	Skin	Other
Normal	Normal or increased	Inflamed	Normal or mildly increased	**Inflamed (reddened) → Blisters**	Delayed onset, relatively painless

SELF / FIRST AID

Immediate pain – consider Lewisite or caustic agent (acid / alkali)
Delayed redness (6-12 hours) – consider sulphur mustard
Remove from scene and immediate decontamination drills
Monitor exposed area for redness / irritation, especially eyes / airway
Report any difficulty with breathing or swallowing, incl hoarse voice or cough

CASUALTY DECONTAMINATION

Liquid and vapour hazard.
Decontamination as soon as possible.
Use of adsorbents and/or active decontaminants.

TRIAGE CATEGORIES

T1 (Severe)	T2 (Moderate)	T3 (Mild)
Airway compromise, respiratory distress, hypotension, > 25% BSA.	Not walking. Burn surface area (sulphur mustard 10-25%), airway irritation, hoarse voice, cough; eye pain AND reddening.	Walking. Erythema. Eye pain.

EMERGENCY MEDICAL TREATMENT

Supportive management:

Airway: Airway manoeuvres, suction (late indication).
Breathing: Oxygen, if hypoxia (late indication). Bronchodilators.
Circulation: Consider fluid therapy, if fluid loss due to blisters (unlikely in first 24 hours).
General: Analgesia
Eyes: Consider cycloplegics or mydriatics if eye pain or blepharospasm. Avoid bright light.

MedCM / Antidotes:

There are no specific MedCM for sulphur / nitrogen mustard.

Clinical Investigations:	Blister fluid, blood, tissue and urine for sulphur / nitrogen mustard, hydrolysis products and DNA/protein adducts.

ADDITIONAL INFORMATION

Eye symptoms and especially temporary blindness will have a significant psychological impact and patients should be reassured of a good prognosis in most cases.
The management of blisters and bullae is controversial and early de-roofing under aseptic conditions should be considered if risk of trauma to blister. The blister fluid is not toxic but is a biohazard.

LEWISITE (ARSENICALS)

Yellow to brown liquid. Smell of geraniums.

MECHANISM OF ACTION

Toxic effects are due to the presence of $-AsCl_2$ group including systemic arsenic poisoning. The mechanism is due to the interference of pyruvate dehydrogenase and cellular energy production. This leads to multiple systemic effects including local tissue necrosis (skin, eye, mucosa and respiratory tract), increase in capillary permeability, distributive hypovolemic shock, haemolysis, pulmonary oedema, nephritic and polyneuropathy.
Skin effects are immediate with pain followed by erythema and tissue necrosis.

QUICK LOOK (CRESS)

Conscious	Respiration	Eyes	Secretions	Skin	Other
Normal	Increase (if inspired)	Painful, inflamed	Normal or mildly increased	Early pain and erythema, silvery, grey lesions	Systemic features (see above)

SELF / FIRST AID

Immediate decontamination
Remove from scene
Remove any clothing with gross liquid decontamination
Monitor for local lesions and suggestion of systemic absorption
No immediate first aid treatment
Seek immediate medical care

CASUALTY DECONTAMINATION

Liquid and vapour hazard.
Decontamination as soon as possible.
Use of adsorbents and/or active decontaminants.

TRIAGE CATEGORIES

T1 (Severe)	T2 (Moderate)	T3 (Mild)
Respiratory distress. Cough (±blood). Blistering. Signs of systemic toxicity or organ failure. Erythema > 5%	Not walking. Airway irritation, hoarse voice, cough; eye pain AND reddening.	Walking. Reddened skin, eye pain.

EMERGENCY MEDICAL TREATMENT

Supportive management:
Airway: Airway manoeuvres, suction (late indication).
Breathing: Oxygen, if hypoxia. Bronchodilators.
Circulation: Consider fluid therapy, if fluid loss due to blisters (unlikely in first 24 hours).
Analgesia.
Eyes: Consider topical BAL, cycloplegics or mydriatics if eye pain or blepharospasm. Avoid bright light.

MedCM / Antidotes:
Dimercaprol (*British Anti-Lewisite*). 3mg/kg i.m. every 4 hours for first 2 days, then 12 hours for next 10 days.
Note. Beware of allergies as in peanut oil.
Alternatives: Succimer (DMSA) 10mg/kg initially, *Unithiol* (DMPS) may also be available.
Indications:
Cough, pulmonary oedema, respiratory distress.
Skin burn or erythema >5%. Systemic features.

Clinical Investigations:	Blister fluid (may contain arsenic); blood arsenic levels; urine. Full blood count. Chest radiograph, venous (arterial) blood gas, lactate, renal and liver baseline.

ADDITIONAL INFORMATION

Initial management should not be delayed and may require parenteral administration.
Seek early toxicological advice due to complex chelating therapy and adverse drug reactions with dimercaprol.
Early conversion to safer oral agents is recommended, although limited evidence for oral administration.
Warning: Blister fluid may contain arsenic.

PHOSGENE OXIME (HALOGENATED OXIMES)

White crystalline powder. Melts at ~40°C

MECHANISM OF ACTION

The mechanism of action is not fully understood. The action on the skin is immediate: phosgene oxime provokes irritation resembling that caused by a stinging nettle followed by intense pain which radiates from the point of application. Visual effects include the affected area turning white and surrounded by a zone of erythema. At one hour, the area becomes swollen and within 24 hours the lesion turns yellow; blisters then appear. Some days later the area shows desquamation with necrosis of the skin followed by crust formation and a purulent discharge. Phosgene oxime affects the eyes, causing corneal lesions and blindness and may affect the respiratory tract causing pulmonary oedema.

QUICK LOOK (CRESS)

Conscious	Respiration	Eyes	Secretions	Skin	Other
Normal	Increase (if inspired)	Painful, inflamed	Normal or mildly increased	Early intense pain and appearance as above	

SELF / FIRST AID

Remove from scene
Immediate decontamination including rinsing with water
Give pain relief
Observe

CASUALTY DECONTAMINATION

Liquid and vapour hazard.
Decontamination as soon as possible.
Use of adsorbents and/or active decontaminants.

TRIAGE CATEGORIES

T1 (Immediate)	T2 (Urgent)	T3 (Delayed)
Respiratory distress or arrest.	Non-ambulatory. Severe pain.	Walking.

EMERGENCY MEDICAL TREATMENT

Supportive management:

Airway: Airway manoeuvres, suction.
Breathing: Oxygen, if hypoxia. Bronchodilators.
Circulation: Consider fluid therapy, if fluid loss due to blisters (unlikely in first 24 hours).
Analgesia.
Eyes: Consider cycloplegics or mydriatics, if eye pain or blepharospasm. Avoid bright light.

MedCM / Antidotes:

No MedCM available.

Clinical Investigations:	

ADDITIONAL INFORMATION

Monitor for any systemic effects.

CHAPTER 21: PULMONARY (CHOKING) AGENTS

21.1. INTRODUCTION

1. Lung damaging agents are chemical agents that produce a toxic inhalational injury, damaging lung tissue and primarily causing pulmonary oedema and other effects. Whether produced for military or industrial use, these chemical agents pose a very real threat to military personnel.

2. *Traditional warfare agents*. The term choking agents has been traditionally applied to the use of certain lung damaging agents as chemical weapons, and includes:

a. Phosgene (CG).

b. Chlorine (CL).

c. Chloropicrin (PS).

d. Diphosgene (DP).

3. Phosgene accounted for 80% of all chemical fatalities in WW1, but at least 14 different respiratory agents were used, as well as obscurants (smokes), harassing agents (chloracetone), and vesicants (sulphur mustard) that could also cause pulmonary injury.

4. *Toxic industrial chemicals*. Today, only a handful of such pulmonary toxicants still exist in stockpiles around the world. However many, such as chlorine and phosgene, are currently produced in large quantities for industrial purposes, as are other toxic industrial chemicals, such as:

a. Ammonia.

b. Isocyanates.

c. Mineral acids.

5. *Other inhaled agents of operational significance*. Other lung damagers, while not likely to be used as agents, are still of operational significance:

a. *Perfluoroisobutylene* (PFIs). This is a toxic pyrolysis product of tetrafluoroethylene polymers encountered in military materiel (e.g. Teflon® found in the interior of many military vehicles). Perfluoroisobutylene (PFIBs is said to be ten times more toxic than phosgene).

b. *The oxides of nitrogen* are components of blast weapons or may be toxic decomposition products.

c. *Military smokes* may contain toxic compounds that cause the same effects as phosgene if used in confined spaces (see Chapter 25).

6. *Non-chemical agents*. As well as chemical agents, biological agents either BWA or endemic may cause respiratory symptoms.

a. *Inhaled biological toxins*. The effects of some toxins especially those that are cytotoxic (ricin, abrin, SEB) are related to the dose and therefore the peak concentration

will be at the site exposed. Inhalation of these toxins may mimic a pulmonary agent with no immediate effects and delayed onset with potential pyrexia (ricin).

b. *Live biological agents*. The site of effect for some biological agents depends on the route of exposure. This applies to aerosolised BWA such as *B.anthracis* (inhalational anthrax) and *Y. pestis* (pneumonic plague), both of which have several forms of infection depending on the route of infection. The incubation period and associated pyrexia reduces the chance of misdiagnosis. However, the onset of some viral respiratory infections such as influenza pneumonitis may overlap with the delayed onset of some pulmonary agents and the inhaled toxins.

c. *Radiation*. At extremely high doses of radiation, there is an associated pulmonary syndrome due to radiation-induced pulmonary fibrosis however this is associated with fatal doses of > 8Gy (see Part 5).

21.2. PHYSICAL AND CHEMICAL PROPERTIES

1. Military dispersion of phosgene during WW1 followed the explosion of liquid filled shells with subsequent rapid evaporation and formation of a white cloud due to its slight solubility in an aqueous environment. It spontaneously converted to a colourless, low-lying gas four times as dense as air. Because of its relatively low boiling point (8.2^{o}C), phosgene was often mixed with other substances. Chlorine was released from pressurised cylinders to form a pungent greenish-yellow gas that was heavier than air causing accumulation in low lying places such as trenches and basements.

2. Chemicals that are highly reactive and/or highly soluble in aqueous solutions tend to act in the upper or central part of the respiratory tract (trachea). Irritants such as sulphur mustard, ammonia and hydrochloric acid, when inhaled, cause pronounced irritation of the epithelial cells lining the upper airway. At low concentrations, these reactive gases and vapours react and are removed in the central conducting part of the airway before they reach the lower portion of the respiratory tract. The particle size of aerosols will determine the site of deposition within the respiratory tract. This effect is used to prevent or treat bronchospasm in the most reactive part of the respiratory tract using inhaled pharmaceuticals with radii of 5µm.

3. Unreactive and water-insoluble gases and vapours, as well as aerosols less than 1µm (such as phosgene, oxides of nitrogen and PFIB) will enter the lower respiratory tract including the respiratory bronchioles and alveoli. They will have the potential of interacting directly with pneumocytes.

4. Following exposure to high concentrations of airway irritating agents such as chlorine, sufficient agent may penetrate into the lower respiratory tract and cause pulmonary oedema. Similarly, high concentrations of phosgene can cause significant airway irritation in the upper airway.

5. *Detection*. Some field detection equipment for classical pulmonary agents is available. The characteristic odour of some pulmonary agents is unreliable as a method of detection. For example, in low concentration phosgene has a smell resembling new mown hay, but the odour may be faint or lost after accommodation. There is also considerable variation in the sense of smell between individuals. Similarly the eye irritation, coughing, sneezing, hoarseness, and other central respirator“ effects seen after exposure to high concentrations of some pulmonary toxicants are also unreliable indicators of exposure, as these may be transient or entirely absent at lower but still potentially lethal concentrations. This is particularly true in the case of phosgene.

21.3. ROUTES OF EXPOSURE

1. The principle route of exposure for pulmonary agents is inhalation although some chemical agents have an indirect action on the lungs. Any CBRN agent that causes an overwhelming systemic inflammatory reporting such as SIRS, may trigger the associated respiratory complication of acute respiratory distress syndrome (ARDS). Although not a chemical agent, the herbicide paraquat when ingested has a very specific respiratory effect causing severe pulmonary fibrosis. This is complicated by the fact that this effect is enhanced by high concentration oxygen by the formation of free radicals.

2. In addition, some pulmonary agents may cause non-respiratory effects such as chemical burns to the skin. Where agents are compressed as a gas, release and direct contact with the skin may cause cold injury. Some non-pulmonary agents such as the vesicants (sulphur mustard and Lewisite) will at high concentrations cause direct pulmonary effects; the lungs also being an effective route for systemic absorption.

3. *Protection*. The warfare canisters of respirators provide protection from traditional chemical warfare agents such as pulmonary agents, either by adsorption or chemical reaction. However only limited or temporary protection against high concentrations of TICs and products of combustion can be assumed including PFIB, and are not to be used in oxygen-depleted or carbon monoxide environments. Self-contained breathing apparatus should therefore be worn by responders, where appropriate.

21.4. MECHANISM OF ACTION

1. Chemically-induced acute lung injury involves a permeability defect in the blood-air barrier (the alveolar-capillary membrane). However, the precise mechanism of toxicity remains largely unknown. Leakage of fluid from capillaries into the pulmonary interstitium is normally opposed by lymphatic drainage from the parenchyma. However, as the fluid leakage increases, normal drainage mechanisms become progressively overwhelmed. After an asymptomatic or latent period (20 minutes to 24 hours), depending on the exposed dose, fluid leakage into the pulmonary interstitium decreases compliance, producing a stiff lung and increasing complaint of tight chest, shortness of breath and dyspnoea. Fluid eventually invades the alveoli and produces clinically evident pulmonary oedema.

2. *Predisposing factors*. Important predisposing factors are/include:

   a. Pre-existing airway damage (such as that caused by prior exposure to a lung damaging agent) may seriously compromise the respiratory system's normal protection and clearance mechanisms. Cigarette smoking may severely compromise airway function with respect to both airway patency and clearance mechanisms.

   b. Hyper-reactive airways (asthma in varying degrees) are seen in up to 15% of the adult population. Exposures to pulmonary intoxicants may trigger bronchospasm in these individuals. This bronchospasm may delay the clearance of the agent, interfering further with gas transport. The development of an acute interstitial process (e.g., phosgene-related pulmonary oedema) may also trigger bronchospasm. Individuals with any of the following characteristics should be considered likely to develop bronchospasm as the result of a exposure to lung damaging agents:

      (1) Prior history of asthma or hay fever (even as a child).

      (2) Prior history of eczema.

(3) Family history of asthma, hay fever, or eczema.

(4) History of chronic sinusitis or seasonal rhinitis.

21.5. PHOSGENE (CG) AND DIPHOSGENE (DP)

1. Phosgene (carbonyl chloride – CCl_2O) is a widely used TIC. It has a smell described as freshly mown hay. The odour threshold for phosgene is about 1.5 mg/m^3, and phosgene irritates mucous membranes at 4 mg/m^3.

2. The outstanding feature of acute lung injury caused by phosgene is massive pulmonary oedema. This is preceded by damage to the bronchiolar epithelium, development of patchy areas of emphysema, partial atelectasis and oedema of the perivascular connective tissue. The trachea and bronchi are usually normal in appearance. This contrasts with the findings in chlorine and chloropicrin poisoning in which both structures may show serious damage to the epithelial lining with desquamation. The lungs are large, oedematous and darkly congested. Oedema fluid, usually frothy, pours from the bronchi and may be seen escaping from the mouth and nostrils. With exposure to very high concentrations, death may occur within several hours; in most fatal cases pulmonary oedema reaches a maximum in 12 hours, followed by death in 24 to 48 hours. If the casualty survives, resolution commences within 48 hours and, in the absence of complicating infection, there may be little or no residual damage.

3. The delayed onset in some cases of prolonged low (asymptomatic) concentrations suggests there may be multiple mechanisms of action including an inflammatory response and ARDS. This may be potentially exploited for future post-exposure prophylaxis including corticosteroids and anti-oxidants.

4. *Diphosgene (DP)*. Diphosgene ($ClCO_2CCl_3$) is an unstable agent that hydrolyses to form phosgene and a chloroform molecule. The latter is at an insignificant concentration to be effective on the battlefield.

21.6. CHLORINE (CL)

1. Chlorine is a widely available TIC and was the first chemical agent to be used in WW1. It is highly soluble in water and forms hydrochloric acid and hypochlorous acid. This gives rise to immediate local effects to the eyes, upper airway and central portion of the respiratory tract. Therefore, the effectiveness as a weapon depends on the ability to concentrate enough gas to cause an effect before personnel can take immediate actions such as withdrawal and donning respiratory protection.

2. On the battlefield in WW1, industrial quantities of chlorine where released during an attack causing mass casualties and fatalities hundreds of metres away from the release point. The mild effects were experienced several kilometres away. The effects of chlorine during trench warfare were potentiated by the fact that chlorine is heavier than air. Opportunistic use of chlorine is possible but will lead to very localised fatal effect although mild or incapacitating symptoms may be experienced some distance away, especially downwind or in low lying structures.

3. The severity of the effects of chlorine is directly related to the concentration and duration of exposure. Due to the irritation of the eyes and airway at low concentrations, it is unlikely a person will be unaware that they are being exposed and so a high Ct due to an insidious exposure is unlikely to occur in the field. However, in enclosed environments, exposure doses may be significant.

21.7. CHLOROPICRIN (PS)

Chloropicrin is a fumigant that has been used as a chemical warfare agent in WW1. It is relatively insoluble in water and although less toxic than phosgene, it has a more irritating and incapacitating effect. Incapacitating effects include lacrimation and vomiting, and skin irritation. At high levels of exposure, it will cause pneumonitis and pulmonary oedema.

21.8. MEDICAL EFFECTS

1. The effects of pulmonary agents can be divided into:

   a. Immediate effects that may allow a person to take action such as withdrawal or donning respiratory protection.

   b. Latent period with a length dependent on inhaled dose and other factors.

   c. Clinical effects.

   d. Complications and death.

2. *Immediate effects*. Exposure to high concentrations of pulmonary agent may irritate moist mucous membranes, depending on their reactivity and solubility in water. Transient burning sensation in the eyes with lacrimation may coexist with early onset cough and a sub-sternal discomfort with a sensation of pressure. These effects may be enough to cause incapacitation. Irritation of the larynx by high concentrations of the agent may lead to sudden laryngeal spasm and death. Irritation of lower respiratory tract especially in those with a predisposition may precipitate bronchospasm.

3. *Latent period*. Clinical effects follow a latent period of variable length (20 minutes to 48 hours, if considering all pulmonary agents) that depends primarily on the intensity of exposure and partly on the physical activity of the exposed individual; this is particularly true for phosgene. After the latent period, the patient experiences worsening respiratory distress that at first is unaccompanied by objectively verifiable signs of pulmonary damage, but may progress to pulmonary oedema and death.

4. *Clinical effects*. The most prominent symptom following the clinical latent period is dyspnoea, perceived as shortness of breath, with or without chest tightness, and in the initial stages there may not be an objective sign of pulmonary damage. These sensations reflect hypoxemia, increased ventilatory drive, and decreased lung compliance, all of which result from the accumulation of fluid in the pulmonary interstitium. Fine crackles appear at the lung bases, but these may not be clearly heard unless auscultation is conducted after a forced expiration. Later, auscultation reveals coarse crackles and râles in both lung fields, and increasing quantities of thin, watery secretions are noted.

5. *Severe features*. The build-up of fluid in the lungs has two clinically pertinent effects.

   a. First, developing pulmonary oedema interferes with oxygen delivery to alveolar capillaries leading to hypoxemia. If there is oxygen desaturation, cyanosis will become apparent.

   b. Secondly, the sequestration of plasma-derived fluid in the lungs (up to one litre per hour) may lead to hypovolemia and hypotension. This is important when considering

therapy and the use of non-invasive ventilation that may further adversely affect cardiovascular status.

6. *Outcome and death*. The development of symptoms and signs of pulmonary oedema within four hours of exposure is an indicator of a poor prognosis; in the absence of immediate respiratory support. Death results from respiratory failure, hypoxemia, hypovolemia, or a combination of these conditions. Hypoxia and hypotension may progress particularly rapidly and suggest a poor prognosis. Complications include infection of damaged lungs and delayed deaths following such respiratory infections.

21.9. PULMONARY AGENT TREATMENT

21.9.1. FIRST AID

First aid consists of:

a. Immediate removal, ideally upwind.

b. If the primary hazard is below toxic levels, respiratory protection may be removed and treatment started on site.

c. Decontamination is less important for pulmonary agents. Remove any clothing that may still have any liquid agent on it.

d. For eye symptoms, irrigate the eye with copious amount of water.

e. Prevent exposed persons from doing any exertion.

f. Avoid oxygen unless the patient is cyanosed. This conserves oxygen supplies and limits any potential free radical damage.

g. If patient develops cough, wheezing or other breathing difficulties, seek immediate medical care.

h. Report any immediate signs and symptoms.

21.9.2. POST-EXPOSURE PROPHYLAXIS

There are no specific MedCM for pulmonary agents. However, administration of inhaled corticosteroids may be beneficial and should be considered.

21.9.3. EMERGENCY MEDICAL TREATMENT

1. Treatment is symptomatic and includes the use of inhalers (with or without spacer devices), nebulised bronchodilators and fluid replacement.

2. Oxygen should be reserved for symptomatic and severe casualties including those with reduced saturation (<93%) and cyanosis.

3. Diuretics and nitrates should be avoided as the principle mechanism is non-cardiogenic.

4. Where it is available, patients with refractory hypoxia should be considered for continuous positive airway pressure (CPAP) respiratory support. Care should be taken to

ensure casualties are well hydrated as high thoracic pressures reduce venous return and can cause circulatory compromise especially if already hypovolemic.

21.10. CASUALTY DECONTAMINATION

No decontamination is required following exposure to classic pulmonary agents in gas/vapour form. As a precaution, clothing should be removed from T1 (severe) casualties.

21.11. PULMONARY ADVANCED MEDICAL CARE

1. There is currently no specific antidote for pulmonary agent treatment. The treatment is therefore supportive and may include critical care. Clinical investigations are also available but are of limited value in all but the most severe cases with the exception of blood pressure and oxygen saturation monitoring.

2. The treatment should be focused on:

a. Ensuring a patent and protected airway.

b. Managing any bronchospasm.

c. Improving oxygenation by increasing inspired oxygen levels and CPAP.

d. Where CPAP is ineffective or not tolerated, intubate and ventilate with high PEEP.

e. Where there is ventilatory failure (i.e. type 2 respiratory failure), intubate and ventilate.

f. The pulmonary effects of some other chemical agents (e.g. sulphur mustard) can cause pseudomembrane formation that may require repetitive broncho-alveolar lavage.

21.11.1. CLINICAL INVESTIGATIONS

Sophisticated laboratory studies are of limited value in the immediate care of casualties. The following studies are of some predictive value in determining the severity of exposure and the likely outcome but treatment is supportive and guided by clinical assessment. Clinical investigations including routine biochemistry and blood counts, and:

a. *Chest radiograph*. The presence of hyperinflation suggests toxic injury of the smaller airways that results in air being diffusely trapped in the alveoli. The presence of “batwing” infiltrates suggests pulmonary oedema. Atelectasis is often seen with more central respiratory exposures. As radiological changes may lag behind clinical changes, the chest radiograph may be of limited value, particularly if normal.

b. *Arterial blood gases*. Type 1 respiratory failure often results from exposure to pulmonary agent. Measurement of the partial pressure of oxygen (PaO2) is a sensitive but non-specific finding; both the central and peripheral effects may produce hypoxia. Arterial blood gases showing a low PaO_2 or $PaCO_2$ may be an early, non-specific warning of increased interstitial fluid in the lung and compensatory hyperventilation. After 4-6 hours, normal arterial blood gas (ABG) values make a lethal complication less likely. Carbon dioxide elevation may be seen in individuals with underlying hyper-reactive airways and is due to bronchospasm. This may be triggered by exposure to the chemical and causes *ventilatory compromise* (type 2 respiratory failure) rather than a *failure of oxygenation* (type 1 respiratory failure).

c. *Haematocrit*. An increase in the haematocrit may reflect the haemoconcentration due to fluid loss into the pulmonary parenchyma

d. *Pulmonary function tests*. Peak expiratory flow rate may decrease early after significant exposures. This non-specific test helps to assess the degree of airway damage and the effect of bronchodilator therapy. Decreased lung compliance and carbon dioxide diffusing capacity are particularly sensitive indicators of interstitial fluid volume in the lung. However, these tests have limited clinical and operational application in the early stages of the condition and may be of more benefit for the assessment of chronic disease.

21.11.2. CRITICAL CARE

1. Severe pulmonary agent casualties will require respiratory support and critical care. The ventilation strategy to be used depends on the severity of symptoms, causative agent and type of respiratory failure (pulmonary oedema, ARDS). The clinical status of the patient may evolve and progress.

2. The period of critical care for non-cardiogenic pulmonary oedema may be relatively short despite severe symptoms initially. Care should also be taken to monitor for any iatrogenic causes for deterioration including a reduction of venous return, fluid overload and complications of ventilation.

3. Complication of pulmonary agent use may include failure to wean from the ventilator and secondary infections.

4. *Multi-organ failure*. The indication of systemic illness should be investigated further. Any early signs of sepsis or multi-organ failure should prompt clinicians to consider alternative causes including respiratory infection and inhaled toxins.

21.11.3. POST-EXPOSURE OBSERVATION

1. The period of observation depends on the agent, downwind location of the casualty, the wearing of effective respiratory protection, immediate symptoms, and clinical features. Because of potentially a large number of casualties, an observation area should be considered with appropriate medical personnel and equipment. Casualties should be monitored and encouraged to rest and avoid exertion. For asymptomatic or mild cases, such as eye and upper airway symptoms, chest radiographs and ABGs are not justified. Casualties with lower respiratory symptoms or signs including wheeze requiring treatment, shortness of breath at rest or on exertion, or resolved cyanosis should be monitored for at least 24 hours.

2. *Phosgene*. The following criteria are suggested following exposure to phosgene:

a. If the casualty had eye or upper airway irritation, and took immediate action including respiratory protection AND is asymptomatic with normal physical examination 12 hours later, they may be returned to duty.

b. If the casualty had eye or upper airway irritation (but had no respiratory protection) AND is asymptomatic with normal physical examination 24 hours later, they may be returned to duty. This is due to immediate effects being associated with very high concentrations of phosgene.

c. If the person's original complaint was dyspnoea AND normal physical examination, chest x-ray, arterial blood gases at 24 hours, he may be returned to duty.

d. If the patient presented initially with symptoms AND an abnormal physical examination, chest x-ray, or arterial blood gas, he requires admission close observation but can be returned to light duties at 48 hours if physical examination, chest x-ray and arterial blood gases are all normal at that time.

3. *Chlorine*. The following criteria are suggested following exposure to chlorine:

a. If the casualty had only eye or upper airway irritation and is asymptomatic with normal physical examination 6 hours later, they may be returned to duty.

b. If the person's original complaint included dyspnoea AND physical examination, chest x-ray, arterial blood gases are all normal at 24 hours, he may be returned to duty.

c. If the patient presented initially with symptoms AND an abnormal physical examination, chest x-ray, or arterial blood gas, he requires close supervision but can be returned to light at 24 hours if physical examination, chest x-ray, and arterial blood gases are all normal at that time.

PULMONARY AGENTS

CHLORINE	PHOSGENE
Green gas. Smell of swimming pool.	Classic smell of freshly mown hay / grass.

MECHANISM OF ACTION

The effects depend on the agent and its solubility. Water soluble gases such as chlorine react with moisture in the airways and on the eyes to cause immediate effects including bronchospasm. These immediate effects are only seen at very high concentrations for phosgene. Effects include acute and delayed damage to the lung epithelium either directly or via free radicals or inflammatory mediators. Damage to the alveoli causes decrease lung compliance, pulmonary oedema and type I (oxygenation) respiratory failure. Late features may be due to an ARDS type mechanism resulting in pulmonary hypertension and Type II respiratory (ventilatory) failure or mixed.

QUICK LOOK (CRESS)

Conscious	Respiration	Eyes	Secretions	Skin	Other
Agitated	Increased	Normal or inflamed (chlorine)	Respiratory secretions ± blood	Normal → cyanosed	

SELF / FIRST AID

Remove from scene; avoid exertion
If respiratory distress AND hazard cleared, remove respirator
If liquid hazard or T1, removed clothing
Basic airway management including head tilt and chin lift
If respiratory secretions, allow free drainage in recovery (semi-prone) position
If cyanosed, give oxygen if available

CASUALTY DECONTAMINATION

No formal decontamination is required for gases. However, T1 casualties should have clothing removed as a precaution.

TRIAGE CATEGORIES

T1 (Severe)	T2 (Moderate)	T3 (Mild)
Respiratory distress. Cyanosis.	Non-ambulatory. Persistent cough. Haemoptysis.	Walking.

EMERGENCY MEDICAL TREATMENT

Supportive management:
Airway: Airway manoeuvres, suction
Breathing: Oxygen if SpO_2 < 93%, CPAP if oxygen not correcting cyanosis. Bronchodilators.
Circulation: Ensure hydration, especially if CPAP delivered.
AVOID OXYGEN UNLESS REQUIRED

MedCM / Antidotes:
No specific MedCM.

PHOSGENE casualties should be observed for up to 24 hours due to the risk of latent effects including sudden death.

Clinical Investigations:	Chest radiograph, arterial blood gas, haematocrit.

ADDITIONAL INFORMATION

The mainstay of the management of casualties following pulmonary agent exposure is supportive and primarily respiratory. The mechanism for pulmonary oedema is non-cardiogenic and so initial management with vasodilators and diuretics should be avoided. The radiological evidence of pulmonary oedema may be delayed and clinical interventions should be based on clinical assessment and evidence of hypoxia. In the operational environment, casualties may be dehydrated and should be rehydrated if CPAP is considered due to the effect of raised intrathoracic pressures on venous return. In severe cases, endotracheal intubation and mechanical ventilation with high PEEP or ARDS ventilation strategies may be required.

CHAPTER 22: CYANIDES AND RELATED POISONS

22.1. INTRODUCTION

1. Cyanogen agents produce their effects by interfering with oxygen utilisation by the –CN group at the cellular level. Although sometimes described as 'blood agents', this term is inaccurate and there are other poisons that have a direct toxic effect on haemoglobin component of blood. (e.g., carbon monoxide, nitrites). The cyanides include:

a. *Hydrogen cyanide (AC)*. This is a gaseous toxic industrial chemical used during WW1 and is still significant as an improvised device because of its ready availability. Hydrogen cyanide is released when cyanide compounds (e.g. sodium cyanide, potassium cyanide, acetone cyanohydrin) are spilled in water or subjected to acid.

b. *Cyanogen halides*. Cyanogen chloride (CK) and cyanogen bromide were used during WWI. The toxicity of cyanogen halides is due to the CN-group and the irritant properties to the halogen component.

c. *Cyanide salts*. These salts as mentioned above are precursors to forming hydrogen cyanide. However, they may also be used for poisoning although the total number of casualties is likely to be relatively low and depends on the amount of salt used and the dilution effect.

2. The common chemical hydrogen sulphide (H_2S) has toxicity comparable to hydrogen cyanide acting by a similar mechanism. H_2S is a significant operational poison due to its association with confined space entry such as into sewage pipes or tanks. It has also been used increasingly as a method of chemical suicide. However, its effectiveness is limited to very confined spaces such as a car or bathroom and is of limited use for a large chemical attack.

22.2. HYDROGEN CYANIDE

1. *Physical Properties.* Under most field conditions, hydrogen cyanide is a colourless gas and represents a non-persistent hazard. It has a boiling point of 26°C and so may be found as a liquid in cold operational environments. The vapour is lighter than air and has a faint odour, similar to bitter almonds, although about up to 40% of people are unable to smell this. The vapour is also flammable with a risk of explosion. It is highly soluble and stable in water.

2. *Chemical Properties*. The CN compounds hydrolyse slowly in water with subsequent loss of toxicity. They are readily oxidised by strong oxidants e.g. potassium permanganate. Hydrogen cyanide has an affinity for oxygen and is flammable and therefore less efficient when dispersed by artillery shells. Compounds which contain labile sulphur atoms such as sodium thiosulphate (a cyanide antidote), react with HCN even in vivo. Cyanide also readily binds to metal ions, being the basis for both its toxicity (ferric iron) and exploited by the cobalt antidotes.

3. *Detection*. Automatic detectors are available which detect attack concentrations of cyanide vapour. Colorimetric tubes are also available, as are water testing kits.

4. *Protection*. Hydrogen cyanide, because of its volatility and low molecular weight, is poorly adsorbed by the activated charcoal in the canister of the respirator. This charcoal is made more reactive by impregnating it with metal salts. Modern CBRN filters remain effective against attack with hydrogen cyanide, but should be changed immediately afterwards. At

higher concentrations and cold climates, skin protection should also be consider if there is a contact or liquid / vapour hazard.

22.3. MECHANISM OF ACTION

1. The cyanide ion forms a reversible complex with the ferric iron (Fe^{3+}) of mitochondrial cytochrome c oxidase. This is part of the enzyme system that is essential for oxidative processes within cells. Cyanide results in cellular anoxia by impairment of cellular oxygen respiration. The cells switch to anaerobic respiration and release high levels of lactic acid. The central nervous system and in particular the respiratory centre is especially susceptible to this effect causing convulsions, respiratory failure and death.

2. The human metabolism has an enzymatic method for the detoxification of cyanide using the sulfurtransferases including rhodanese. This converts cyanide (-CN) into the less toxic thiocyanate (SCN-). The presence of this metabolic pathway means that humans have a threshold and can tolerate low levels of cyanide before showing toxic effects. This mechanism of detoxification is the basis for the use of sodium thiosulphate as an antidote.

22.4. CLINICAL EFFECTS

1. *High concentrations*. At high concentrations, the following effects are seen:

   a. *Pink skin*. Inhibition of oxygen extraction by the tissue, leading to arterialisation of venous blood and initial pink skin colouration.

   b. *Increased respiratory drive and rate*. An increase in the depth of respiration within a few seconds. This stimulation may be so powerful that a casualty cannot voluntarily hold his or her breath and is due to a metabolic lactic acidosis.

   c. *Loss of consciousness* with possible *convulsions* occurs after 20 to 30 seconds.

   d. *Cyanosis*. Despite oxygenated blood, a lactic acidosis causes a shift in the oxygen dissociation curve to the right resulting in the casualty looking cyanosed.

   e. *Respiratory arrest* within 1 minute.

   f. *Cardiac dysrhythmia* and *cardiac arrest* follows within a few minutes.

Note. Sudden loss of consciousness in the absence of other characteristic features of nerve agent intoxication is a key indicator of potential lethal cyanide poisoning, if supported by the threat, circumstances and other supporting evidence. Sudden loss of consciousness may also be due to entry into an anoxic environment, carbon monoxide or hydrogen sulphide as well as non-toxic causes if single casualties.

2. *Low Concentrations.* At low concentrations, the early symptoms are weakness of the legs, vertigo, nausea and headache.

3. *Neurotoxicity*. Convulsions may follow with coma and may last for hours or days depending on the duration of exposure to the agent. Recovery from prolonged coma may disclose residual damage to the central nervous system manifested by irrationality, altered reflexes and unsteady gait which may last for several weeks or longer. Temporary or permanent nerve deafness has also been described. In mild cases there may be headache, vertigo and nausea for several hours before complete recovery.

Note. These symptoms are also associated with carbon monoxide poisoning and may be due to a similar CNS mechanism in addition to the effects on haemoglobin. The effects may also be additive. Treatment for both is high flow oxygen and consideration of hyperbaric therapy although this remains controversial in some countries.

22.5. CYANIDE TREATMENT

1. The key to treatment of cyanide casualties is speed as first aid and EMT. Although there is disagreement regarding the ideal antidote to use, there is consensus for the need for urgent action. High concentration oxygen and ventilation is vital in the early management as it is an antidote as a direct competitor with cyanide for cytochrome c oxidase as well as correcting tissue hypoxia.

2. Death may occur within minutes without treatment as a respiratory arrest followed by cardiac arrest.

3. *Clinical investigations*. Near patient and/or laboratory testing for cyanide levels is not widely available. The diagnosis of cyanide poisoning should be considered with an arterial and venous blood gas sample with high PaO_2 AND high lactate levels (>10 mmol/L). Oxygen saturation (high or low) may be misleading especially with a high PaO_2, if there is a low blood pH (<7.0). This is due to the metabolic acidosis and the shift in the oxygen dissociation curve causing haemoglobin to deoxygenate.

Note: Falsely high lactate levels may also be seen in cases of ethylene glycol poisoning due to a measurement error with some biochemical analysers.

22.5.1. FIRST AID

1. The casualty should be removed from the source of hydrogen cyanide as soon as possible. Rescue workers should wear adequate PPE although hydrogen cyanide is a gas and is non-persistent.

2. Any patient that is symptomatic should be given high flow oxygen, where available.

3. Patients fitting, in respiratory or cardiac arrest should be considered for immediate antidote therapy. If exposure or arrest was less than 10 minutes AND resuscitation is possible, resuscitation with oxygen should be started or continued. This may be stopped if overwhelmed by casualty numbers or under medical advice. Mouth-to-mouth ventilation should <u>not</u> be performed.

Note: Some ventilation equipment has the option to use filtered air supplemented with oxygen As hydrogen cyanide is non-persistent and oxygen is an antidote as high a concentration as possible should be given.

22.5.2. EMERGENCY MEDICAL TREATMENT

1. The priorities for the EMT of cyanide casualties is:

    a. Removal from the hazard.

    b. Administration of high flow oxygen, as soon as possible.

c. Early administration of antidotes to severe (T1) cases (see below). Where possible, the diagnosis should be confirmed or based on good evidence of a cyanide threat or exposure.

2. For patients that are symptomatic but conscious, high concentration oxygen should be given unless there is demand by other casualties with greater severity.

3. For patients fitting but cyanide has not been confirmed, standard anticonvulsant therapy with benzodiazepines may be given. Antidote treatment can be started once cyanide exposure has been confirmed.

4. Early venous and arterial blood gas analysis may support the diagnosis of cyanide poisoning (see below). The report of the smell of bitter almonds may also support the diagnosis.

22.6. CASUALTY DECONTAMINATION

Because of its physical properties (boiling point 26°C), hydrogen cyanide will not remain for long if in a liquid state. Decontamination should not be necessary. Cyanide salts are crystalloid and soluble, decontamination for dry material in an open environment should be used as well as a management of a persistent liquid hazard as run-off. Contact with acid should be avoided.

22.7. CYANIDE ANTIDOTE THERAPY

1. Cyanide is detoxified in the body by the sulfurtranferases such as rhodanese. This process may be enhanced by one of the antidotes to be discussed below (sodium thiosulphate). This antidote may have a role for treated casualties with residual symptoms or metabolic disorder, such as a lactic acidosis.

Note: Any casualty who is fully conscious and breathing normally more than 5 minutes after presumed exposure to cyanide agents has ceased will recover spontaneously with oxygen and does not require antidote treatment.

2. There are three main approaches to the antidote treatment of cyanide poisoning and two (a. and b.) are often used in combination. The route of antidote administration should also be considered with the intraosseous route becoming more widely used in environment and on casualties where intravenous access is a challenge. The approaches used vary between nations but all nations should be aware of the antidotes used by partner nations contributing to the continuum of casualty care:

a. *Methaemaglobin-formation*. This method provides alternative ferric iron (Fe^{3+}) tissue (intrinsic) binding sites for the cyanide ions away from cytochrome c oxidase and thereby restoring the enzyme's activity. Cyanide binds to form the non-toxic cyanomethaemoglobin although this is reversible. Methaemoglobin formers include *amyl nitrite, sodium nitrite* and *4-dimethylaminophenol* (4-DMAP). Methaemoglobin forming compounds are contraindicated in casualties with concurrent carbon monoxide poisoning (smoke inhalation), hypoxia or trauma due to its impact on oxygen delivery. Although this method is rapid, it does not remove cyanide from the body, it is often followed by enhanced detoxification.

b. *Enhanced detoxification*. Endogenous detoxification by sulfurtransferases is at a rate that is of little practical importance. However, the provision of additional sulphur groups, using sodium thiosulphate, enhances the detoxification of cyanide to thiocyanate.

c. *Cobalt chelation*. This method uses extrinsic binding sites provided by drugs such as dicobalt edetate and hydroxocobalamin (vitamin B12a) and is independent of detoxification. Cyanide has a greater affinity for cobalt ions than ferric or ferrous ions. It binds to form a non-toxic complex that is eliminated by the kidneys.

22.7.1. METHAEMOGLOBIN FORMERS

1. *Amyl Nitrite*. Amyl nitrite may be of benefit when used as a first aid measure following immediate exposure and moderate symptoms. This is prior to the use of intravenous antidotes and where oxygen is not available. The antidote may be given either by crushing the ampoule and holding near the casualties face or placing it inside the face piece of a respirator. However, these methods rely on the casualty breathing. It should not be used with concurrent oxygen administration due to the risk of explosion. Treatment with amyl nitrite should be followed by oxygen and sodium thiosulphate, once available.

2. *Sodium Nitrite*. Sodium nitrite (300mg) must be administered intravenously over a period of 3 minutes. The sodium nitrite is given to produce methaemoglobin, thus sequestering the cyanide on the methaemoglobin. Subsequent intravenous administration of sodium thiosulphate is required to promote the conversion of cyanide to thiocyanate ion which is then excreted from the body. Hypotension is a side effect although the decrease in blood pressure following sodium nitrite injection is negligible unless the patient is allowed to get into an upright position. However, this may be more severe in the presence of trauma or hypovolemia.

3. *4-Dimethylaminophenol-hydrochloride (4-DMAP)*. 4-DMAP induces a methaemoglobinaemia more rapidly than nitrites (within minutes). A single 250 mg (or 3.3mg/kg) of 4-DMAP should be given intravenously over 3 - 5 minutes. The intramuscular route is also effective but may cause necrosis at the injection site. Used alone (or with oxygen), this antidote is considered a first aid measure until further treatment is given such as sodium thiosulphate.

Note. 4-DMAP may cause a life-threatening haemolysis. A repeat dose of 4-DMAP must not be given and haemoglobin and methaemaglobin monitoring is advised.

4. *Reversal of methaemoglobinaemia*. The development of a mild or transient cyanosis may be associated with the production of methaemoglobinaemia. This does not require respiratory support and oxygen should already be given, although it may not have an immediate effect on measured saturation (SpO_2). Ferric iron is continually being converted back to haemoglobin by methaemoglobin reductase. If methaemoglobinaemia is severely symptomatic, methylene blue may be given to convert methaemoglobin to haemoglobin. *Methylene blue* is given–as 1 - 2 mg/kg by slow IV injection over 5 - 10 minutes; repeated once after 1 hour if no response. Reversal should be considered, if the cyanide-induced lactic acidosis has been reversed and co-oximetry shows excessive methaemoglobin levels (>50%). The treatment of iatrogenic methaemoglobin can also be applied to accidental or deliberately-induced methaemoglobinaemia due to medication, recreational drug use and some TICs.

22.7.2. ENHANCED DETOXIFICATION

Sodium thiosulphate. Sodium thiosulphate enhances cyanide detoxification synergistically with methaemoglobin-formers and cobalt chelators. The antidote provides additional thiosulphate ions and these combine with cyanide ions in a reaction catalysed by sulfurtranferases to produce thiocyanate. The dose is 12.5g (50ml of 25%) given intravenously over a 10 minute

period. As well as part of immediate therapy, other indications include a persistent lactic acidosis after cobalt chelation therapy.

Note. Sodium thiosulphate and other antidotes should not be mixed together because of potential interactions.

22.7.3. COBALT CHELATION THERAPY

1. *Dicobalt edetate.* This antidote is only used for severe cyanide poisonings including peri-arrest, and respiratory and/or cardiac arrest. Dicobalt edetate once injected rapidly releases cobalt ions that react directly with free cyanide ions independent of any rhodanese activity. Stable and non-toxic cyanide-cobalt complexes are formed and excreted by the kidneys. The antidote must be given intravenously initially as 300mg, immediately followed by a glucose infusion (250ml of 10% glucose). This may be repeated with a further 300mg followed by an additional glucose infusion. This antidote and its relatively low volume are useful for the treatment *of the most severe cases of confirmed cyanide poisoning* by the intraosseous or intravenous route. Dicobalt edetate may be followed by an intravenous injection of sodium thiosulphate especialy if there is a persistent lactic acidosis.

Note: Severe side effects have been described especially *in the absence of free cyanide ions*, and include hypertension, facial and laryngeal oedema, and hypotension.

2. *Hydroxocobalamin.* Hydroxocobalamin (vitamin B12a) is another cobalt containing antidote that binds cyanide to form non-toxic cyanocobalamin. Both are excreted by the kidneys. It must be given intravenously and the initial adult dose is 5g (70 mg/kg). Due to formulation this is a significant volume to give and has to be infused. Repeated doses may be given depending on the severity of poisoning. The evidence for its use in cyanide toxicity is mainly in the context of smoke inhalation. Discolouration of the urine is expected and there have been suggestions of urticarial rashes and anaphylactoid reactions even in low dose therapy for blood disorders.

22.8. CYANOGEN HALIDES

1. Cyanogen chloride and cyanogen bromide, usually in mixtures with other warfare agents (hydrogen cyanide, bromoacetone), were used during WW1. Their effects on the body are similar to those of hydrogen cyanide but they also have irritant effects on the eyes and upper respiratory passages.

2. *Physical properties*. Under most field conditions, cyanogen chloride is a colourless, strongly irritant gas. Although only slightly soluble in water, it dissolves readily in organic solvents. Its vapour, heavier than air, is very irritating to the eyes and mucus membranes. Cyanogen chloride has pungent, biting odour. Normally cyanogen chloride is non-persistent.

3. *Detection*. Detection is available including water testing kits.

4. *Decontamination*. As with hydrogen cyanide, because of its physical properties the agent will not remain for long in its liquid state. Decontamination should not, therefore, be necessary.

5. *Mechanism of Action*. Cyanogen chloride acts in two ways:

   a. *Systemic cyanide effect*. Its systemic effects are similar to those of hydrogen cyanide.

b. *Local effects*. It also has local irritant effects on the eyes, upper respiratory tract and lungs as a result of the formation of hydrogen chloride. Cyanogen chloride injures the respiratory tract, resulting in severe inflammatory changes in the bronchioles and congestion and oedema in the lungs. Very low concentrations produce eye irritation and lachrymation.

6. *Medical effects*. The signs and symptoms caused by cyanogen chloride are a combination of those produced by hydrogen cyanide and a lung irritant such as phosgene. Initially, cyanogen chloride stimulates the respiratory centre and then rapidly paralyses it. In high concentrations, however, its local irritant action may be so great that dyspnoea is produced. Exposure is followed by an immediate intense irritation of the nose, throat and eyes, with coughing, tightness in the chest and lachrymation. Afterwards the exposed person may become dizzy and increasingly dyspnoeic. Unconsciousness is followed by failing respiration and death within a few minutes. Convulsions, retching and involuntary defecation may occur. If these effects are not fatal, the signs and symptoms of pulmonary oedema may develop. There may be persistent cough with much frothy sputum, rales in the chest, severe dyspnoea and marked cyanosis.

7. *Treatment*. Cyanogen halide poisoning should be treated in the same way as hydrogen cyanide poisoning as regards its cyanide-like effects. Pulmonary irritation should be treated in the same way as phosgene poisoning.

22.9. HYDROGEN SULPHIDE

1. Hydrogen sulphide is a toxic gas often associated with decomposing tissue and sewage. It has a characteristic bad egg smell. It has increasingly been used as a form of chemical suicide using binary commercial products. This however would have limited mass effect as a terrorist weapon unless in an enclosed environment.

2. The mechanism of action is similar to cyanide with CNS effects and inhibition of intracellular respiration. The cellular effect causes similar rapid onset effects as cyanide with loss of consciousness and sudden death.

3. Treatment includes oxygen and the potential use of methaemoglobin-formers to redistribute the poison away temporarily from the mitochondrial respiratory enzymes. Sodium thiosulphate is not indicated as hydrogen sulphide itself is metabolised to thiosulphate by mitochondrial enzymes.

CYANIDES / HYDROGEN SULPHIDE (H_2S)

HYDROGEN CYANIDE	CYANOGEN CHLORIDE
Colourless gas. Smell of almonds. LCt_{50} 2500mg.min/m^3 LD_{50} 100mg/kg (skin)	Colourless liquid and gas. Odourless. LCt_{50} 11,000mg.min/m^3

MECHANISM OF ACTION

Inhibition of intracellular aerobic respiration by binding to the mitochondrial ferric cytochrome a_3. There is an arterialisation of venous blood due to a failure of oxygen extraction and a transient pink colour to the skin. Respiration is switched to anaerobic causing a severe lactic acidosis, respiratory distress and the rapid failure of aerobic dependant organs such as the brain and heart. Antidote treatment is either by displacing and binding cyanide to form non-toxic complexes or to enhance liver detoxification of cyanide by the enzyme rhodanese.

QUICK LOOK (CRESS)

Conscious	Respiration	Eyes	Secretions	Skin	Other
Unconscious / convulsions	Increased → apnoea	Normal → dilated pupils	Normal	Pink → cyanosis	Sudden / rapid onset

SELF / FIRST AID	CASUALTY DECONTAMINATION
Remove from scene immediately If breathing and symptomatic, administer oxygen Administer cyanide MedCM immediate therapy (amyl nitrite inhaled or 4-DMAP i.m. (if available)) Start resuscitation, if arrested and witnessed or within 10 minutes	Removal of clothing if symptomatic

TRIAGE CATEGORIES

T1 (Severe)	T2 (Moderate)	T3 (Mild)
Unconscious, convulsions. Hyperventiolation (cyanosed), respiratory distress or arrest.	Not walking. Confusion, history of loss of consciousness, hyperventilation (not cyanosed).	Walking. Nausea, vertigo, mild agitation.

EMERGENCY MEDICAL TREATMENT

Supportive management:

Airway: Airway manoeuvres, suction
Breathing: Oxygen.
Disability: Treat convulsions.

MedCM / Antidotes:

Oxygen.
Met-Hb formation (nitrite or 4-DMAP) and enhanced detoxification
Sodium nitrite 300mg IV over 5-10 minutes, followed by sodium thiosulphate (12.5g) IV over 5 minutes.
Cobalt chelation:
Dicobalt edetate (if HCN confirmed) 300mg IV followed by 500ml of 10% glucose, repeat 300mg if required; or
Hydroxocobalamin 5-10g IV.

Clinical Investigations:	Venous gas (↑ pO2 and lactate > 10mmol/L, suggestive of HCN poisoning). Met-Hb to monitor any antidote efficacy.

ADDITIONAL INFORMATION

The choice of cyanide antidotes varies between nations. All nations should be aware of partner nations' treatment protocols to ensure a continuum of care including compatible antidote protocols.
Nations should also be able to monitor any side effects of antidote treatment including met-Hb. This can be treated is symptomatic or levels >50% with methylene blue (1-2mg/kg).
Cobalt chelation therapy may be followed by sodium thiosulphate, especially if there is a persistent lactic acidosis.

CHAPTER 23: INCAPACITATING AGENTS

23.1. INTRODUCTION

An incapacitant is a chemical agent which usually produces a temporary disabling condition that persists for hours to days after exposure to the agent has occurred (unlike that produced by riot control agents). Medical treatment while not essential may in some cases facilitate a more rapid recovery. Incapacitants are unlikely to produce death or permanent injury in concentrations which are militarily effective. There is significant overlap in classification between incapacitants, RCA and pharmaceutical-based agents both for legitimate use, and illicit or military warfare use.

23.2. TYPES OF INCAPACITATING AGENTS

Many compounds have been considered as incapacitants and medical staffs must be on the alert to detect and report any unusual clinical appearances. Incapacitating agents can cause physical and mental (CNS) incapacitation. With regards to casualty care, the main groups are:

a. Mental incapacitants.

(1) Psychoactive (psychotomimetic) agents.

(2) Sedating agents.

b. Physical incapacitants.

(1) Vomiting agents / sternutators (sneezing agents).

(2) Lachrymators (tearing agents).

2. This chapter will discuss the psychotomimetic agents in detail and some of the military physical incapacitating agents, while Chapter 24 will discuss the physical incapacitants used commonly as RCA and pharmaceutical based agents (PBA).

23.3. PSYCHOACTIVE AGENTS

Psychoactive agents, also referred to as psychotomimetic and psychotropic, produce a psychotic-like state by interfering with neurotransmission. Examples may include:

a. 3-quinuclidinyl benzilate (BZ);

b. D-lysergic acid diethylamide (LSD);

c. Phencyclidine; and potentially

d. Cannabinols.

23.3.1 BZ (3-QUINUCLIDINYL BENZILATE)

1. BZ and its analogues are glycolic acid esters. Some members of the group are liquid at ambient temperatures but BZ is a stable white crystalline powder that may be aerosolised. BZ is only slightly soluble in water and these agents are metabolised primarily in the liver and

excreted by the kidneys. It is specifically listed in the CWC as an incapacitant and as such is prohibited.

2. *Mechanism of action*. BZ is an anticholinergic (antimuscarinic) agent that at single doses of less than 1 mg produces delirium lasting several days. It resembles the well-known belladonna alkaloids, atropine and scopolamine, except that it is more potent and its effects last longer. It is 25 times more potent than atropine.

3. BZ is effective by all routes of administration, but its effectiveness percutaneously (when mixed with a suitable solvent) is limited, so that route is not likely to be used.

4. After exposure to an effective dose, mild peripheral effects of BZ occur within 1 hour and central effects occur after about 4 hours, with a peak–at 8 – 10 hours and lasting 24 – 48 hours. Some other compounds in this group may take longer for their effects to develop and to disappear. Doubling the dose prolongs the duration of severe central effects by about 40 hours and shortens the onset time of severe effects to about 1 hour.

5. *Signs and symptoms*. Small doses of BZ cause sleepiness and diminished alertness. Diagnosis can be made by noting increased heart rate, dry skin and lips, drowsiness and progressive signs of intoxication in the untreated individual as follow–:

a. 1 – 4 hours*:* Tachycardia, dizziness, ataxia, vomiting, dry mouth, blurred vision, confusion, sedation progressing to stupor.

b. 4 – 12 hours: Inability to respond to the environment effectively or to move about–

c. 12 – 96 hours: Increasing activity, random unpredictable behaviour with delusions and hallucination; gradual return to normal 48 to 96 hours after exposure.

6. *Treatment*. For most casualties, symptomatic treatment is all that will be necessary. Restraint should be avoided and only when necessary. All dangerous objects must be removed and anything likely to be swallowed should be kept away from the subject as bizarre delusions may occur.

7. *Heat illness*. The most important single medical consideration is the possibility of heat stroke. Clothing should be removed if the temperature is greater than 25°C. If the body temperature is greater than 39°C vigorous cooling is indicated. The casualty should be placed in the shade and air allowed to circulate. Water may be sprayed on the casualty to aid cooling, ice should not be applied to the skin.

8. *Physostigmine*. Physostigmine, where available, is used as an antidote for anticholinergic toxicity including atropine and BZ. Treatment should only start once symptomatic as physostigmine also has toxic effect offset by the effects of an anticholinergic. The importance of supportive care is as important as antidote treatment. Where this treatment is indicated, an initial dose of 1-2 mg and repeated injections at intervals of approximately 15-60 minutes to build up a sufficient level of effect. Doses of 2-4 mg every 1 or 2 hours may be required and sustained over a 24 hour period due to the prolonged effect of BZ. Side effects include a nerve agent-like bradycardia, excessive secretions and convulsions. An alternative to physostigmine is 7-methoxytacrine (7-MTHA).

23.3.2. D-LYSERGIC ACID DIETHYLAMIDE (LSD)

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

2. *Mechanism of action*. Very small doses (e.g. 50 µg per person) are capable of inducing a psychotic state, but the precise mechanism of action is not yet known. LSD has been shown to facilitate neural activity in the reticular activating system of the brain stem. It appears to interfere with the normal filtering action of this system, permitting sensory input to reach higher integrative centres without regard to its importance or relevance. The result is a decrease in the ability of the brain to process information selectively and in logical sequence.

3. *Medical effects*. LSD is the most potent of the biologically active indole compounds, as little as 50 µg being required to produce dramatic psychological changes. Doses of 2 to 5 mg have been taken without permanent sequelae, and animal studies suggest that much higher doses may be tolerated, however, convulsions may occur at doses above 2 mg.

4. LSD may be inhaled or ingested. Initial effects appear within a few minutes of inhalation or within 30 to 60 minutes of ingestion. Maximum effects are reached within 2 to 3 hours and gradually subside over the next 4 to 8 hours. The half-life in human plasma is about 3 hours. Tolerance is acquired rapidly on repeated exposures at daily intervals, but is short-lived.

5. LSD appears to interact with endogenous neurotransmitters such as serotonin with which it shares the common feature of an indole nucleus. It is metabolised by the liver and excreted through the kidneys.

6. *Signs and symptoms*. The clinical manifestations of LSD intoxication often include an early stage of nausea followed 45-60 minutes after dosage by a confused state in which delusions and hallucinations are common but not always experienced. There is some evidence that the effects may be held off, at least for a time, by determination to continue duty and that the presence of non-intoxicated personnel enables affected subjects to maintain contact with reality.

7. Subjects intoxicated with LSD show evidence of sympathetic stimulation (rapid heart rate, sweating palms, pupillary enlargement, cold extremities) and mental excitation (nervousness, trembling or spasms, anxiety, euphoria and inability to relax or sleep). Hyperthermia has been reported. Subjectively, feelings of tension, heightened awareness, exhilaration, kaleidoscopic imagery, emotions of every type, hilarity and exultation are characteristic. Paranoid ideas and more profound states of terror and ecstasy may also occur, especially in highly suggestible individuals. True hallucinations are rare, as is homicidal or suicidal behaviour.

8. *Treatment*. There is no known true antagonist to the indoles. The best treatment at present for LSD intoxication is the administration of a benzodiazepine and placing in a reduced stimulating environment.

9. *Clinical course and prognosis*. The question of long term effects is still unresolved, but single exposures to doses in the clinical range (0.1 to 1.0 mg total dose) appear unlikely to cause any permanent biological damage. There has been concern of recreational LSD and the phenomenon of flash back with a reoccurrence of LSD effects including hallucinations.

23.3.3 PHENCYCLIDINE (PCP)

1. Phencyclidine (1-(1-phenylcyclohexyl)piperidine) is a dissociative and sympathomimetic drug which can act as a stimulant or depressant depending on dose. As a recreational drug it is known as Angel Dust. It has a rapid onset within minutes if inhaled.

2. *Clinical effects*. Effects include euphoria, agitation, combativeness and disorientation. Some of the manifestations are similar to the effects of ketamine which is also dose-dependent. Severe effects include ataxia, muscle rigidity and myoclonus, hyperpyrexia, convulsions and coma. Death can occur due to hypertensive crisis, rhabdomyolysis and resulting renal failure as well as death due to loss of inhibition and self-destruction. Effects may be prolonged over several days.

3. *Treatment*. Treatment is generally supportive with standard management of any agitation or convulsions with close monitoring of renal function and biomarkers for muscle breakdown such as creatinine kinase and myoglobinuria.

   a. *Hyperpyrexia*. Dantrolene may be considered for resistant hyperpyrexia.

   b. *Rhabdomyolysis.* Consider urine alkalinisation.

   c. *Acute dystonic reactions*. Consider procyclidine (5-10mg) intravenous.

4. *Detection*. Urinalysis, at least up to 8 days, can be used for confirmation of exposure.

23.3.4 CANNABINOLS

The cannabinols are a group of potential mental incapacitants based on the derivative of the *cannabis sp*. of plants. Effects include euphoria, stimulation and sedative effects. Treatment is supportive.

23.3.5 DETECTION AND DIAGNOSIS

In general, no automated stand off point detector systems exist for agents in this category and only limited field laboratory methods exist for the identification of such agents in environmental samples. Therefore, initial diagnosis may rest on clinical suspicion supported by intelligence. Toxicological investigation is possible with clinical sampling including urinalysis.

Note: As well as psychotropic agents, heat illness and especially heat stroke should be considered especially in warm environments or following physical exertion.

23.3.6 DECONTAMINATION

Removal of contaminated clothing and rinsing of the skin with soap and water should be done at the earliest opportunity. Symptoms may appear as late as 36 hours after percutaneous exposure, even if the skin is washed within an hour. A delay in onset of several hours is typical and this time, if the release was detected, should be used to prepare for the outcome 6 to 24 hours after the attack.

23.3.7 MENTAL INCAPACITANT CASUALTY MANAGEMENT

1. Following a suspected chemical attack with incapacitating agents, the medical and additional personnel should be prepared to take the following steps:

a. Resistant or disoriented casualties will need to be disarmed.

b. Avoid physical restrain due to the risk of heat illness. They may have to be restrained chemically (sedated) or physically during evacuation.

c. Once the diagnosis of a nerve agent or other lethal substance has been ruled out, the principal signs and symptoms to consider are those shown below. Significant non-chemical causes include:

(1) Heat illness (individual and multiple cases).

(2) Acute stress reaction (individual and multiple cases).

(3) Acute psychosis (individual cases only).

(4) Mass psychogenic (sociogenic) illness event.

2. Severe life-threatening effects include:

a. Heat stroke (exertional / combative) – aggressive cooling techniques.

b. Drug-induced hyperpyrexia – consider dantrolene.

c. Rhabdomyolysis – consider urine alkalinisation and, if available, renal support.

d. Convulsions – early use of benzodiazepines with critical care support.

Table 23-1: Causes of Transient Mental Incapacitation.

SYMPTOMS AND SIGNS	POSSIBLE CAUSES
Restlessness, dizziness or giddiness; failure to obey orders, confusion, erratic behaviour; stumbling or staggering; vomiting.	Anticholinergics (e.g. BZ), indoles (e.g. LSD), cannabinols (e.g. Marihuana), anxiety reaction, other intoxications (e.g. Alcohol, bromides, barbiturates, lead).
Dryness of mouth, tachycardia at rest, elevated temperature, flushing of face; blurred vision, pupillary dilation; slurred or nonsensical speech, hallucinatory behaviour, disrobing, mumbling and picking behaviour, stupor and coma.	Anticholinergics. Heat illness. Infection.
Inappropriate smiling or laughter, irrational fear, distractibility, difficulty expressing self, perceptual distortions; labile increase in pupil size, heart rate, blood pressure. Stomach cramps and vomiting may occur.	Indoles. (Schizophrenic psychosis may mimic in some respects although for individual cases only).
Euphoric relaxed, unconcerned daydreaming attitude, easy laughter; hypotension and dizziness on sudden standing.	Cannabinols.

Tremor, clinging or pleading, crying; clear answers, decrease in disturbance with reassurance; history of nervousness or immaturity, phobias.	Anxiety reaction.

23.3.8 DIAGNOSIS DURING A MASS EFFECT EVENT

In a large-scale attack, the diagnosis will be simplified by the epidemiological distribution of the casualties. It is better to look for characteristics common to all or most casualties, than to be focused on atypical features. For example, some anticholinergics are capable of causing marked disorientation, incoherence, hallucinations and confusion (the pathognomonic features of delirium) with very little, if any, evidence of peripheral autonomic effect (such as tachycardia and dilated pupils). This should not dissuade the medical officer from considering the likelihood of a centrally predominant anticholinergic being the causative agent, since very few other pharmaceutical classes can produce delirium in militarily effective doses. The disturbance produced in indoles (such as LSD) or the cannabinols (such as marihuana extracts) is not really delirium, because the casualties remain receptive to their environment and can comprehend quite well, even though they may have great difficulty reacting appropriately (see Table 23-1).

23.4. VOMITING AGENTS / STERNUTATORS

1. Vomiting agents produce strong pepper-like irritation in the upper respiratory tract with irritation of the eyes and lachrymation. Some of these agents are also referred to as sternutators and cause violent uncontrollable sneezing, cough, nausea, vomiting and a general feeling of bodily discomfort. They are dispersed as aerosols and produce their effects by inhalation or by direct action on the eyes. The principal agents in this group are:

   a. Diphenylchlorarsine (DA).

   b. Diphenylaminochlorarsine (Adamsite (DM)).

   c. Diphenylcyanarsine (DC).

2. Arsenical smokes have in the past been used on the battlefield. As well as lachrymatory action they also provoke other effects, e.g., bronchoconstriction and emesis (hence the term vomiting agents). For historical reasons some older, more toxic compounds are mentioned and cannot be used as RCA.

3. *Properties*. They are non-persistent agents. The particles fall to the ground after dispersion and are virtually ineffective unless re-suspended. DC is the most irritating of the group. It should be remembered that the colour of the solid agent depends on the degree of purity (technically raw products are often coloured) but the colour and odour of the smoke after dispersion may no longer be noticeable in concentrations which are nevertheless still highly irritant, so that odour and colour cannot be relied upon for detection. The following data are applicable to DM. The LCt50 estimated for people is 13000 to 44000 $mg.min.m^{-3}$ depending on the means of dissemination of the agent. The Ict50 for man ranges from 22 to 150 $mg.min.m^{-3}$. The maximum concentration which is stated to cause no permanent damage after inhalation for 5 minutes is 100 $mg.m^{-3.}$

4. *Detection.* No field detectors exist. The use of these agents may be suspected by the clinical symptoms and signs.

5. *Mechanism of Action.* Irritation of exposed mucosa and respiratory tract.

6. *Protection*. Respiratory protection is only required, although individual protective equipment will provide complete protection.

7. *Signs and symptoms*. The onset of symptoms may be delayed for several minutes after initial exposure (especially with DM); effective exposure may, therefore, occur before the presence of the smoke is suspected. If the mask is put on then, symptoms will increase for several minutes despite adequate protection. As a consequence, the casualties may believe their mask is ineffective and by removing it expose themselves further.

8. Inhalation is followed by a burning sensation in the nose and throat, hypersalivation, rhinorrhoea, coughing, sneezing, nausea and vomiting. Mental depression may occur during the progression of symptoms. The paranasal sinuses are irritated and fill with secretions and severe frontal headache results. Prolonged exposure may cause retrosternal pain, dyspnoea and asthma-like symptoms. Symptoms reach their climax after 5 to 10 minutes and disappear 1 to 2 hours after cessation of exposure. Effects on the eyes are slight and are restricted to a burning sensation and lachrymation. Exposure of the skin to high concentrations will cause erythema and itching, proceeding to a burning sensation and vesicle formation. On the battlefield, high concentrations are not likely to occur so that affection of the eyes and skin is unlikely. Ingestion of food and water contaminated by sternutators may cause nausea, vomiting, diarrhoea (sometimes bloodstained) and weakness and dizziness have been reported.

9. High concentrations are not expected in the open owing to movement of air, but may be met within enclosed spaces (shelters, tents, etc.), and under these circumstances the skin may show vesicle formation, capillary damage and localised swelling, while corneal necrosis and pulmonary oedema are possible results. Unsteady gait and a positive Romberg sign have been reported. Other neurological results of severe exposure include hyperaesthesia, anaesthesia and paraesthesia, especially in the legs. Loss of consciousness has been reported.

10. *Treatment*. The respirator should be worn in spite of coughing, sneezing, salivation and nausea. Partial removal of the respirator from the face briefly may be required to permit vomiting or to drain saliva. Combat duties usually can be performed despite the effects of vomiting agents. In spite of the dramatic appearance of the syndrome, usually the only treatment necessary is first aid. The patient should not smoke for some hours. If necessary the mouth may be rinsed with water, but the water should not be swallowed. The eyes and skin may be washed with water. Clothing should be well brushed. In cases of severe exposure treatment as for lung damaging agent poisoning may be required. A mild analgesic may be given to relieve headache and general discomfort.

11. *Prognosis*. Symptoms of exposure to field concentration of vomiting agents usually disappear in 20 minutes to 2 hours, leaving no residual injury. However, a few instances of severe pulmonary injury and death have occurred due to accidental exposure to high concentrations in confined spaces.

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CHAPTER 24: RIOT CONTROL AGENTS AND PHARMACEUTICAL BASED AGENTS

24.1. INTRODUCTION

The CWC prohibits certain named chemical agents as well as imposing limitations such as quantities, stockpiling and/or method of use for other agents including some toxic industrial chemicals (e.g. phosgene) and RCA. Another group of emerging chemicals from the pharmaceutical industry have the potential for legitimate use as well as misuse. The use of fentanyl-derivatives during the Moscow Theatre Siege in 2002 was confirmed as being legitimate and within a legal framework as tested by the European Court of Human Rights. This chapter gives an overview of both RCA and PBA as agents of *low-lethality*.

24.2. RIOT CONTROL AGENTS (LACHRYMATORS)

1. RCA are irritants characterised by a very low toxicity, rapid onset and a short duration of action. In general, these agents have a very wide margin of safety. The CWC defines RCA as "*Any chemical not listed in a Schedule, which can produce rapidly in humans sensory irritation or disabling physical effects which disappear within a short time following termination of exposure*". RCA are permitted for law enforcement but prohibited from use as a method of warfare. The main group of RCA are lachrymators (tearing agents).

2. The most common lachrymators in use are:

   a. Orthochlorobenzylidene malononitrile (CS) (tear gas).

   b. Chloracetophenone (CN) (mace spray).

   c. Oleoresin capasicum (OC) (pepper spray).

3. Other RCA listed although not necessarily in common use include:

   a. Dibenzoxazepine (CR).

   b. Chloropicrin (PS), also considered a pulmonary agent.

   c. Bromobenzylcyanide (CA).

4. For the purposes of general management of the most common RCA, CS is described in detail although the management of RCA exposure is similar for CS, CN, OS and CR. For choropicrin, see Chapter 21 – Pulmonary Agents.

24.2.1 CS (ORTHOCHLOROBENZYLIDENE MALONONITRILE)

1. CS is used as a riot control agent in many countries. It is also commonly used as a training agent for simulation of chemical warfare agent exposure and in the testing of respirators performance. The limit of the threshold of human detection (slight irritation of the nasal passages) of CS is approximately 0.004 $mg.m^{-3}$. The minimal irritant concentration ranges from 0.1 to 1.0 $mg.m^{-3}$ and intolerable signs and symptoms of exposure occur at concentrations of 4.0 to 10.0 $mg.\ m^{-3}$. The estimated human lethal concentration time of CS is between 25 000 and 150 000 $mg.min.m^{-3}$ giving a safety ratio of the order of 25 000 to 1 500 000, assuming an exposure of up to 10 minutes.

2. *Properties*. On account of its potent irritant effects and its lower toxicity CS has superseded CN as the riot control agent of choice. It is a white crystalline solid substance. Solubility is very poor in water, moderate in alcohol and good in acetone, chloroform, methylene dichloride, ethylacetate and benzene. CS is unstable in aqueous solution. CS is usually dispersed as an aerosol generated pyrotechnically, but it can also be disseminated by spraying a solution of CS in a suitable solvent. Solvents may contribute to adverse effects including eye damage.

3. *Persistency*. Although the smoke is non-persistent, CS may stick to rough surfaces (e.g., clothes) from which it is released only slowly. At least 1 hour of aeration is necessary to cleanse such materials from CS after exposure.

4. *Detection*. No field detectors for CS exist. The CS smoke is white at the point of release and for several seconds after release. Exposure is associated with a pepper-like odour, and the onset of intense eye irritation, dyspnoea, coughing and rhinorrhoea.

5. *Protection*. Full individual protective equipment will provide complete protection. Protection against field concentrations of CS is provided by the respirator and ordinary field clothing secured at the neck, wrists and ankles. Individuals who handle CS should wear rubber gloves, hood, rubber boots, rubber apron and respirator and secure their field clothing at the neck, wrists and ankles.

6. *Decontamination*. Exposed persons should if possible move to fresh air, separate from other contaminated individuals, face into the wind with eyes open and breathe deeply. Contaminated eyes and skin should be flushed with copious amounts of water. Following exposure, clothing and individual equipment should be inspected for residue. If a residue is found, individuals should change and wash their clothing to protect themselves and other unmasked persons.

7. *Mechanism of Action*. Lachrymators act on the nerve endings, the cornea, mucous membranes and the skin. The reaction is very rapid.

8. *Signs and Symptoms*. Exposure to CS causes the following symptoms:

a. *Eyes.* Symptoms include a violent burning sensation, conjunctivitis (lasting up to 30 minutes), erythema of the eyelids (lasting about an hour) blepharospasm, violent lachrymation (over 10-15 minutes) and photophobia.

b. *Respiratory Tract.* The first symptom is a burning sensation in the throat, developing into pain and extending to the trachea and bronchi. At a later stage a sensation of suffocation may occur, often accompanied by fear. In addition a burning sensation in the nose, rhinorrhoea, erythema of the nasal mucous membranes and sometimes mild epistaxis occurs. The sense of taste is often distorted for some hours after exposure. Nausea, diarrhoea and headache have been observed. Sneezing occurs after mild exposure and may be persistent. Many exposed people have reported fatigue for some hours afterwards. Coughing, choking, retching and (rarely) vomiting occur after exposure. Exposure to high concentrations of CS may result in pulmonary oedema.

c. *Skin.* A burning sensation occurs especially in moist areas, but soon disappears. This burning sensation may recur some hours later, often while washing the area. Prolonged exposure to large amounts (e.g., when handling CS in bulk) can cause erythema and vesicle formation. Prolonged exposure, continuous or intermittent, to high

concentrations, combined with high temperatures and humidity in the field may result in a cumulative effect. Sensitivity to CS may be provoked.

9. *First Aid*. In practically all cases it is sufficient to remove the casualty into fresh air where the symptoms will soon disappear. Clothing should be changed. If symptoms persist, the eyes, mouth and skin may be washed with water (and with soap in the case of the skin). Oil based lotions should *not* be used. Skin decontaminants containing bleach should *not* be used, but should be reserved for more dangerous contamination (e.g., vesicants or nerve agents); bleach reacts with CS to form a combination which is more irritant to the skin than CS alone.

10. *Treatment*.

a. *Eyes.* Ordinarily the eye effects are self-limiting and require no treatment. If large particles or droplets of agent have entered the eye, treatment as for corrosive materials may be required. Prompt irrigation with copious amounts of water is the best treatment for solid CS in the eye. After complete decontamination corticosteroid eye preparations may be used in consultation with an ophthalmologist.

b. *Skin.* Early erythema and stinging sensation (up to 1 hour), especially in warm moist skin areas, are usually transient and require no treatment. Inflammation and blistering similar to sunburn may occur after heavy or prolonged exposure, especially in fair skin. Acute contact dermatitis should be managed initially in the same way as any other acute dermatitis. Corticosteroid cream or calamine lotion may be applied to treat existing dermatitis or to limit delayed erythema. Significant pruritus can be treated with calamine lotion or corticosteroid preparations. If blisters develop these should be treated as any other second degree burn. Secondary infection is treated with appropriate antibiotics.

c. *Respiratory effects.* In the rare event of pulmonary effects from massive exposure, evacuation is required. Management is the same as that for pulmonary agent (see Chapter 7 and Chapter 21).

24.3. PHARMACEUTICAL BASED AGENTS

1. The opportunistic or planned use of pharmaceuticals is unlikely to have a mass lethal effect on the battlefield when compared to other agents such as nerve agents. However some drugs may have specific properties such as a sedative effect and be used as an incapacitant or as a poison. The regulation of pharmaceuticals under CWC is limited and in some countries, both human and veterinary pharmaceuticals are more widely available. Some pharmaceuticals may be potent enough at supra-therapeutic doses to have the potential to be used over a wider area or confined space.

2. PBA of interest can generally be described as sedatives and be classed into pharmacology groups. Potential PBA include:[1]

a. Opioids.

b. Benzodiazepines.

c. α_2-adrenoceptor agonists.

[1] Adapted from Tracey & Flower. The warrior in the machine: neuroscience goes to war. *Nature Reviews Neuroscience* 15(2014) 825-834.

d. Dissociative anaesthetics.

e. Inhalational anaesthetics.

24.3.1. DOSE RESPONSE AND SAFETY

1. PBA differ to the traditional chemical agents because they have an effective or therapeutic dose. For the drug to be licensed, it must have a safety profile with a wide therapeutic index (ratio). There is a calculation comparing the effective dose for its use in 50% of the population (ED_{50}) compared to its toxic or lethal dose in the same population (TD/LD_{50}). Where the PBA might be used to sedate a person, the ED_{50} can also be considered the incapacitating dose 50 (ID_{50}) (see Figure 24-1).

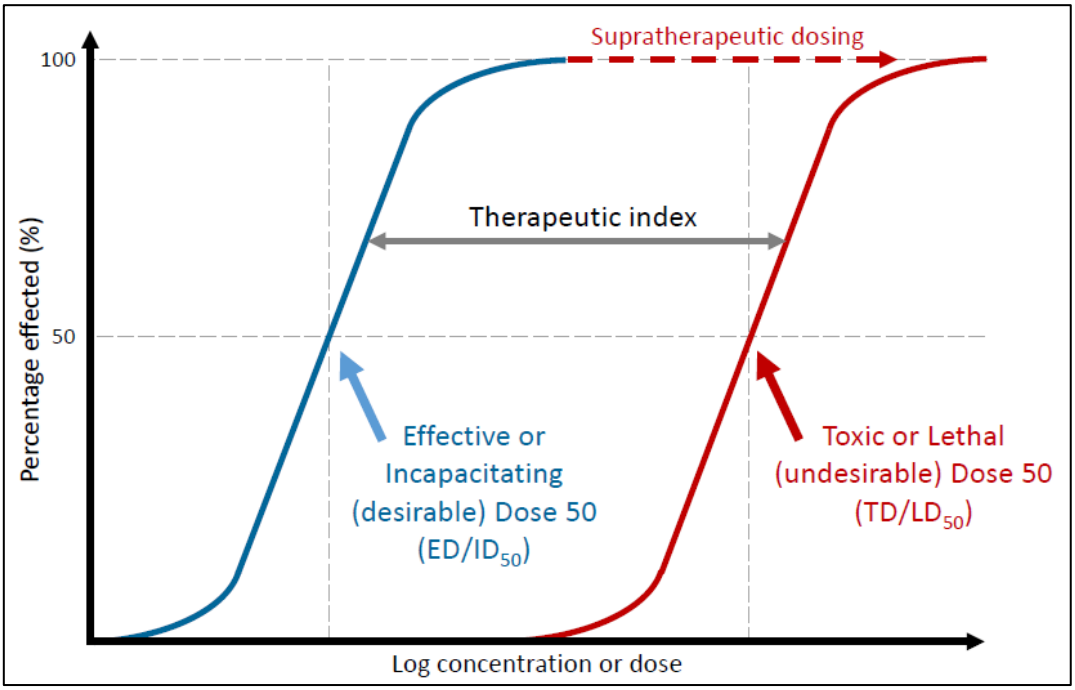

Figure 24-1: Dose Response and Therapeutic Index.

2. While PBA may also be classed as '*non-lethal agents*', the risk of overdosing or an adverse drug reaction (e.g. vomiting) means that no sedative PBA is non-lethal and are better described as '*low-lethality*'.[2] Even at an effective incapacitating dose, an unconscious person may obstruct their airway and sustain hypoxic brain injury with fatal consequences.

3. The potential causes for toxic or lethal effects during PBA use as a law enforcement or 'non-lethal' weapon includes:

a. Non-uniform distribution of the PBA in the target area.

b. Over-dosing of target area.

c. Failure to clear PBA from the target area.

[2] The preferred NATO term for a sedation PBA is 'low-lethality'.

d. Increased vulnerability to the PBA for reasons including drug sensitivity, altered pharmacokinetics due to target state (e.g. dehydration, positioning).

e. Varying respiratory state, such as minute volume, and therefore total inhaled dose received.

f. Sedation causes airway obstruction, collapse, or fall with concurrent trauma.

g. Failure to deliver antidote, if available, or supportive care.

h. Lack of human data to support dosing, concentration and duration.

24.3.2. VETERINARY PHARMACEUTICALS

Veterinary pharmaceuticals may be less well known to medical personnel and have unanticipated effects in human subjects. The following issues should be considered:

a. Drugs are likely to be potent to support the use of the intramuscular route (e.g. for use in darting).

b. Drugs may have been considered for human use also but failed clinical safety trials due to human-specific toxicity.

c. Drugs may have been considered for human use also but failed clinical safety trials due to lack of efficacy for the intended licensed use in humans.

d. Drugs may also have antidotes that are unlicensed for human use.

e. Drugs may be less regulated and controlled in secure areas.

24.3.3. GENERAL MANAGEMENT OF SEDATED CASUALTIES

1. The general management of sedated casualties is supportive. At supratherapeutic doses, sedatives may have a toxic effect on the respiratory and cardiovascular system causing hypoxia, cardiac dysrhythmias and hypotension. Additionally the airway may be obstructed due to position, foreign body or vomit. Casualty management follows the same priorities as for other CBRN agents[3] and includes:

a. Any airborne agent should be cleared / ventilated as soon as possible or casualties removed from the target area.

b. Any contamination should be removed but not delay the provision of LSI by suitably protected personnel.

c. Treat any catastrophic haemorrhage, if present.

d. Basic airway management and suction.

e. Administer any antidote, if available.

f. Provide adequate respiratory support including oxygen and ventilation.

[3] <C>AaBC-Decon-Evac (see Chapter 4)

g. Where there is cardiac toxicity, the heart should be monitored, dysrhythmias corrected and any hypotension managed initially with fluids and/or vasopressors and inotropes.

2. Any further drug specific information is provided below.

24.3.4. OPIOIDS[4]

1. Opioids may be encountered in a number of scenarios including iatrogenic use, overuse of morphine auto-injectors and use of narcotics. The use of a fentanyl derivative during the Moscow Theatre siege also raises the use of aerosolised opioids either by a host nation, protagonist or aggressor for mass effect. Examples include fentanyl, alfentanyl, carfentanil and remifentanil.

2. *Mechanism*. Opioids are generally centrally acting agonists of the μ and κ receptors.

3. *Acute effects*. Symptoms and signs of opioid intoxication include:

a. Euphoria (early sign).

b. Miosis.

c. Respiratory depression.

d. Reduced level of consciousness.

e. Cyanosis.

f. Respiratory arrest.

4. *Treatment*. The management of opioid casualties includes supportive care and the use of antagonists such a *naloxone* (initial dose 200 – 2000mcg). Depending on the type of agent, route of exposure, dose and pharmacokinetics, the initial dose and continuing requirements may be varied. The effect of the opioid may outlast the effect of the antidote and continuing treatment by infusion may be required or use of a longer acting antagonist such as *naltrexone*.

24.3.5. BENZODIAZEPINES

Benzodiazepines are widely used drugs used as anxiolytics, sedation and muscle relaxation. They potentiate $GABA_a$ receptors and examples include diazepam, lorazepam, midazolam and temazepam. All may cause respiratory and cardiovascular depression. Treatment is generally supportive although an antagonist, flumazenil, is available for iatrogenic overdoses although its use should be avoided if the agent is unknown or there is a risk of unopposed convulsions.

24.3.6. α_2-ADRENOCEPTOR AGONISTS

α_2-adrenoceptor agonists include dexmedetomidine, clonidine and the veterinary medetomidine and xylazine. Dexmedetomidine and clonidine are used mainly on critical care units for sedation and no significant respiratory depression at therapeutic doses is expected.

[4] The term *opioids* refers to synthetic drugs with opiate-like actions. Opiates describe a group of drugs derived from opium.

It can cause cardiovascular depression. Treatment is supportive. There is no human antidote, however there is a reversal agent for veterinary use called atipamezole.

24.3.7. DISSOCIATIVE ANAESTHETICS

Dissociative anaesthetics such as ketamine are used for analgesia, sedation and anaesthesia depending on dose. Ketamine is a glutamate-NMDA receptor antagonist which causes a trance like state while maintaining airway and breathing reflexes and little respiratory or circulatory compromise. It is the induction agent of choice in some nations for trauma due to its safety profile and cardiovascular stability. It is also a drug of choice for induction in asthma as it relieves bronchospasm. It may cause sensory distortion and psychiatric effects similar to some of the psychotropic incapacitating agents such as BZ and LSD. Effects are generally short duration and treatment is supportive. Any psychiatric disturbance may require sedation usually with a benzodiazepine and avoiding physical restraint and risk of injury or heat illness.

24.3.8. INHALATIONAL ANAESTHETICS

Inhalational anaesthetics are gases and volatile liquids that, when inhaled, reduce synaptic function due to uptake into the lipid bilayer in cell membranes. The onset of effects is dependent on the concentration of the agent. Inhalational anaesthetics include halothane, isoflurane and sevoflurane. Most of these agents have odours and may cause irritation of the mucous membranes, although sevoflurane is generally pleasant and rapid acting. Most agents will cause loss of consciousness but may also be associated with increased respiratory and cardiovascular depression at higher concentrations. Treatment is supportive and good respiratory ventilation is vital in order to allow for off-gassing as well as redistribution of the lipid-soluble agent.

Note: Some chemical agent monitors using vapour ionisation mobility studies may mistake inhalational anaesthetic agents as chemical agents such as nerve agent and sulphur mustard vapour.

24.4. POST-EXPOSURE CLINICAL SAMPLING

Following any possible exposure to a PBA or unknown RCA, clinical sample may be used to determine causation. Initial management will be supportive and in the case of opioids will be based on clinical symptoms and signs (toxidrome recognition). Some PBA may be detected using environmental sampling if a CBRN specialist unit is present with appropriate equipment and agent catalogue. Handheld assays used for drug of misuse assay may also detect narcotic based agents. However in many cases, clinical samples may require an analytical laboratory with mass spectroscopy and chromatography. Suitable samples include early blood samples ideally using glass and non-rubber bungs or a urine sample. The effectiveness of a urine sample depends on the agent and its elimination. However, the agent, its metabolites or adjuncts may be present for several days. Advice should be sought from the receiving laboratory on appropriate volume and sample handling required.

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CHAPTER 25: MILITARY SMOKES AND INCENDIARIES

25.1. MILITARY SMOKES

1. Smokes are used to obscure troops and equipment on the battlefield as well as marking locations. They consist of liquid or solid particulate aerosols that disrupt portions of the electromagnetic radiation at wavelengths ranging from the microwave / infrared through the visible part to the ultraviolet end of the spectrum. Some smokes may also be used for training.

2. The general smoke types are:

   a. Petroleum oil smokes.

   b. Zinc oxide mixtures (HC).

   c. Sulphur trixoxide-chlorosulphuric acid (FS).

   d. Titanium tetrachloride (FM).

3. Not all smokes are hazardous in concentrations which are used for obscuring purposes. However, exposure to heavy smoke concentrations for extended periods, particularly if near the source of emission, may cause acute and long-term health effects, and even death. Medical personnel should, therefore, be prepared to treat potential reactions to military smokes once smokes have been introduced to the battlefield although the mainstay is supportive management. Acute exposures may occur during the handling of corrosive acids used for generation of some smokes (see below).

4. The standard respirator gives the respiratory tract and eyes adequate protection against all smokes and should always be worn when smokes are used in confined spaces. In the open air, a respirator should be considered if:

   a. The smoke irritates the airway.

   b. The smoke is very thick (i.e. loss of all sight through the cloud).

   c. The stay time is longer than 5 minutes in a diluted cloud.

   d. The smoke is likely to or has exceeded safe limits.

Note: Despite protection against aerosols, respirator will not protect against carbon monoxide, a common by-product of combustion including smoke generation. Avoidance is also generally preferred to the use of protective equipment.

25.1.1. PETROLEUM OIL SMOKES

These are some of the less harmful of the military smokes. They are generated by the use of generator to vaporise fuel such as petroleum oil or diesel. The fuel condenses in the cooler air to form a dense white smoke. Some mineral oils are also used in some nations for smoke generation including the validation of respirator fitting. Although ill effects are seldom seen, respirator protection should be worn for prolonged periods of smoke exposure.

25.1.2. ZINC OXIDE / HEXACHLOROETHANE SMOKES

1. Several methods of producing smoke by dispersing fine particles of zinc chloride have been developed. The mixture in common use contains zinc oxide and hexachloroethane, as well as aluminium powder. Upon burning, the mixture produces zinc chloride, zinc oxychlorides and HCl vapour which rapidly absorb moisture from the air to form a greyish white smoke. HC mixtures can be dispersed by several methods, including grenades, candles, smoke pots, cartridges, and air bombs.

2. *Protection*. Respirator use should be considered to protect the respiratory tract whenever HC smokes are used. In some countries, this is mandatory.

Note: High concentrations of zinc oxide / hexachloroethane (HC) smoke have caused fatalities. Under no circumstances should HC munitions be used indoors or in closed compartments.

3. *Mechanism of Action*. The production of grey-white smoke clouds is based on the pyrotechnic reaction between powdered zinc or zinc oxide and hexachloroethane. The reaction produces a number of compounds including zinc chloride and zinc oxychloride. Phosgene may also be produced although the concentration in the cloud is likely to be low. Other intermediates and products may include hydrogen chloride, tetrachloroethylene, carbon tetrachloride and carbon monoxide. Many of these chemicals have been implicated in respiratory toxicity following exposure but experimental studies suggest that HC inhalation produces a pattern of pulmonary disease that is identical to that seen following smoke inhalation.

4. *Acute effects*. In high concentration or following prolonged exposure, HC smoke is highly irritating and generalised chemical pneumonitis has been reported following accidental overexposure of human subjects. Symptoms following inhalation of high concentrations of HC smoke include dyspnoea, hoarseness of the voice, retrosternal pain, cough, lachrymation and occasionally haemoptysis. In many cases reported in the literature, the latter development of pulmonary oedema has been a consistent finding.

5. *Chronic effects*. Following the resolution of the initial pulmonary inflammatory reaction and oedema, there is evidence of several additional chronic effects following inhalation of HC smoke. These include: focal atelectasis, bronchiolar-alveolar hyperplasia and pulmonary fibrosis. More recently, concerns have been raised regarding the possible mutagenicity/carcinogenicity of some of the components of the smoke, and in particular unburnt hexachloroethane and tetrachloroethylene. Studies of both *in vitro* and *in vivo* unscheduled DNA synthesis have, however, confirmed an overall lack of genotoxic effects in smoke condensates.

6. *Treatment*. The casualties should put on their respirator or be removed from the source of exposure. Oxygen should be administered in cases of hypoxia. Bronchospasm should be treated appropriately including inhaler bronchodilators and even intramuscular epinephrine. Early steroid therapy has also been considered. Adequate symptom relief and supportive treatment is recommended (see Chapter 7 and Chapter 21).

7. *Prognosis*. The prognosis is related entirely to the extent of the pulmonary damage. All exposed individuals should be kept under observation for 24 hours. Most individuals recover in a few days. At moderate exposures, some symptoms may persist for 1 to 2 weeks. In severe exposures, survivors may have reduced pulmonary function permanently if pulmonary fibrosis results. The severely exposed patient may progressively develop pulmonary oedema, resulting in dyspnoea, cyanosis and possible death.

25.1.3. SULPHUR TRIOXIDE-CHLOROSULPHONIC ACID (FS)

1. Chlorosulphonic acid (FS) is a heavy, strongly acidic liquid which, when dispersed in air, absorbs moisture to form a dense white fog consisting of small droplets of hydrochloric and sulphuric acids. In moderate concentrations it is highly irritating to the eyes, nose and skin. The respirator should be worn in all concentrations which are sufficient to cause any cough, irritation of the eyes or prickling of the skin. A risk exists when FS comes in contact with water due to the generation of intense heat and the scattering of acid in all directions. Owing to its highly corrosive nature careful handling is required.

2. *Symptoms*. The symptoms are usually limited to a prickling sensation of the skin, but exposure to high concentrations or long exposures to lower concentrations as found in the field, may result in severe irritation of the eyes, skin and respiratory tract. Conjunctival irritation and oedema, lachrymation and mild photophobia may occur. Mild cough and soreness in the chest and moderate chemical dermatitis of the exposed skin are occasionally seen. Splashes of liquid in the eye are extremely painful and cause acid burns with corneal damage. Liquid FS solution on the skin may cause painful acid burns.

3. *Treatment*.

a. *First aid*. Remove from area immediately with respiratory protection worn, if available. Irrigate the skin and eyes if any direct liquid contact or symptoms with copious amounts of water or saline. Seek medical attention if symptomatic, or any eye contact or injuries suspected.

b. *Eye.* Irrigate the contaminated eye with water or saline as soon as possible. Examine the cornea for damage by staining it with fluorescein. If corneal damage is present, the casualty should be managed with chloramphenicol eye ointment and referred to an ophthalmologist. For eye pain, consider mydriasis with the use of atropine, tropicamide or cycloplegic eye drops or ointment. Conjunctival lesions should heal readily, but corneal erosions may lead to residual scarring.

c. *Skin.* Irritated skin or skin burns should be washed with water and then with sodium bicarbonate solution. The burns are then treated as for thermal burns of like severity.

d. *Inhalation.* Any pulmonary effects should be treated symptomatically with inhaled bronchodilators and if severe cases supportive care similar to pulmonary agents described in Chapter 7 and Chapter 21.

4. *Prognosis*. The skin burns, conjunctival lesions and respiratory irritation heal readily. Corneal damage is more serious and may lead to residual scarring.

25.1.4. TITANIUM TETRACHLORIDE (FM)

1. This is a yellow corrosive fluid which on contact with damp air gives off a heavy dense white cloud. It is disseminated by aircraft for the production of vertical smoke curtains extending down to ground and sea level. The smoke consists of fine particles of free hydrochloric acid and titanium oxychloride. The smoke is unpleasant to breathe. Respirator should be worn when the spray is falling due to the risk of droplets entering the eyes. Full protective clothing should be worn when handling the liquid to avoid contamination of eyes and skin.

2. *Effects and treatment*. Liquid FM produces acid burns of the skin or eyes and is treated similar to FS described above.

25.2. INCENDIARY AGENT

1. An incendiary agent is a chemical, or mixture of chemicals, that liberates a large quantity of heat upon combustion. The reaction is that of a fuel with oxygen. Incendiary agents can be classified according to their composition:

a. Hydrocarbon fuels with or without thickeners (normally gasoline with a napalm aluminium soap thickener and with or without polymer thickeners).

b. Metal fuels (magnesium incendiaries or thermite incendiaries).

c. Hydrocarbon-metal fuel combinations.

d. Pyrophoric aluminium alkyls with thickeners (e.g., triethyl aluminium thickened with polyisobutylene).

e. White phosphorous (WP).

f. Red phosphorous (RP).

2. Thickeners are added to fuels to increase the range of flamethrowers, to impart slower burning properties, to impart clinging qualities and to cause flames to rebound off surfaces and go around corners.

a. Napalm aluminium soap – mixed aluminium soap co-precipitated from a mixture of coconut oil and oleic and naphthenic acids.

b. Polymers (e.g., isobutyl methacrylate, polyisobutylene).

c. Thickened fuels are generally used in mechanical or portable flamethrowers or in incendiary bombs.

3. Incendiary weapons are used for anti-personnel and anti-material operations and military operations in urban territory.

Note: The principal action of flame and incendiary weapons is to cause burns. Casualties will be treated as burn casualties not as chemical casualties.

25.2.1. WHITE PHOSPHOROUS

1. At ordinary temperatures, white phosphorus (WP) is a solid which can be handled safely under water. When dry, it burns fiercely in air, producing a dense white smoke. Fragments of melted particles of the burning substance may become embedded in the skin of persons close to a bursting projectile, producing burns which are multiple, deep and variable in size. The fragments continue to burn unless oxygen is excluded by flooding or smothering.

2. WP may be used to produce a hot dense white smoke composed of particles of phosphorus pentoxide which are converted by moist air to droplets of phosphoric acid. The smoke irritates the eyes and nose in moderate concentrations. Field concentrations of the smoke are usually harmless although they may cause temporary irritation to the eyes, nose or throat. The respirator provides adequate protection against white phosphorus smoke.

3. In an artillery projectile white phosphorus is contained in felt wedges which ignite immediately upon exposure to air and fall to the ground. Up to 15% of the white phosphorus

remains within the charred wedge and can re-ignite if the felt is crushed and the unburned white phosphorus exposed to the atmosphere.

25.2.2. RED PHOSPHOROUS

Red phosphorus (RP) is less reactive as white phosphorus. It reacts slowly with atmospheric moisture and the smoke does not produce thermal injury, hence the smoke is less toxic.

25.2.3. MANAGEMENT OF PHOSPHOROUS BURNS

1. *First Aid*.

   a. If burning particles of phosphorus strike and stick to clothing, contaminated clothing should be removed quickly before the phosphorus burns through to the skin.

   b. If burning phosphorus strikes the skin, smother the flame with water, a wet cloth, or mud. Keep the phosphorus covered with the wet material to exclude air until the phosphorus particles can be removed.

   c. Try to remove the phosphorus particles with a knife, bayonet, stick or other available object. It may be possible to remove some particles by rubbing with a wet cloth.

   d. It may be necessary to repeat the first aid measures to completely remove all phosphorus.

2. *Treatment*. At the earliest opportunity all phosphorus should be removed from the skin and placed in a container so as to prevent further contamination and injuries. The affected part should be bathed in an isotonic bicarbonate solution to neutralise phosphoric acid, which then allows removal of visible phosphorus. Remaining fragments will be observed in dark surroundings as luminescent spots.

3. The burn should be debrided promptly, if the patient's condition will permit, to remove bits of phosphorus which might be absorbed later and possibly produce systemic poisoning. An ointment with an oily base should not be applied until it is certain that all phosphorus has been removed. Further treatment should be carried out as for thermal burn.

4. If the eyes are affected, treatment should initially be commenced by irrigation with a 1% solution of copper sulphate or sodium bicarbonate 5%, followed by repeated lavage using water or saline. The lids must be separated and a local anaesthetic instilled to aid in the removal of all embedded particles. In eyes with severe ulceration once all particles have been removed, a mydriatic should be instilled. The patient should be transferred to the care of an ophthalmologist as soon as possible.

5. *Copper sulphate solution*. Copper sulphate reacts with phosphorous to form dark coloured deposits. Some nations consider washing the skin with a 0.5-2.0% copper sulphate solution and wounds may be rinsed with a 0.1%-0.2% copper sulphate solution, if available.

Note: There is a significant risk following the application of copper sulphate on to an open wound / burn of causing copper poisoning and some nations recommend against its use.

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AMedP-7.1 PART 4: MANAGEMENT OF THE BIOLOGICAL CASUALTY INCLUDING SEOSIS

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CHAPTER 26: INTRODUCTION TO BIOLOGICAL AGENTS

26.1. INTRODUCTION

1. The use of biological agents (BA) dates back to antiquity with the use of toxins and possibly live agents to enhance conventional weapons. Biological weapons (BW) are unique in their potential to inflict large numbers of casualties over a wide area with minimal logistical requirements and by means that can be virtually undetectable and untraceable. Although wide area delivery of a BW may be technically challenging, the ease and low cost of producing an agent, the difficulty in detecting its presence and protecting (and treating) its intended victims makes biological agents a signiciant hazard both for state and non-state actors.

2. BA could be targeted against animals or plants either to act as a vector for human disease or as a form of warfare targeting self-sufficiency or economic stability. The possibility to selectively target humans, animals, or plants conspire to make defence against this class of weapon particularly difficult.

3. The cost of developing a BW is relatively low when compared to other CBRN and conventional weapons and can use emerging bio- and nanotechnology. Because of its dual-use (permitted and illegal use), biological technology is difficult to monitor.

4. The use of a contagious (transmissible) live agent means that the primary casualties become the delivery mechanism for further waves of casualties. A BW attack using an agent transmissible from man-to-man may spread to susceptible personnel and will require control measures such as RoM and other public health measures.

5. In contrast to all other weapon systems the full impact of a BW attack may take several days or even weeks to develop (slowly evolving scenario), and is difficult to predict during the early stages. A BW attack may take place against a background of continuing natural endemic disease already present in the operational area. The initial effects of a biological attack on exposed personnel may be difficult to distinguish from a natural outbreak of a disease. Biological incident (outbreak) management will differ from chemical or conventional incident management and may require operational epidemiology and the deployment of specialist teams (see Chapter 17).

26.1.1. BIOLOGICAL AGENTS OF OPERATIONAL SIGNIFICANCE

This concept relates to a spectrum of biological agents that may have an impact on operational effectiveness either due to deliberate, accidental or natural release. These include:

a. *'Biological warfare agents'*. These are a group of BA identified as agents of concern either due to intelligence, weaponisation programmes, defensive programmes or actual use. Examples includes anthrax and tularaemia.

b. *Endemic disease*. This includes natural diseases local to the deployed area. Examples include malaria or a filovirus such as Ebola endemic to a West African deployment.

c. *Emerging disease*. This includes diseases that may present a new threat. Examples include new strains of influenza and coronavirus (SARS / MERS).

d. *Imported disease*. This includes disease that are from out of area and may be be introduced by the movement of patients, personnel, animals, water or foodstuffs. Examples include norovirus, *Salmonella* sp. and cholera.

e. *'Bio-mimickers'*. These are a group of agents that may mimic a biological outbreak but are not a traditional biological agent. This might include low dose chemical agent exposure, chronic heavy metal exposure, and radiation prodromal symptoms appearing as an outbreak of food poisoning.

26.1.2. BIOLOGICAL AGENTS OF STRATEGIC SIGNIFICANCE

1. This is a term relating to a group of potential biological agents, as described above, that may:

a. Be used in an Article 5 attack (i.e. attack on a NATO nation by a hostile nation).

b. Cause an outbreak that is beyond the capability of the civilian health sector to respond effectively to.

2. In addition, a biological agent may have strategic significance if:

a. A formal request has been received by a civilian regional or international body e.g. WHO to assist due to global health, security and/or stabilisation issues (medical mission).

b. The outbreak is in a non-permissive environment for civilian responders or organisation to be able to work (security and stabilisation mission).

c. The biological agent has agricultural significant that may cause global health, economic, security and/or stabilisation issues.

26.2. TYPES OF BIOLOGICAL AGENTS

1. The medical classification of BA is important to the medical services in terms of identification, prophylaxis, and treatment. BA may be genetically modified in order to evade standard detection, identification and MedCM. Disease-causing BA (pathogens) that may be used as weapons can be classified as follows:

a. *Live biological agents*. Live BA (micro-organisms) usually have the shared characteristics of self-replication, infectious dose, incubation period and varying virulence. They do not have consistent dose-dependent observable effects as there is significant host variation. Live BA are also susceptible to specific forms of hazard management including physical removal and biological decontamination (sterilisation). Live BA include:

(1) *Bacteria*. Bacteria are small free-living organisms, most of which may be grown on solid or liquid culture media. The organisms have a structure consisting of nuclear material, cytoplasm, and cell membrane. They reproduce by simple division. Some bacteria such as rickettsias and chlamydia can only grow inside host cells and therefore cannot be grown readily on artificial media. Other bacteria, for example that cause anthrax, can form spores that enable them to survive for long periods in a dormant state in the environment. Diseases produced by bacteria often respond to specific therapy with antibacterial drugs such as antibiotics. However, bacteria can

become resistant to antibiotics, either naturally, including following the use of antibiotics or through bioengineering.

(2) Viruses. Viruses are organisms that require other living cells (animal or bacteria) in which to replicate. They produce diseases which do not respond to antibiotics but which may be responsive to antiviral compounds, of which there are few available, and those that are available are of limited operational use.

(3) *Fungi*. Fungi are simple organisms widespread in nature. Most fungi form spores, and are free-living forms that are found in soil. Fungal diseases may respond to various antifungal drugs. Although some fungi are capable of causing disease in humans they are unlikely to be used as a BW agent. Endemic fungi may cause wound infections if gross contamination occurs. Fungi are not considered further in this document.

b. *Toxins*. Toxins are poisonous substances produced and derived from living organisms including bacteria, animals (venom) and plants. Toxins may be countered by specific antisera although there is only a limited range available. Hazard management for toxins is similar to that for chemical agents.

2. Table 28-3 provides a summary of biological agent properties, in order to provide guidance to field commanders on their impact on operations. Operational consideration of the impact of an agent may be based upon lethality (anthrax, plague), infectivity (toxaemia, Q fever), transmissibility (plague, smallpox) and persistence (anthrax spores).

26.3. CHARACTERISTICS OF BIOLOGICAL AGENTS

1. Intrinsic features of BA that influence their potential for use as weapons include: infectivity, virulence, toxicity (toxins), pathogenicity, incubation period, transmissibility, lethality and stability. Infectious organisms have to multiply within the body in order to cause disease, in contrast to toxins where the effect is dependent upon the dose received.

a. *Transmissibility*. Some infectious BA can be transmitted directly from person-to-person (smallpox, pneumonic plague). Indirect transmission (e.g. via arthropod vectors such as body lice) may also be a significant means of spread. In the context of BW defensive operations the relative ease with which an agent is passed from person-to-person (transmissibility) constitutes a primary concern and may require casualty hazard management including containment and isolation as well as physical protection (individual protective equipment). For a severe outbreak of a transmissible biological agent, quarantine and RoM should be considered. Transmissibility can be described by R_0, the reproduction ratio, and is described in Chapter 17.

b. *Pathogenicity*. This reflects the capability of an infectious BA to cause disease (pathology) in a susceptible host.

c. *Virulence*. The virulence of an agent reflects the relative severity of disease produced by that BA. Different microorganisms and different strains of the same microorganism may cause diseases of different severity and resistance to treatment. An example of a virulent strain is that of the *Escherichia coli* (*E.coli*) bacteria O157.

d. *Infectivity*. The infectivity of a BA reflects the relative ease with which microorganisms establish themselves in a host species. Pathogens with high infectivity cause disease with relatively few organisms (low infective dose), while those with low

infectivity require a larger number (high infective dose). This may not be linear dose response. High infectivity does not necessarily mean that the symptoms and signs of disease appear more quickly, or that the illness is more severe. Infection may initially be asymptomatic.

e. *Infectious Dose (ID)*. The dose of an organism needed to infect a person varies widely between individuals and is therefore usually given as the median infectious dose (ID_{50}) – the dose needed to infect 50% of those exposed. The dose required to infect all the population may be 100 or 1000 times greater than this in some cases, while a few percent of the population may be infected by much smaller doses. The effect of MedCM, such as chemoprophylaxis and vaccination, is to increase the median infectious dose for the protected population.

f. *Toxicity*. The toxicity of a BA applies only to toxins. It reflects the relative severity of illness or incapacitation produced by a toxin, and is dose dependent. The relative effects of each agent, usually toxins, can be compared using the median dose to cause the observed effect (lethality (LD_{50}), incapacitation (ID_{50})).[1]

g. *Incubation (Latent) Period*. A sufficient number of microorganisms or quantity of toxin must penetrate the body to initiate infection or intoxication. Live agents must then multiply (replicate) to produce disease. Although toxins do not multiply, once in the body they need time to reach target organs (toxicokinetics) before producing their effects. The time between exposure and the appearance of symptoms is known as the incubation period (microorganisms) or latent period (toxins). This is governed by many variables, including the initial dose, virulence, route of entry, rate of replication and host immunological factors.

h. *Lethality*. Lethality reflects the relative ease with which an agent causes death in an infected host. Lethality can be described as the *case fatality rate* expressed as the number of deaths in relation to the number of cases. For toxins, this is dose dependent and is expressed by the lethal dose as is similar to chemical agents (see below).

i. *Lethal Dose*. The dose of a dose dependent BA, usually toxin, needed to cause death in a given population of individuals. It is usually given as the median lethal dose (LD_{50}), i.e. the dose needed to kill 50% of those exposed and is useful when comparing toxicity between various toxins and chemical agents.

j. *Stability (persistency)*. Stability may describe the viability of an agent in a weapon or other delivery device, during dissemination and after deposition. The viability of an agent once deployed is affected by various environmental factors including temperature, relative humidity, atmospheric pollution and sunlight (UV sensitivity). An example of a non-persistent biological agent is the influenza virus which is sensitive to UV light while spores such as anthrax are very stable and will persist in the environment for years.

2. Additional factors that may influence the suitability of a BA as a biological weapon include ease of production, stability when stored or transported and ease of dissemination.

26.4. DISSEMINATION OF BIOLOGICAL AGENTS

1. Dissemination is the process by which BA are dispersed to cause infection, disease or intoxication. The same routes of entry pertinent to natural spread of diseases (that is, through

[1] ID_{50} is used for both infective dose (live agents) and incapacitating dose (chemicals and toxins).

inhalation, ingestion, or percutaneous inoculation) are also relevant when the BA is delivered intentionally by weaponisation. BA are most likely to be delivered covertly either by aerosol or the food chain. Other routes of entry are thought to be less important than inhalation but are nonetheless potentially significant.

a. *Aerosol*. BA can be delivered effectively by a wide range of platforms. The agent can be formulated as either a liquid or dry powder fill. The dissemination can be performed using simple or sophisticated spray devices, by an explosive charge, or simply packaged and delivered e.g.in the regular mail. Most forms of aerial delivery, including bombs, shells, missiles and low flying aircraft sprays can be deployed. Spray devices can also be effective from ground level. Depending on the efficiency of the delivery system used, some agent may be destroyed at the time of release, larger particles (droplets) will fall to the ground producing local contamination while any respirable particles (aerosols) generated will present predominantly as an inhalation hazard travelling long distances downwind. Aerosol delivery systems aim to generate invisible clouds with particles between 0.5 and 10 microns in diameter, which can remain suspended for significant periods.

b. *Contamination of Food and Water*. Direct contamination of consumables, such as drinking water or foodstuffs, could be used as a means to disseminate infectious (live) agents or toxins. Some foodstuffs, for example chocolate, can allow organisms, especially spores, to survive for long periods, and significantly reduce the number of organisms required to cause disease. This method of attack would be most suitable for sabotage activities and might be used against limited targets such as water supplies or food supplies of a military unit or base. Water purification systems significantly reduce this hazard, but supplies may be contaminated following treatment.

2. Other considerations include:

a. Attempts might be made to spread typical vector-borne diseases by releasing infected natural (or unnatural) arthropod hosts such as mosquitoes, ticks or fleas. These live vectors can be produced in large number and infected by allowing them to feed on infected animals, infected blood reservoirs, or artificially-produced sources of a BA.

b. Long-term survival of infectious agents, preservation of toxin activity during extended periods, and the protective influence of dust particles onto which microorganisms adsorb when spread by aerosols have all been documented. The potential exists, therefore, for the re-suspension of infectious particles from previously contaminated surfaces. To a lesser extent, particles may adhere to an individual or to clothing creating additional but less significant exposure hazards.

c. A persons may unknowingly be a carrier of a transmissible agent and thereby become an effective mechanism for dissemination (for example, with plague, or smallpox). Consideration should be given to movements of non-military populations including displaced persons, and refugees and local employed contractors.

26.5. MANAGEMENT OF BIOLOGICAL CASUALTIES

The management of a biological casualty and potential outbreak or attack, whether due to deliberate release, natural or other type of biological agent of operational significance, is based upon:

a. A syndromic approach to biological casualty assessment (See Chapter 27).

b. Appropriate diagnostic sampling (see Chapter 29 and Annexes).

c. Early treatment including the use of biological MedCM (See Chapter 29).

d. Infection control and prevention, including isolation and quarantine (see below).

e. Wider public health interventions including RoM (see Chapter 6).

f. Operational epidemiology (see Chapter 17).

26.6. MEDICAL COUNTERMEASURES FOR BIOLOGICAL AGENTS

MedCM used for the protection against biological agents follow the same classification described in Chapter 3 and may include:

a. Active immunisation (live and inactivated vaccines).[2]

(1) Routine vaccination as part of a wider vaccination programme including public health and travel vaccinations.

(2) Pre-deployment (operational) vaccination.

(3) Post-exposure vaccination.

b. Passive immunisation.

(1) Anti-toxins including immunoglobulins and Fab fragments.

(2) Human immunoglobulin.

(3) Agent specific immunoglobulins.

c. Antibiotics (antibacterials) including pre-exposure and post-exposure prophylaxis and treatment.

d. Antivirals, usually as treatment but limited selection.

e. Immunomodulation, this is the enhancement of the host's immune system by use of agents such as cytokines.

26.7. INFECTION PREVENTION AND CONTROL

1. Chapter 6 provides general guidance on casualty hazard management and the principles apply to biological agents. There are specific considerations for the level of isolation and decontamination (sterilisation and disinfection) required and depends on transmissibility from person to person, the route of infection and the stability of the agent. The hazard may be due to the BA or the method of emanation i.e. aerosolising procedure, or blood spill and risk of spore formation.

2. Measures for the control of transmissible infections include isolation with negative pressure clinical areas, personal protective equipment, hand hygiene, good clinical practice, wound management, and blood spill and waste management.

[2] Vaccination is not available for all BA or may not be used by all nations.

3. The methods of transmission during patient contact are:

a. *Contact hazard*. This is through direct contact with surface contamination. Control of this hazard is through standard precautions (see below) and hand hygiene.

b. *Droplet hazard*. Particles that are >5 microns in size are generally generated by coughing, sneezing and talking. These particles, especially over 10 microns, are unlikely to travel any great distance (approximately 2 metres). Within this distance respiratory precautions are recommended in addition to standard precautions as well as eye protection.

c. *Airborne hazard*. Particles that are <5 microns in size are likely to remain suspended in the air and travel significant distances and through air ventilation systems. The presence of an airborne hazard may be due to the particle size of the organism (smallpox) or by an aerosolising procedure such as intubation, suction or cardiopulmonary resuscitation. Control of this hazard is through airborne infection isolation and standard precautions with high grade particulate respiratory filter (N95 or FFP2/3) and eye protection, as appropriate.

d. *Faecal-oral*. This hazard may be a contact hazard but it is also a hazard for conditions where there is poor sanitation and potential for faecal waste contaminating drinking water or food products.

26.7.1. MEDICAL PERSONAL PROTECTIVE EQUIPMENT

1. PPE for patient care is determined by the type of infection hazard, severity of the infection and clinical procedure (aerosolising procedure). The basic level of PPE is standard precautions with increasing levels of respiratory precautions and as general PPE is described in Chapter 9 Section 3 – *Individual and Personal Protective Equipment*.

2. *Standard precautions* include:

a. Heath care facility policy for infection control and prevention.

b. Hand hygiene.

c. PPE including:

(1) Gloves

(2) Gown

(3) Face protection including the eyes, mucous membranes and hair.

d. Respiratory hygiene and cough etiquette. In some cases, the use of a surgical mask for the casualty is advised to reduce respiratory secretions especially during transport.

e. Equipment and surface cleaning.

3. *Enhanced PPE* describes additional protective equipment such as double gloving and aerosol / airborne respiratory protection.

4. *Strict PPE* describes protective equipment and supporting robing and disrobing areas, hazard management, including waste and fatality management, to support the management of casualties with high level pathogens such as VHF.

5. *Laboratory Biosafety Levels (BSL)* are briefly described in Chapter 9.

CHAPTER 27: SYNDROMIC APPROACH TO BIOLOGICAL AGENTS

27.1. INTRODUCTION

1. Although medical planning for CBRN protection is based on a threat list of potential biological warfare agents, casualty presentations may initially be non-specific or present as a syndrome of symptoms and signs. Clinical assessment of casualties should therefore be based upon a syndromic approach. The biological syndromes described may be due to the agent's pathological properties acting at a specific site such as cholera enterotoxin acting on colonic chloride channels or viral haemorrhagic fever causing a coagulopathy. For other agents such as anthrax, the effects are due to the route of exposure and cause different syndromes despite the same pathogen i.e. inhalational, intestinal and cutaneous anthrax.

2. The disease process for biological agents and in particular live agents is more complex than that for chemical agents. The process can be differentiated into six different possible phases from exposure through to death. Although it is possible to go through all phases consecutively, some diseases may omit some of the phases. Understanding these phases will support medical planning and response with the use of MedCM optimised for each phase. Early and effective post-exposure prophylaxis or treatment will prevent progression of a disease to phase 3-4, or to phase 5-6 respectively.

27.2. PHASES OF BIOLOGICAL ILLNESS

The phases of biological illness including initial exposure through to death (or recovery) are:

a. *Phase 1 – Exposure*. This initial phase of the disease is the exposure of the host to the pathological agent following a deliberate, accidental or natural event. The route of exposure may determine the manifest syndrome that will present later during the disease process. Recognition of this phase is by detection or direct observation of suspicious activity both supported by intelligence. Mitigation during this phase is by avoidance or physical protection such as respiratory protection. Pre-exposure MedCM may have already been taken but should not be relied upon for 100% protection.

b. *Phase 2 – Incubation (latency) period*. The pre-symptomatic incubation period is a form of latency period specific to live organisms. This period is required during which the host immune system is overcome by the agent which is able to reproduce itself in sufficient quantities to establish an infection. For toxins, there is no agent reproduction and the latency period is the time it takes for the agent to act at the target site. Recognition of this phase is by *pre-symptomatic screening* either by detection of the BA within the host or the host response to the agent. Mitigation during this phase for both live agents and toxins may be post-exposure prophylaxis depending on intelligence, threat analysis and detection capability, as well as MedCM availability, window of opportunity and efficacy. The type of post-exposure prophylaxis available depends on the type of BA and may be used in combination:

(1) Bacteria – vaccination and antibiotics.

(2) Viruses – vaccination and antivirals.

(3) Toxins – vaccination and anti-toxins.

c. *Phase 3 – Prodrome*. This early prodromal phase occurs as live agents and occasionally toxins stimulate an inflammatory reaction within the host causing a raise in temperature and non-specific symptoms such as arthralgia and malaise. For some organisms with low virulence, this may be the only clinical manifestation. During this phase, some contagious diseases are highly infectious without any other clinical features. While toxins do not reproduce, some such as ricin are highly antigenic and cause an inflammatory reaction including a temperature rise. Tissue damage and necrosis will also cause an inflammatory reaction but this is part of the disease process at the site of action and is therefore part of the next phase. Recognition of this phase is by the presence of a fever which is an objective sign of disease. Fever can be used for screening as well as part of a case definition for operational epidemiology (see Chapter 17). During this phase, clinical investigations can also be started and may provide a diagnosis in isolation or in conjunction with the more specific next phase (manifest syndrome). Mitigation during this phase is generally supportive treatment unless the agent is suspected. If suspected, treatment for bacterial infections are antibiotics, viruses are antiviral treatment where available and for toxins are specific antitoxins. Where there is a high index of suspicion, agent specific immunoglobulin may also be available for some live agent infections.

d. *Phase 4 – Biological (manifest) syndrome*. Once an infection is established, the agent will have a pathological effect either by direct action or by the production of toxins in the case of some live agents. The type of effect may be local to the site of infection or through the systemic circulation to the site of action. The systems involved are often affected initially in isolation and so specific syndromes can be described and are listed in the next section. Recognition during this phase is clinical diagnosis based upon the assessment of the casualty and clinical investigations. Mitigation during this phase is empirical treatment for the syndrome observed based upon intelligence, detection warnings and clinical investigations.

e. *Phase 5 – Sepsis*. As the disease progresses, a systemic inflammatory response syndrome may develop and in the presence of an infection this is called *sepsis*. Sepsis is not certain for all infections but is life threatening if unrecognised or treatment delayed. Further details on sepsis and the levels of severity are detailed later in this chapter (see 27.4) while the treatment of sepsis using the *sepsis pathway* is covered in Chapter 29.

f. *Phase 6 – Death (or recovery)*. The final phase and irreversible phase is usually due to cardiovascular collapse often with multiple organ failure (MOF) and an irreversible metabolic acidosis. In some cases such as neurotoxins, respiratory failure may be the cause of death. The mortality for a septic patient is significant and prevention of death requires rapid recognition and treatment. For a patient with severe sepsis and organ failure in three systems mortality is over 50%, and may be the criteria for expectant treatment in mass casualty incidents or epidemics.

Note: The early recognition and appropriate treatment has a significant impact on the mortality of sepsis and recovery. Some initial treatment regimens including antibiotics may not cover some BA of operational significance and highlight the importance of intelligence and early laboratory diagnosis.

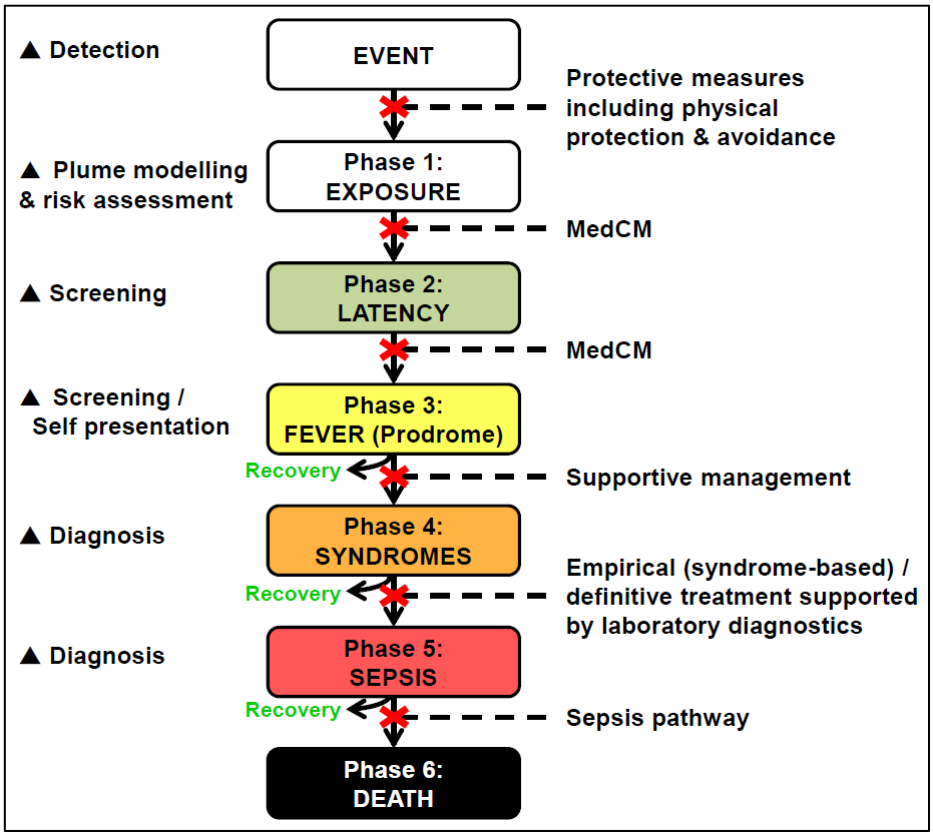

Figure 27-1: Phases of Infection, Recognition ▲ and Mitigation X.

27.3. BIOLOGICAL SYNDROMES

1. During phase 4, as described in the section above, may develop into a number of symptoms and signs that when grouped form a biological manifest syndrome. These syndromes supported by intelligence and clinical investigations will make a diagnosis. Correlation between the syndrome and clinical investigation will provide the certainty with which the diagnosis is made (suspect, probable and confirmed).

2. The phase 3 and 4 biological syndromes most associated with BA are:

   a. Fever including influenza-type symptoms (Phase 3 – Prodrome).

   b. Cutaneous.

      (1) Generalised.

      (2) Localised.

   c. Gastrointestinal.

      (1) Upper.

      (2) Lower without blood.

      (3) Lower with blood.

   d. Haemorrhagic.

e. Lymphadenopathy.

f. Neurological (Central).

g. Neurological (Peripheral / neurotoxin-like).

h. Respiratory.

3. Table 27-1 provides a summary of the biological syndromes, features and potential pathogens and includes examples of biological agents of operational significance both used for BW and naturally occurring.

4. Initial choice of treatment including antibiotics and antiviral drugs will often depend on the manifest syndrome. The choice of empirical treatment for a respiratory syndrome (e.g. pneumonia) will differ to a central neurological syndrome (e.g. cerebral irritation / encephalitis).

Note: Where the syndrome and the clinical investigation are inconsistent with the provisional diagnosis, the clinician should reassess the information and diagnosis while also considering alternative diagnoses including non-endemic disease, emerging diseases and atypical presentations (e.g. unnatural aetiology and deliberate release) and modify treatment e.g. the initial choice for a respiratory infection is unlikely to include ciprofloxacin for anthrax.

27.4. SEPSIS

The 2016 guidelines for sepsis defines sepsis as *life-threatening organ dysfunction resulting resulting from a dysregulated host response to infection*.[1] Diagnosis requires two of the three criteria (sepsis-3) to be met. For military deployments, a fourth criterion has been recommended.[2] The four criteria are:

a. Blood pressure (BP) < 100 or lack of radial pulse (suggestive of septic shock).

b. Respiratory rate > 22 breaths per minute.

c. Altered mental status.

d. Non-blanching rash, decreased capillary refill or skin mottling.

27.4.1. OTHER FEATURES OF SEPSIS AND ORGAN DYSFUNCTION

Other features of severe sepsis are:

a. Lactate above upper normal limit (> 4 mmol/L).

b. Requirement for oxygen to maintain SpO_2 > 90%.[3]

c. Urine output < 0.5 ml/kg/hr for 2 hours despite adequate fluid resuscitation.

[1] ✍ *Surviving Sepsis 2016* (www.survivingsepsis.org) updated the definition of sepsis and identified 3 criteria for sepsis.
[2] ✍ The fourth criterion was agreed during the 2016 Bio-Medical Panel due to the types of BA and as a surrogate sign for thrombocytopenia or coagulopathy (it remains unvalidated).
[3] This is surrogate marker for arterial hypoxemia (PaO_2/FiO_2 < 250 in absence of pneumonia as infection course and <200 with pneumonia as infection course).

d. Coagulopathy (International Normalised Ratio (INR) > 1.5).

e. Platelets < 100 x 10^9/L (100,000µL).

f. Bilirubin > 2 mg/dL or 34.2 µmol/L.

g. Creatinine > 2 mg/dL or 176.8 µmol/L.

27.4.2. SEPTIC SHOCK

Sepsis-induced hypotension is defined as a systolic blood pressure (SBP) <90mmHg or mean arterial BP <70mmHg. Septic shock is defined as sepsis-induced hypotension persisting despite adequate fluid resuscitation (and/or lactate > 4mmol/L). Fluid resuscitation should commence within 10 minutes of recognition by medical personnel.

Table 27-1: Biological Syndromes, Features and Potential Biological Agents.

SYNDROME	SYMPTOMS AND SIGNS	POSSIBLE CONDITIONS
FEVER / INFLUENZA-LIKE (PRODROME)		
	Temperature > 38C or 100.5F. Sometimes associated with chills, myalgia, lethargy & rigors.	Any agent causing a prodrome. Brucellosis (usually self-limiting). Malaria‡. Influenza A‡. (Toxins are less likely to be associated with fever unless causing SIRS)
CUTANEOUS		
Generalised rash	Rashes can be defined as erythema, maculopapular, vesicular and pustular.	Smallpox.
Localised lesion	Localised oedema and/or cutaneous lesion (ulcer, abscess necrosis or eschar).	Cutaneous anthrax. Melioidosis. Rickettsiosis. Tularaemia. Leishmaniasis‡. Local radiation injury*.
GASTROINTESTINAL		
Upper	Nausea, vomiting, epigastric pain.	SEB (ingestion). Radiation prodrome*.
Lower without blood	Diarrhoea, ± mucus & includes rice water stool.	Cholera. Norovirus‡.
Upper with blood	Diarrhoea with blood (dysentery).	Gastrointestinal anthrax.
HAEMORRHAGIC		
	Bleeding, purpuric rash, bruising, petechial rash.	Viral haemorrhagic fevers. Disseminated intravascular coagulopathy (DIC) causing pathogens e.g. plague septicaemia.
LYMPHADENOPATHY		
	Enlarged and painful lymph nodes including groin and axilla.	Bubonic plague. Tularaemia. HIV seroconversion‡.
NEUROLOGICAL (CENTRAL)		
Encephalitis	Disorders of mental status, confusion, visual hallucinations, disorders of consciousness.	Japanese encephalitis, Venezuelan equine encephalitis.
Meningitis	Neck pain, photophobia.	Anthrax related haemorrhagic meningitis (cardinal cap–sign – post-mortem) Meningococcal meningitis‡.
NEUROLOGICAL (PERIPHERAL)		
	Paralysis of cranial nerves, ptosis, blurred vision, double vision (diplopia), dysphasia, dysphagia, descending paralysis.	Botulinum toxin. Post-synaptic neurotoxic venoms.
RESPIRATORY		
	Cough, dyspnoea, pleuritic chest pain, haemoptysis.	Pneumonic plague, inhalational anthrax. Inhaled ricin or SEB. Viral pneumonitis.

* non-biological syndrome, ‡ examples of other endemic diseases

CHAPTER 28: SIGNIFICANT BIOLOGICAL AGENTS

28.1. INTRODUCTION

1. Each BA has a different profile with an impact on the suite of protective measures required. Table 28-1 gives a summary of these properties that will support medical and command decision making based on the type of hazard (lethality, transmissibility) and risk using CDC classification.

2. The properties of these agents can be quantified as follows:

   a. *Agent types*: Bacteria, Virus, (Fungus), Toxin

   b. *Lethality*: Low (< 5%), Significant (5-49%), High (50-90%), Very high (>90%).[1]

   c. *Infectivity*: Low, Moderate, High.

   d. *Transmissibility* (contagious): Yes, No.

   e. Centers for Disease Prevention and Control (CDC) classification (see Table). This is included for rapid reference and high light agents for particular significance.

3. Individual information sheets for specific BA of operational significance or interest are provided at the end of this chapter. A key to the icons used is included in Table 28-2 below.

Table 28-1: Centers for Disease Control (CDC) Classification.

CLASS A	CLASS B	CLASS C
Properties include: High lethality High infectivity Person-to-person spread	Properties include: Moderate lethality Moderate infectivity Public health interest Possible person-to-person spread	Properties include: Emerging threat Potential for deliberate release

Table 28-2: Key for Biological Agent Factsheets.

	Standard (universal) precautions recommended include: **apron** (or IPE ensemble, including overboots), **gloves** and, **surgical mask**, if droplet hazard.		Isolation, Quarantine and Operational RoM may be required.
	High specification respiratory protection recommended including eye protection. (EN:FFP2/3, N95, military respirator with particulate filter)		Considered as Public Health Emergency of International Concern (PHEIC) and Strategic RoM may be mandated.
			

[1] Case fatality rate based on untreated casualties.

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ANNEX 28A – BIOLOGICAL AGENT FACTSHEETS

Table–28.3 – Main Characteristics of Some of the Biological Agents of Operational Significance.

Agent	BA Type	Condition	Lethality	Infectivity	Trans	CDC risk
Alphavirus	Virus	Chikungunya Eastern equine encephalitis Venezuelan equine encephalitis Western equine encephalitis	Low	Variable	N	B
Arenavirus ⓘ	Virus	Lassa fever	High	High	Y	A
Bacillus anthracis ⓘ	Bacteria	Inhalational anthrax	Very High	Moderate	N*	A
		Cutaneous anthrax	Significant	Moderate		
		(Gastro)Intestinal anthrax	High	Moderate		
		Injectional anthrax	High	Moderate		
Brucella sp. ⓘ	Bacteria	Brucellosis	Low	High	N	B
Bunyavirus ⓘ	Virus	Crimean-Congo haemorrhagic fever	High	High	Y	A
Burkholderia mallei ⓘ	Bacteria	Glanders	High	High	N	B
Burkholderia pseudomallei ⓘ	Bacteria	Melioidosis	Variable	High	N	B
Clostridium botulinum toxin ⓘ	Toxin	Botulism	High	-	N	A
Clostridium perfringens	Bacteria	Gas gangrene	High	Low	N	
Clostridium perfringens toxin ⓘ	Toxin	Clostridium perfringens toxin	Low	-	N	B
Coxiella burnetii ⓘ	Bacteria	Q fever	Low	High	N	B
Filovirus ⓘ	Virus	Ebola haemorrhagic fever Marburg haemorrhagic fever	High	High	Y	A
Flavivirus	Virus	Japanese encephalitis	Low	High	N	B
Francisella tularensis ⓘ	Bacteria	Oculoglandular tularaemia Pneumonic tularaemia Typhoidal tularaemia Ulceroglandular tularaemia	High	High	N	A
Influenza virus	Virus	Flu	Low	High	Y	
Norovirus	Virus	Winter-vomiting	Low	Moderate	Y	
Ricin toxin ⓘ	Toxin	-	High	-	N	B
Salmonella typhi	Bacteria	Typhoid fever	Significant	Moderate	Y	B
Shigella sp.	Bacteria	Shigellosis	Low	Moderate	Y	B
Staphylococcus enterotoxin B ⓘ	Toxin	-	Variable	-	N	B
Trichothecene (T-2) mycotoxins ⓘ	Toxin	-	High	-	N	B
Vibrio cholera ⓘ	Bacteria	Cholera	Significant	Moderate	Y	B
Variola sp. ⓘ	Virus	Smallpox	Very high	High	Y	A
Yersinia pestis ⓘ	Bacteria	Bubonic plague Septicaemic plague Pneumonic plague	Very high	High	Y	A

* While anthrax is not contagious, care should be taken with the handling of dried blood spills, waste materials and fatalities due to potential spore formation.

ⓘ Factsheet is available

ANTHRAX

CDC **A**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
NO	MODERATE	1-6 days (may be prolonged)	3-5 days	VERY HIGH

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

STANDARD PRECAUTIONS*
WARNING: High level of protection required when handling bodies

Anthrax is a zoonotic disease caused by *Bacillus anthracis.* The organism forms spores readily in the environment and the spore is the infectious form. Under natural conditions, humans become infected by contact with infected animals or contaminated animal products. The human form is usually manifested by cutaneous lesions, or in the case of consumption of contaminated material as gastrointestinal anthrax. A BW attack with aerosolised spores would primarily cause inhalational anthrax, a rare form of the naturally occurring disease. A new variation of presentation has been seen in intravenous drug users with infected injection sites ('injection' anthrax). Many of the effects of anthrax are mediated through its toxin, which consists of 3 components: the Protective Antigen (PA), Lethal Factor (LF) and Oedema factor (EF).

* While anthrax is not contagious, care should be taken with the handling of dried blood spills, waste materials and fatalities due to potential spore formation.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Respiratory (inhalational)	Cutaneous / 'Injection' (localised lesion)	Gastrointestinal

Clinical features. Inhalational anthrax begins after an incubation period that may be up to 60 days and is believed to be dependent on dose and susceptibility. The onset is gradual and non-specific with fever and malaise, nausea, vomiting and abdominal pain sometimes in association with a non-productive cough and mild chest discomfort. In some cases, there may be a short period of improvement. The initial symptoms are followed in 2-3 days by the abrupt development of severe respiratory distress with dyspnoea, diaphoresis, stridor and cyanosis. Physical findings may include evidence of pleural effusions, oedema of the chest wall and haemorrhagic meningitis (pathological finding of 'cardinal cap'). Thoracic radiography commonly reveals a widened mediastinum with pleural effusions, further cases being diagnosed by thoracic CT. Septic shock and death usually follow within 24-36 hours of the onset of respiratory distress.

MEDICAL MANAGEMENT

Investigations	**Radiology**: Chest radiography – widened mediastinum (not all cases) **Labs**: Blood cultures, immunoassays and PCR
MedCM	**Active immunisation (pre & post exposure)**: Consider anthrax vaccination **Antibiotics (pre & post exposure)**: *Ciprofloxacin* 500mg (oral) every 12 hours (1st line) OR *doxycycline* 100mg (oral) every 12 hours (2nd line)
Treatment regimen(s)	**Immediate severe sepsis management** **Antibiotics (multidrug-therapy regime)**: *Ciprofloxacin* 400mg (intravenous) every 12 hours plus one or two of the following: *penicillin, rifampicin, vancomycin, imipenem, clindamycin* or *clarithromycin*. Alter according to sensitivities and convert to oral. Continue for 60 days, switching to oral as tolerated. **Passive immunisation**: Consider immunoglobulin, if available and appropriate.

BOTULINUM TOXIN

CDC **A**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	TOXICITY (LD_{50})	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
NO	0.8µg	Hours to days	Days to months	HIGH

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

STANDARD PRECAUTIONS

Botulism is caused by several forms of neurotoxins produced by the bacillus *Clostridium botulinum*. The toxins block acetylcholine neurotransmission by binding to the pre-synaptic membrane of neurons and preventing acetylcholine release. The blockade is most evident clinically in the parasympathetic autonomic nervous system and at the neuromuscular junction. A BW attack with botulinum toxin delivered by aerosol or as an inoculation would be expected to cause symptoms similar to those observed with food-borne botulism.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Peripheral neurological

Clinical features. Rapid descending flaccid paralysis with initial signs and symptoms including ptosis, generalised weakness, lassitude, and dizziness. Motor symptoms usually are present early in the disease; cranial nerves are affected first with blurred vision, dilated pupils, diplopia, ptosis and photophobia. Bulbar nerve dysfunction causes dysarthria, dysphonia and dysphagia. This is followed by a symmetrical, descending, progressive weakness of the extremities along with weakness of the respiratory muscles. Development of respiratory failure may be abrupt. On physical examination, the patient is alert, oriented and afebrile. Diminished salivation with extreme dryness of the mouth and throat may cause complaints of a sore throat. Urinary retention or ileus may also occur. Postural hypotension may be present. Mucous membranes of the mouth may be dry and crusted. Neurological examination shows flaccid muscle weakness of the palate, tongue, larynx, respiratory muscles and extremities. Deep tendon reflexes vary from intact to absent. No pathologic reflexes are present and the sensory examination generally is normal, although reports suggest that obtundation or sensory involvement may sometimes occur. Differential diagnosis includes Guillain-Barre syndrome (Miller Fisher variant), myasthenia gravis and tick paralysis.

MEDICAL MANAGEMENT

Investigations	**Toxicology**: Mouse inoculation studies, immunoassays.
MedCM	**Anti-toxin**: Polyvalent anti-toxin (pre and post exposure considerations)
Treatment regimen(s)	**Anti-toxin**: Early administration is essential to allow neutralisation of circulating toxin. Where the differential diagnosis includes botulism, anti-toxin is advised. **Supportive therapy**: Supportive therapy including ventilation may be the only therapy for delayed presentations.
Comments	**Note:** Ensure resuscitation facilities are available during anti-toxin administration due to the risk of hypersensitivity reactions including anaphylaxis.

BRUCELLOSIS

CDC **B**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
NO	HIGH	Days to months	Weeks to years	LOW

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

STANDARD PRECAUTIONS

Brucellosis is a systemic zoonotic disease caused by a number of *Brucella sp*. Human disease virulence varies between species. Their natural reservoir is domestic animals, such as goats, sheep, and camels (*B. melitensis),* cattle (*B. abortus)* and pigs (*B. suis). B. canis* is primarily a pathogen of dogs, and only occasionally causes disease in humans. Humans are infected when they inhale contaminated aerosols, ingest raw (unpasteurised) infected milk or meat, or have abraded skin or conjunctival surfaces that come into contact with the bacteria. The bacteria are small gram-negative, aerobic, non-motile coccobacilli that grow within monocytes and macrophages. They reside quiescently in tissue and bone marrow, and are extremely difficult to eradicate even with antibiotic therapy. Laboratory infections are quite common, but there appears to be no human-to-human transmission. *Brucella sp.* have been considered potential candidates for use in BW due to a relatively low ID_{50}. Under selected environmental conditions (darkness, cool temperatures, high CO_2), persistence of up to 2 years has been documented. When used as a BW agent, *Brucellae* would most likely be delivered by the aerosol route and the resulting infection would be expected to mimic natural disease.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Fever	Respiratory (low incidence)

Clinical features. Brucellosis presents after a wide varying incubation period. Clinical disease presents typically as an acute, non-specific febrile illness with chills, sweats, headache, fatigue, myalgias, arthralgias, and anorexia. Cough occurs in 15-25%, but the chest radiograph is usually normal. Complications include sacroiliitis, arthritis, vertebral osteomyelitis, epididymo-orchitis and rarely endocarditis. Physical findings include lymphadenopathy in 10-20% and splenomegaly in 20-30% of cases. Untreated disease can persist for months to years, often with relapses and remissions. Disability may be pronounced. Lethality may approach 6% following infection with *B. melitensis*, but the disease is rarely fatal (0.5% or less) after infection with other serotypes (usually after complications such as endocarditis develops).

MEDICAL MANAGEMENT

Investigations	**Labs**: Blood (& bone marrow) cultures (limited), serology and PCR
MedCM	**Antibiotics**: *Doxycycline* 100 mg (oral) every 12 hours plus *Rifampicin* 900 mg (oral) once daily for 21 day (low risk) or 6 weeks (high risk)
Treatment regimen(s)	**Antibiotics**: *Doxycycline* 100mg (oral or IV) every 12 hours plus either: *rifampicin* 900mg (oral) once daily, *streptomycin* 1G (IM) daily (max. 3 weeks) OR *gentamicin* 5mg/kg/day (IM/IV).
Comments	**Caution**: Relapses do occur even with patient compliance.

CHOLERA
(*Vibrio cholera*)

CDC **B**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
FAECAL-ORAL	MODERATE	1-5 days	1-2 weeks	SIGNIFICANT

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

CONTACT PRECAUTIONS

Cholera is a diarrhoeal disease caused by *Vibrio cholera,* a short, curved, gram-negative bacillus. Humans acquire the disease by consuming water or food contaminated with the organism. The organism multiplies in the small intestine and secretes an enterotoxin that causes secretory diarrhoea. Cholera is often associated with poor sanitation and insecure water supply and remains a biological agent of operational concern. If employed as a BW, *V. cholera* will most likely be used to contaminate water or food supplies.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Gastrointestinal

Clinical features. Cholera may present as mild diarrhoea or as a fulminant disease characterised by profuse watery diarrhoea (*rice water stool*) with fluid losses exceeding 5 to 10 litres or more per day. Without treatment, death may result from severe dehydration, hypovolaemia and shock in 50-60% of cases. Vomiting is often present early in the illness and may complicate oral replacement of fluid losses. There is little or no fever or abdominal pain. Adequate treatment will bring the fatality rate down to <1%.

Differential diagnosis. Watery diarrhoea can also be caused by food- and water-borne pathogens (enterotoxigenic *E coli,* shigella, rotavirus or other viruses, other *vibrio* species), or food poisoning due to ingestion of pre-formed toxins such as those of *Clostridium perfringens, Bacillus cereus,* or *Staphylococcus aureus.*

MEDICAL MANAGEMENT

Investigations	**Labs**: Stool dark-field or phase-contrast microscopy. Stool culture. PCR. Cholera toxin tests are available.
MedCM	**Active immunisation**: Vaccination (limited efficacy)
Treatment regimen(s)	**Supportive**: Fluid and electrolytic replacement of fluid and electrolyte losses. World Health Organisation solution (3.5g NaCl, 2.5g NaHCO3, 1.5g KCl and 20g glucose per litre). Intravenous fluid replacement is occasionally needed when vomiting is severe, when the volume of stool output exceeds 7 litres/day, or when severe dehydration with shock has developed. **Antibiotics**: There will shorten the duration of diarrhoea and reduce fluid losses although not mandatory. *Ciprofloxacin* is highly effective (500 mg every 12 hours).

CLOSTRIDIUM PERFRINGENS TOXIN

CDC **B**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	TOXICITY (LD_{50})	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
NO	0.1-5µg/kg	1-5 days	24 hours	SIGNIFICANT

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

STANDARD PRECAUTIONS

Clostridium perfringens is a common anaerobic bacterium associated with 3 distinct syndromes: gas gangrene (clostridial myonecrosis), enteritis necroticans and clostridium food poisoning. Each of these syndromes has very specific requirements for delivering inocula of *Cl. perfringens* to specific sites to induce disease, and it is difficult to imagine a general scenario in which the spores or vegetative organisms could be used as a BW agent. However, there are at least 12 protein toxins identified, and one or more of these could be produced and weaponised. Waterborne disease is possible but unlikely. The alpha toxin would be lethal by aerosol. This is a well-characterised, highly toxic phospholipase-C. Other toxins from the organism might be co-weaponised and enhance effectiveness. For example, the epsilon toxin exhibits neurotoxicity in laboratory animals.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Respiratory	Non-specific organ failure	Gastrointestinal	"Gas gangrene"

Clinical features. The clinical picture of aerosolised *Cl. perfringens* alpha toxin would be expected to be that of a serious acute pulmonary insult. Absorbed alpha toxin could produce vascular leak, haemolysis, thrombocytopaenia, and liver damage. Other toxins admixed could modify the illness. There is insufficient information available to speculate on a clinical syndrome produced by other *Cl. Perfringens* toxins.

MEDICAL MANAGEMENT

Investigations	**Radiology***: Tissue gas may suggest bacteria component to toxin production.* **Lab***:* Blood cultures if natural (live bacterial) disease. Immunoassays (limited).
MedCM	Nil
Treatment regimen(s)	Surgical: Surgical debridement if concurrent infection. Antibiotics: *Penicillin and clindamycin* for wound contamination with live *Cl. perfringens*.

GLANDERS
(*Burkholderia mallei*)

CDC **B**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
LOW	HIGH	10-14 days	Weeks	HIGH

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

STANDARD PRECAUTIONS

Glanders is caused by *Burkholderia mallei*, a gram-negative bacillus. *B. mallei* is primarily noted for producing disease in horses, mules, and donkeys. The disease is not widespread with human disease being very rare despite frequent and often close contact with infected animals. Human cases occur primarily among veterinarians, horse and donkey caretakers, abattoir workers and laboratory personnel. Infection occurs by the organism invading the nasal, oral and conjunctival mucous membranes, inhalation into the lungs and invading abraded or lacerated skin. In a BW attack, the primary threat would most likely be an aerosol release.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Respiratory (inhalational)	Cutaneous (localised lesions)	Septicaemia

Clinical features. Glanders may occur in an acute localised form, acute pulmonary infection or an acute fulminant sepsis (rapidly fatal). The incubation period ranges from 10 – 14 days, depending on the inhaled dose and virulence. The septicaemic form begins suddenly with fever, rigors, sweats, myalgia, pleuritic chest pain, granulomatous or necrotising skin lesions, generalised erythroderma, jaundice, photophobia, lacrimation and diarrhoea.

Physical examination may reveal fever, cervical adenopathy and mild hepatomegaly or splenomegaly.

Chest radiographs may show pulmonary nodules (0.5-1.0 cm) and/or a bilateral bronchopneumonia, segmental or lobar pneumonia with consolidation and / or cavitating lung lesions.

MEDICAL MANAGEMENT

Investigations	**Labs**: Gram staining, blood culture (late finding), complement fixation, PCR
MedCM	Consider trimethoprim / sulfamethoxazole (8mg & 40mg/kg/day respectively)
Treatment regimen(s)	**Immediate sepsis resuscitation** **Surgical**: Consider surgical drainage of abscesses **Antibiotics (mild/localised)**: *Augmentin* (60mg/kg/day); *Tetracycline* (40mg/kg/day); *Trimethoprim-sulfamethoxazole* (4mg/kg/day-20mg/kg/day respectively); for mild cases consider combination of 2 regimes for 30 days and *ciprofloxacin* 400mg (IV) every 12 hours or 500mg (oral) every 12 hours **Antibiotics (severe)**: *Imipenem* 1g; *Gentamycin 5mg/kg initial dose; Ceftazidime* (120mg/kg/day) combined with *Trimethoprim-sulfamethoxazole* (8mg/kg/day-40mg/kg/day respectively) for 2 weeks converting to oral regime. Continue oral regime for 6 months

MELIOIDOSIS
(*Burkholderia pseudomallei*)

CDC **B**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
NO	HIGH	1 to >6 day	3-5 days	VARIABLE

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

STANDARD PRECAUTIONS

Melioidosis is an infectious disease of humans and animals caused by *Burkholderia pseudomallei,* a gram-negative bacillus. It is especially prevalent in Southeast Asia but has been described from many countries around the world. The disease has a variable and inconstant clinical spectrum. A BW attack with this organism would most likely be by the aerosol route.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Respiratory (inhalational)	Lymphadenitis (bubonic)	Septicaemia, including DIC

Clinical features. Infection by inoculation results in a subcutaneous nodule with acute lymphangitis and regional lymphadenitis, generally with fever. Pneumonia may occur after inhalation or haematogenous dissemination of infection. It may vary in intensity from mild to fulminant, usually involves the upper lobes, and often results in cavitation. Pleural effusions are uncommon. An acute fulminant septicaemia may occur characterised by rapid appearance of hypotension and shock. A chronic suppurative form may involve virtually any organ in the body.

MEDICAL MANAGEMENT

Investigations	**Labs**: Gram staining, blood culture (late finding), complement fixation, PCR
MedCM	Consider trimethoprim / sulfamethoxazole (8mg & 40mg/kg/day respectively)
Treatment regimen(s)	**Immediate sepsis resuscitation** **Surgical**: Consider surgical drainage of abscesses **Antibiotics (mild/localised)**: *Augmentin* (60mg/kg/day); *Tetracycline* (40mg/kg/day); *Trimethoprim-sulfamethoxazole* (4mg/kg/day-20mg/kg/day respectively); for mild cases consider combination of 2 regimes for 30 days. **Antibiotics (severe)**: *Imipenem* or *Meropenem* 1g; *Ceftazidime* (120mg/kg/day) combined with *Trimethoprim-sulfamethoxazole* (8mg/kg/day-40mg/kg/day respectively) for 2 weeks converting to oral regime. Continue oral regime for 6 months. Although gentamicin is not recommended, it is likely to be given as part of the management of septic shock if present.

PLAGUE
(*Yersinia pestis*)

CDC **A**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
AEROSOL (Pneumonic)	HIGH	2-3 days (inhaled) 2-10 days (bubonic)	1-2 days	VERY HIGH

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

AIRBORNE PRECAUTIONS

Plague is a zoonotic disease caused by *Yersinia pestis.* Under natural conditions, humans become infected as a result of contact with rodents and their fleas. The transmission of the gram-negative coccobacillus is by the bite of an infected flea. Under natural conditions, 3 syndromes are recognised: bubonic, septicaemic and pneumonic. A proportion of cases of bubonic plague develop pneumonia, which may then spread by droplets to cause primary pneumonic plague. In a BW scenario, the plague bacillus could be delivered via infected vectors (fleas) causing the bubonic type or via aerosol causing the pneumonic type, which is highly contagious (transmissible).

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Respiratory (inhalational)	Lymphadenitis (bubonic)	Septicaemia, including DIC

Clinical features. *Bubonic plague* – The onset is acute and often fulminant with malaise, high fever and tender lymph nodes (buboes). Inguinal lymphadenitis predominates, but cervical and axillary lymph nodes can also be involved. The nodes infected are tender, fluctuant and necrotic.

Septicaemic plague – Bubonic plague may progress spontaneously to the septicaemic form with organisms spread to the central nervous system, lungs (producing pneumonic disease) and elsewhere. The mortality is 50% in untreated patients with the terminal event being circulatory collapse, haemorrhage, and peripheral thrombosis.

Pneumonic plague – For primary (inhaled) pneumonic plague, the incubation period is more rapid (2 to 3 days). The onset is acute and fulminant with malaise, high fever, chills, headache, myalgia, and cough with production of a bloody sputum. The pneumonia progresses rapidly. In untreated patients, the mortality is 100% with death due to respiratory failure, circulatory collapse or bleeding.

MEDICAL MANAGEMENT

Investigations	**Labs**: Blood & lymph node aspirate, microscopy (safety pin sign), serology and PCR
MedCM	**Antibiotics (pre & post exposure)**: Oral doxycycline or ciprofloxacin as below
Treatment regimen(s)	**Immediate sepsis resuscitation** **Antibiotics (severe)**: *Gentamicin* 5mg/kg (IV) once daily, *streptomycin* 1G (IM) every 12 hours or *ciprofloxacin* 400mg (IV) every 12 hours. Add *chloramphenicol* if meningitis. Continue for 10 days, switching to oral as tolerated. **Antibiotics (mild)**: *Ciprofloxacin* 500mg (oral) or *doxycycline* 100mg (oral) every 12 hours.

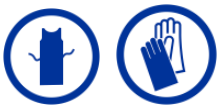
Q FEVER
(*Coxiella burnetii*)

CDC **B**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
RARE	HIGH	10-20 days	2 days to 2 weeks	LOW

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT



STANDARD PRECAUTIONS

Q fever is a zoonotic disease caused by *Coxiella burnetii.* The gram-negative obligate intracellular organism survives for long periods in the environment. The most common animal reservoirs are sheep, cattle and goats, and it is particularly concentrated in parturition fluids. Humans acquire the disease by inhalation of particles contaminated with the organisms. A BW attack would cause disease similar to that occurring naturally and has a very low ID_{50} < 10.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Fever	Respiratory

Clinical features. Following an incubation period of 10-20 days, Q fever generally occurs as a self-limiting febrile illness lasting 2 days to 2 weeks. Pneumonia occurs frequently, usually manifested only by an abnormal thoracic radiograph. A non-productive cough and pleuritic thoracic pain occur in about 25% of patients with Q fever pneumonia. Complications include chronic fatigue, chronic hepatitis, endocarditis, aseptic meningitis, encephalitis, and osteomyelitis.

MEDICAL MANAGEMENT

Investigations	**Radiology**: Chest x-ray (abnormal in 50% of cases) **Labs**: Serology
MedCM	**Active immunisation (pre-exposure)**: Vaccination, where available **Antibiotics (post exposure)**: *Doxycycline* 100mg (oral) every 12 hours or *erythromycin* 500mg (oral) every 6 hours for 5 days. **Note**. Antibiotics are to be started at least 8 days after exposure.
Treatment regimen(s)	**Antibiotics**: *Doxycycline* 100mg (oral/IV) every 12 hours, *erythromycin* 500mg every 6 hours (oral/IV) or *ciprofloxacin* 400mg (IV) every 12 hours. Monitor and treat any long term sequelae.

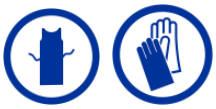
RICIN

CDC **B**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	TOXICITY (LD_{50})	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
NO	0.34mg	Hours	Days	HIGH

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT



STANDARD PRECAUTIONS

Ricin is a glycoprotein toxin (66,000 daltons) from the seed of the castor plant. The toxin blocks protein synthesis by altering the ribosomal mRNA processing, thus killing the cell. Ricin's significance as a potential BW agent relates to its availability world-wide, its ease of production, and extreme pulmonary toxicity when inhaled.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Fever	Respiratory or localised (depending on route of exposure)

Clinical features. The clinical picture seen depends on the route of exposure. All reported serious or fatal cases of castor bean ingestion have taken a similar course: rapid onset of nausea, vomiting, abdominal cramps and severe diarrhoea with vascular collapse; death has occurred on the third day or later. Following inhalation, one might expect non-specific symptoms of weakness, fever, cough, and hypothermia followed by hypotension and cardiovascular collapse. In monkeys, inhalation toxicity is characterised by a dose dependent pre-clinical period of 24-36 hours followed by anorexia and a progressive decrease in physical activity. Death occurs 36-48 hours post challenge. In mice, histopathological change is characterised by necrotising, suppurative airway lesions: rhinitis, laryngitis, tracheitis, bronchitis, bronchiolitis, and interstitial pneumonia with perivascular and alveolar oedema. Histopathological change in the airways is seen as early as 3 hours post challenge. The exact cause of death is unknown and probably varies with route of intoxication. High doses by inhalation appear to produce sufficient pulmonary damage to cause death.

MEDICAL MANAGEMENT

Investigations	**Radiology**: Chest x-ray suggestive of pulmonary agent. **Labs**: ELISA. Immunohistology. Serology.
MedCM	Nil
Treatment regimen(s)	**Anti-toxin**: Under development. **Supportive therapy**: Organ support and intensive care.

SMALLPOX

CDC **A**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
AIRBORNE	HIGH	8-18 days	3 weeks	HIGH

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

AIRBORNE PRECAUTIONS (and destroy contaminated items such as clothing)

Smallpox virus, an orthopoxvirus specific to humans, was an important cause of morbidity and mortality in the developing world until recent times. Eradication of the natural disease was completed in 1977 and the last human cases (laboratory infections) occurred in 1983; the world was declared free of smallpox virus in 1979. The virus officially exists today in only 2 laboratory repositories in the US and Russia. Appearance of human cases outside these laboratories would signal use of the virus as a BW weapon. Under natural conditions, the virus is transmitted by direct (face-to-face) contact with an infected case, by fomites, and by aerosols. Smallpox virus is highly stable and retains infectivity for long periods outside of the host. A related zoonotic virus, monkeypox, clinically resembles smallpox and causes sporadic human disease in West and Central Africa.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Respiratory (inhalational)	Cutaneous (localised lesion)

Clinical features. The illness begins with a prodrome lasting 2-3 days, with generalised malaise, fever, rigors, headache, and backache. This is followed by the appearance of a typical skin eruption characterised by progression from macules to papules to vesicles to pustules. The latter finally form crusts and, upon healing, leave depressed depigmented scars. The disease is infectious from the appearance of the fever until the last scab has healed. The distribution of lesions is centrifugal (more numerous on face and extremities than on the trunk). Lesions are in the same stage of development at any point in time. Fever may reappear around the 7th day after onset of rash. The case fatality rate is approximately 35% in unvaccinated individuals. A subset of patients develop a haemorrhagic diathesis with disseminated intravascular coagulopathy and have a poor prognosis. Other complications include arthritis, pneumonia, bacterial superinfection of skin lesions, osteomyelitis and keratitis. Fully immune individuals exposed to the virus by the respiratory route may develop fever, sore throat, and conjunctivitis ("contact fever") lasting several days. These individuals may be seriously ill but have a low mortality. However, even mild cases excrete fully virulent virus, which can lead to secondary spread of full-blown smallpox in susceptible individuals.

MEDICAL MANAGEMENT

Investigations	**Labs**: Skin samples (incl. vesicular fluid, pus or scabs) by direct electron microscopy. Agar gel immunoprecipitation or immunofluorescence, serology and PCR.
MedCM	**Active immunisation (pre & post exposure)**: Smallpox (vaccinia) vaccination.
Treatment regimen(s)	**Anti-viral**: ST-246 available to some Nations.

STAPHYLOCOCCAL ENTEROTOXIN B (SEB)

CDC **B**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	TOXICITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
NO	**0.026 µg (ED_{50})** **1.66 µg (LD_{50})**	**Hours**	**Hours to days**	**VARIABLE** depends on route

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

STANDARD PRECAUTIONS

Staphylococcal Enterotoxin B (SEB) is one of several exotoxins (superantigen) produced by *Staphylococcus aureus,* causing food poisoning when ingested. A BW attack with aerosol delivery of SEB to the respiratory tract produces a distinct syndrome causing significant morbidity and potential mortality.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Respiratory (inhalational)	Gastrointestinal (ingestion)

Clinical features. The disease begins 1-6 hours after ingestion with the sudden onset of fever, chills, headache, myalgia, and non-productive cough. In more severe cases, dyspnoea and retrosternal thoracic pain may also be present. Fever, which may reach 40°C, has lasted 2-5 days, but cough may persist 1-4 weeks. In many patients nausea, vomiting, and diarrhoea will also occur. Physical findings are often unremarkable. Conjunctival injection may be present, and in the most severe cases, signs of pulmonary oedema would be expected. The thoracic radiograph is generally normal, but in severe cases, there will be increased interstitial markings, atelectasis, and possibly overt pulmonary oedema. In moderately severe laboratory exposures, lost duty time has been <2 weeks, but, based upon animal data, it is anticipated that severe exposures will result in fatalities.

MEDICAL MANAGEMENT

Investigations	**Labs**: Serology, immunoassay.
MedCM	Nil
Treatment regimen(s)	**Supportive only**

TRICHOTHECENE (T-2) MYCOTOXINS

CDC **B**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	TOXICITY (LD_{50})	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
NO	28mg	Hours	Hour	HIGH

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

STANDARD PRECAUTIONS

The trichothecene mycotoxins are a diverse group of more than 40 compounds produced by fungi. They are potent inhibitors of protein synthesis, impair DNA synthesis, alter cell membrane structure and function, and inhibit mitochondrial respiration. Secondary metabolites of fungi, such as T-2 toxin, produce toxic reactions called mycotoxicoses upon inhalation or consumption of contaminated food products by humans or animals. Naturally occurring trichothecenes have been identified in agricultural products and have been implicated in animal disease. There limited information on human exposure to trichothecenes. However, strong circumstantial evidence has associated these toxins with alimentary toxic aleukia (ATA), the fatal epidemic seen in Russia during World War II and alleged BW incidents ('yellow' rain') in Cambodia, Laos and Afghanistan.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Respiratory	Cutaneous	Haemorrhagic	Gastrointestinal

Clinical features. Consumption of these mycotoxins results in weight loss, vomiting, skin inflammation, mucosal membrane irritation, bloody diarrhoea, diffuse haemorrhage, and possibly death. The onset of illness following acute exposure to T-2 (IV or inhalation) occurs in hours, resulting in the rapid onset of circulatory shock characterised by reduced cardiac output, arterial hypotension, lactic acidosis and death within 12 hours. Clinical signs and symptoms of ATA were haemorrhage, leucopenia, ulcerative pharyngitis, and depletion of bone marrow. The purported use of T-2 as a BW agent resulted in an acute exposure via inhalation and/or dermal routes, as well as oral exposure upon consumption of contaminated food products and water. Alleged victims reported painful skin lesions, light-headedness, dyspnoea, and a rapid onset of haemorrhage, incapacitation and death. Survivors developed a radiation-like sickness including fever, nausea, vomiting, diarrhoea, leucopenia, bleeding and sepsis.

MEDICAL MANAGEMENT

Investigations	**Labs**: Immunoassay
MedCM	Nil
Treatment regimen(s)	Supportive and replacement therapy. Consider cytokine therapy for bone marrow suppression.

TULAREMIA
(*Francisella tularensis*)

CDC **A**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
NO	HIGH	2-10 days	2 weeks	HIGH

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT

STANDARD PRECAUTIONS

Tularaemia is a zoonotic disease caused by *Francisella tularensis*, a gram-negative bacillus. Humans acquire the disease under natural conditions through inoculation of skin or mucous membranes with blood or tissue fluids of infected animals, or bites of infected deerflies, mosquitoes, or ticks. Less commonly, inhalation of contaminated dusts or ingestion of contaminated foods or water may produce clinical disease. A BW attack with *F. tularensis* delivered by aerosol would primarily cause septicaemic (typhoidal) or pneumonic symptoms of tularaemia, with a mortality of 30% untreated (natural mortality < 5-10%). Many exposed individuals would develop pneumonic tularaemia (primary or secondary), but clinical pneumonia may be absent or non-evident. Tularaemia therefore has a variable fatality rate depending on route of exposure and delay in treatment.

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Respiratory (pneumonic)	Cutaneous (ulceroglandular)	Eye (oculoglandular)	Gastrointestinal	Septicaemia (typhoidal)

Clinical features. The clinical form of tularaemia seen depends upon the route of exposure and virulence of the strain. In humans, as few as 10-50 organisms will cause disease if inhaled or inoculated, or 10^8 organisms via the oral route. Under natural conditions, ulceroglandular tularaemia generally occurs about 3 days after inoculation and manifests as regional lymphadenopathy, fever, chills, headache, and malaise, with or without a cutaneous ulcer. 5-10% of cases have no visible ulcer (glandular tularaemia). Primary ulceroglandular disease confined to the throat is referred to as pharyngeal tularaemia. Oculoglandular tularaemia is due to exposure of the conjunctivae by a hand contaminated by tissue fluids from an infected animal. Gastrointestinal tularaemia occurs after drinking contaminated ground water and is characterised by abdominal pain, nausea, vomiting, and diarrhoea. The "typhoidal" septicaemic form may occur as a primary condition in 5-15% of naturally-occurring cases with features including fever, prostration, and weight loss, without adenopathy. Diagnosis may be difficult, as signs and symptoms are non-specific and there frequently is no suggestive exposure history. Pneumonic tularaemia is a severe atypical pneumonia that may be fulminant, and can be primary (inhalational) or secondary. Symptoms include fever, headache, malaise, sub-sternal discomfort, and a non-productive cough; radiological evidence of pneumonia or mediastinal lymphadenopathy may or may not be present.

MEDICAL MANAGEMENT

Investigations	**Radiology**: May show pneumonia or mediastinal lymphadenopathy. **Labs**: Blood cultures (difficult), antigen detection, serology and PCR.
MedCM	**Active immunisation**: Vaccination (in development) **Antibiotics (pre & post exposure)**: *Ciprofloxacin* 500mg (oral) every 12 hourly
Treatment regimen(s)	**Immediate severe sepsis resuscitation** **Antibiotics**: *Gentamicin* 7mg/kg (IV) daily OR *streptomycin* 1G (IM) every 12 hours OR *ciprofloxacin* 400mg (IV) every 12 hours. Continue for 14 days.

VIRAL HAEMORRHAGIC FEVERS

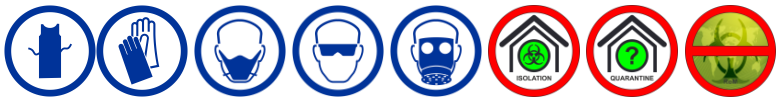
(contagious including Ebola, Lassa, Marburg, CCHF)

CDC **A**

BASIC INFORMATION

TRANSMISSIBLE (Person – Person)	INFECTIVITY	INCUBATION OR LATENCY PERIOD	DURATION OF ILLNESS	LETHALITY
HIGH	HIGH	1-21 days	7-9 days	HIGH

PERSONAL PROTECTIVE EQUIPMENT (PPE) & CASUALTY HAZARD MANAGEMENT



STRICT PRECAUTIONS

WARNING: High level of protection required when handling bodies

VHF ranged from non-transmissible self-limiting and mild (dengue) to fatal and highly contagious illnesses (Ebola, Lassa, Marburg and CCHF). All are zoonoses and have animal reservoirs with insect vectors or exposure to animal excretions. Transmission from person to person includes mucocutaneous exposure to infected blood and body fluids, aerosolisation during medical intervention or inoculation. Natural spread is most likely to be droplet including hematemesis and haemoptysis as well as gastrointestinal (Ebola virus disease).

CLINICAL ASSESSMENT

SYNDROMIC PRESENTATION(S)	Haemorrhagic	Gastrointestinal (Ebola/Marburg)

Clinical features: A febrile prodrome (up to 7 days) precedes the manifest illness. The haemorrhagic syndrome consists of bruising and bleeding. Associated symptoms depend on the virus:
Ebola/Marburg: Rapid fever, vomiting and diarrhoea, maculopapular rash, encephalopathy, hepatomegaly, multi-organ failure, mortality 30-90%.
Lassa: Slow fever, erythematous eyes, facial oedema, chest pain, vomiting and diarrhoea, effusions, encephalopathy, significant recovery with third having hearing impairment. Significant mortality in pregnant woman – up to 50% with 95% in foetuses.
CCHF: Rapid fever, petechial rash, erythematous eyes, sore throat, vomiting and diarrhoea, lethargy, hepatomegaly, CNS features, mortality 30-50%.

Differential diagnosis*:* Malaria, typhoid, meningococcal disease, gram negative sepsis or cause of DIC such as rickettsial HF, late acute radiation syndrome (coagulopathy & neutropenia sepsis).

MEDICAL MANAGEMENT

Investigations	Serology. **Note:** Limit laboratory testing to a minimum.
MedCM	*Ribavirin* – experimental. Candidate vaccines under evaluation.
Treatment regimen(s)	Supportive management. Empirical treatment for other treatable causes (antibiotics and antimalarials if +ve). Experimental anti-virals including *ribavirin* (Lassa, CCHF) and *favipiravir* (potential broad-spectrum RNA antiviral).

CHAPTER 29: MANAGEMENT OF THE BIOLOGICAL OR SEPTIC CASUALTY

29.1. INTRODUCTION

1. Although the presentation of a biological casualty is delayed due to a longer latency or incubation period, treatment on recognition is as great a priority as it is for a chemical or trauma casualty. Rapid recognition, identification of the agent and treatment are paramount to survival with evidence that rapid and aggressive treatment significantly saves lives. It must be started early and directed at clinical and biochemical endpoints. Although the general critical care issues are covered in Chapter 7, management in the first hour following recognition is vital and is likely to start at Role 1 or in the Emergency Department/Room at a Role 2 or 3 MTF.

2. The main priority for biological casualty management is supportive management with empirical and definitive management, where available. Following the recognition of sepsis, a life-threatening condition, it is a requirement to provide LSI and initial sepsis management as soon as possible. For bacterial infections, there is the option of antibiotics as well as supportive management. Antivirals have a limited application and efficacy with a greater reliance on vaccines for pre-exposure prophylaxis or control of an outbreak of a transmissible agent. Antitoxins have been used in civilian scenarios including the management of toxin generating infections (wound botulism) but are agent specific.

29.2. PRINCIPLES OF BIOLOGICAL CASUALTY MANAGEMENT

1. The management of the biological casualties follows the same principles described in Part 1 of this AP including EMT and Casualty Hazard Management. In addition, there are evidence based guidelines on the management of sepsis casualties ('Sepsis Pathway') that can be applied to both natural and BWA disease[1].

2. The principles of biological casualty management are:

   a. Recognition.

   b. Personal safety (see Chapter 9).

   c. Initial resuscitation including empirical treatment (part of EMT).

   d. Casualty hazard management, including possible isolation and quarantine (see Chapter 6).

   e. Source of infection identification and control for treatment as well as supporting forensics, if deliberate release is suspected.

   f. Critical care supportive management.

   g. Definitive management usually following agent identification.

   h. Post-exposure prophylaxis of responders and close contacts.

[1] International guidelines for management of severe sepsis and septic shock 2012. Critical Care Medicine. (2012).

29.3. RECOGNITION

1. The recognition of a biological event on low CBRN threat operations will most likely be a medical diagnosis. For CBRN defensive operations, recognition may be due to the detection of the release of the agent, routine screening of asymptomatic exposed persons and/or diagnosis of symptomatic casualties. These issues have been discussed generally in Chapter 5 and more specifically with regard to biological casualty recognition in Chapter 27.

29.3.1. RECOGNITION

1. The initial management of a biological casualty depends on the presence of organ failure and other life-threatening condition. Life-threatening conditions include:

   a. Septic shock.

   b. Life-threatening toxin exposure including paralysis, coagulopathy and SIRS.

2. A T1 casualty is a patient with septic shock (i.e. BP < 90mmHG after initial fluid management) and requires immediate treatment (See below).

3. Septic patients not in septic shock (T2) should have initial treatment within the first hour following recognition.

4. Non-critical (T3) infections will be treated with standard therapy guided by the biological syndrome and modified once the causative agent has been identified.

29.4. INITIAL RESUSCITATION – SEPSIS 6

1. The septic casualty requires immediate medical management. This should be started as early in the medical chain as possible although some may be limited by the capability of the MTF. The six interventions (sepsis-6) are:

   a. Fluid resuscitation.

   b. Oxygen.

   c. Early antibiotics.

   d. Septic screen (source of infection identification).

   e. Lactate and haemoglobin level.

   f. Urine output monitoring.

2. The resuscitation goals are:

   a. Reversal of lactic acidosis.

   b. Maintain adequate blood pressure > 100mmHg (or palpable radial pulse in austere environment).

   c. Urine output > 0.5ml/kg/hr.

29.4.1. THE ROLE 1 MANAGEMENT OF SEPSIS

1. The recognition of sepsis is more limited at Role 1 but consistent with the new 2016 criteria (described early).

2. The minimum management for sepsis (and presumed severe sepsis) in the pre-hospital environment and at Role 1 (emergency medical treatment) includes:

   a. Fluid resuscitation.

   b. Oxygen.

   c. IV/IO antibiotics (oral if responder unable to give parentral antibiotics).

3. If blood cultures cannot be taken before antibiotics are started at Role 1, 10ml EDTA and 10ml serum sample should be taken, labelled and forwarded with the casualty to the Role 2/3 MTF for further microbiological diagnostics.

4. Where an anti-toxin is available, it should be given as soon as possible but resuscitation facilities including the treatment of anaphylaxis should be available.

29.4.2. THE MANAGEMENT OF SEPTIC SHOCK

The casualty with sepsis and hypotension (< 90mmHg or no palpable radial pulse) will be triaged as T1 (Immediate). Casualties will be treated with a fluid bolus (1000ml or 20ml/kg crystalloid) within ten minutes of recognition by medical personnel. Following an initial fluid bolus, if the hypotension persists the casualty has septic shock and remains T1 triage category.

29.5. SOURCE OF INFECTION IDENTIFICATION AND CONTROL

1. The biological syndrome may suggest the cause for the illness and guide source identification for sampling from the patient. The septic screen should be carried out for any septic casualty. For localised sources of infection such as abscesses, these should be drained and a sample sent for microbiology. Infection prevention and control measures should be maintained and strict adherence to the use of PPE especially if a contagious illness is suspected. Where the source is not obvious diagnosis imaging using computed tomography should be considered looking for localised infections including abscesses, mediastinitis associated with inhalational anthrax and encephalitis in casualties that are pyrexial and have an altered level of consciousness or sensorium.

2. The accurate reporting of clinical findings may be critical in alerting other units to both the possibility and nature of a BW attack. However, attempts to reach a firm diagnosis on clinical grounds alone may not be possible. Rapid identification of the biological agent is of prime importance for casualty and risk management. Molecular biology and immunoassays are likely to provide local provisional diagnostic capabilities. However, establishing a definitive or confirmed diagnosis will often require reference laboratory facilities and in the context of a BW attack will be a requirement for any legal investigation.

29.5.1. SOURCES FOR SAMPLING

Unlike chemical where identification is mainly through environmental detection or toxidrome recognition, biological agent identification is most likely to be through clinical investigations and specifically laboratory testing. Laboratory testing requires clinical specimens for source identification and these include:

a. Blood for cultures and serology.

b. Urine.

c. Faeces.

d. Saliva.

e. Sputum.

f. Central spinal fluid (CSF).

g. Pleural fluid.

h. Wounds and localised lesions including vesicular fluid.

29.5.2. LABORATORY INVESTIGATIONS[2]

1. Specimens taken can be processed in a number of ways and the selection of identification method will depend on the initial diagnosis and suspected agents. Some of the methods used for clinical specimens may also be used in environmental sampling for BA detection.

2. Any request should be supported with the minimum dataset including:

a. Patient identification (last name, first name, date of birth (if known), unique idenfication (ID) / service number).[3]

b. Case history including any relevant travel history and exposure risks.

c. Signs and symptoms including any biological syndromes.

d. Type of specimen or sample.

e. Suspected agent(s) especially if requiing BSL 3/4 laboratory.

f. Any relevant treatment given including antiobiotic therapy.

3. Where deliberate release is suspected, a strict chain of evidence should be adhere to although this is also expected of any laboratory service involved with patient care. Additional information may include:

a. Suspected agent based on threat assessment and situational awareness.

b. Detected agent based on DIM.

4. The laboratory investigations include:

[2] ☑ The QR link provides an example of a laboratory investigation request form (courtesy of the Bundeswehr Institute of Microbiology).

[3] On multi-national operations, the ID or service number may require nation also due as the unique number may only be unique to that nation or have a different format.

a. *Microscopy*. This is a rapid method and appropriate for a deployed MTF. However, it lacks sensitivity and specificity although it may support a provisional diagnosis.

b. *Culture*. The culturing of a specimen on appropriate media, or even cell culture, provides definitive identification of the causative agent. It can allow microorganism typing and assessment of susceptibility to treatments.

Note: The culturing of dangerous pathogens requires highly specialised personnel as well as safe and secure facilities. The diagnosis is likely to be delayed due to the time for the assay depending on the organism.

c. *Serology*. The infection may be identified by the host's immune response to the agent. This uses a number of methods including agglutination, complement fixation, virus neutralisation, enzyme linked immunosorbent assay (ELISA), radioimmunoassay (RIA) and immunofluorescence. The diagnosis is likely to be delayed due to the time for the assay and the usual requirement to detect rising and falling antibody levels over a period of at least a week.

d. *Immunoassays*. This method of detection uses antibodies or immunoglobulins to detect macromolecules with a label. Popular detection methods include enzyme-linked immunosorbent assays (ELISAs). These are particularly useful for toxin detection.

e. *Molecular biology*. Molecular techniques can provide a rapid, sensitive and specific method for agent identification. Methods include DNA hybridisation using specific labelled probes and nucleic acid amplification techniques used for identifying small quantities of nucleic acid material where growth may be slow or dangerous. This is the most sensitive and fastest method (~1-3 hours depending on capability).

f. *Sequencing*. This technique identifies and characterises precisely the organism based on its genetic code. A delay in the diagnosis may be due to sample preparation, sequencing and analysis. This technique may also support molecular epidemiology and bioforensics.

29.5.3. FORENSIC (POST-MORTEM) SAMPLING

A post-mortem examination will include external examination, gross appearance of internal organs and histological samples. Some macroscopic appearances are pathognomonic for some conditions such as the haemorrhagic mediastinitis and haemorrhagic meningitis ('cardinal's cap') associated with anthrax. Some of the sampling sources described above will also be included in post-mortem investigations. In some cases of highly virulent or infectious agents, a post-mortem may require a high level of biosafety and may be restricted to basic sampling in the case of suspected BSL-4 pathogens. Alternative methods of identification of cause of death such as molecular biology or diagnostic imaging (computed tomography or magnetic resonance scanning) may be used.

29.5.4. ENVIRONMENTAL SAMPLING

1. General policies for collecting samples in order to facilitate identification of biological agents are essential. Medical capabilities are primarily focused on the collection and submission of diagnostic (clinical) material from humans and animals. However environmental sampling is another important element in corroborating the occurrence of a BW attack and will require a close collaboration between medical subject matter experts and CBRN units (compare AEP-66: *NATO Handbook for Sampling and Identification of Biological, Chemical*

and Radiological Agents). Close co-ordination and co-operation between CBRN and medical staffs is vital to optimise sampling and interpret any positive findings.

2. Environmental smaples should be:

   a. Identified with a precise location.

   b. Location information and weather conditions.

   c. Potential live stock or vectors.

   d. Suspected agent and any results such as hand-held assays.

29.6. CRITICAL CARE SUPPORTIVE MANAGEMENT

Chapter 7 describes the basis for critical care management that includes the biological casualty even if the causative agent is unknown. The management of the septic casualty is supported by recommendations described as a critical care bundle (see Figure 29-1). The elements of the critical care bundle are listed:

a. *Antibiotic therapy*. A combination of antibiotics is recommended as initial empirical management. Once the agent and sensitivities are known a more focused antibiotic regime is recommended. Antibiotics should be stopped if no bacteria are identified or another agent such as viral, fungal or toxin is identified.

b. *Vasopressors*. These will be used to maintain a MAP > 65mmHg and include norepinephrine and epinephrine.

c. *Inotropic therapy*. These are used to improve cardiac output and drugs include dobutamine and catecholamines.

d. *Blood products*. Packed red cells may be require to maintain the minimum haemoglobin level of 7-9g/dL (70-90g/L) depending on the patient's risk of bleeding, ischemic heart disease and other risk of tissue hypoperfusion. Other products include platelets and fresh frozen plasma.

e. *Ventilation strategies*. Casualties ventilated and septic are at risk of hypoxia due to localised infection (pneumonia) and a complication of sepsis known as Acute Respiratory Distress Syndrome (ARDS). For the latter, a modified ventilation method is recommended using low tidal volume, balancing inspired oxygen and peak end expiratory pressures (PEEP), and tolerating a higher carbon dioxide blood concentration (permissive hypercapnia). This prevents secondary lung injury during excessive volume and shearing forces.

f. *Low dose steroid trial*. This should be considered if the casualty has failed to respond to fluid resuscitation and vasopressors or is on maximal inotrope therapy, especially with evidence of adrenal suppression. However, treatment should not be delayed for specific investigations.

g. Other interventions include sedation, renal replacement, bicarbonate therapy, avoidance of hyperglycaemia, deep vein thrombosis and stress ulcer prophylaxis and a consideration on the limitation of support. The latter may also depend on the size of an outbreak, resources available and mortality associated with the identified agent.

29.7. DEFINITIVE MANAGEMENT

1. Definitive management is limited for biological casualty management until the agent is identified by detection, screening or diagnosis confirmed by laboratory investigation. Antibiotic treatment should be as focused as possible. Antiviral therapy is limited to only a small number of agents with varying efficacy.

2. Toxins may be difficult to identify and some have a very non-specific presentation. Deployed medical detection capability is challenging and the decision to use an antitoxin may require a risk benefit decision using intelligence, detection and comparison with the observed casualty assessment.

29.8. POST EXPOSURE PROPHYLAXIS FOR EXPOSED PERSONNEL

1. Contact personnel, including medical staff, should be assessed if a casualty is identified to be contagious especially if the agent is highly infectious and has a significant mortality or morbidity. Particular points in the medical evacuation chain that may increase the risk are interventions that may cause aerosolisation such as intubation and cardiopulmonary resuscitation. Re-aerosolisation of biological particles and exposure of responders may occur during the decontamination process or while on scene. Post-exposure prophylaxis interventions include the use of antibiotics, vaccination, antitoxins, quarantine and medical screening.

2. Any potential exposures and MedCM use must be recorded.

29.9. MASS CASUALTY CONSIDERATIONS

During an outbreak, triage may be started to optimise care for those requiring finite resources, a further level of care including hospital admission and critical care, as well as priority and suitability for MEDEVAC. In some cases, especially during a mass casualty event, a ceiling of treatment may be implemented including the limiting of certain types of replacement therapy and critical care. Senior clinical decision making will be required supported by a clear understanding of resource availability, casualty flow including TACT and STRAT MEDEVAC, allied support and outbreak prediction with casualty rate estimation. As a general rule, casualties with sepsis and at least three system failure have less than a 50% chance of survival. As well as the triage of biological casualties, the lack of critical beds over an extended period may have an impact on the care of other patients requiring the same resources as well as the overall mission.

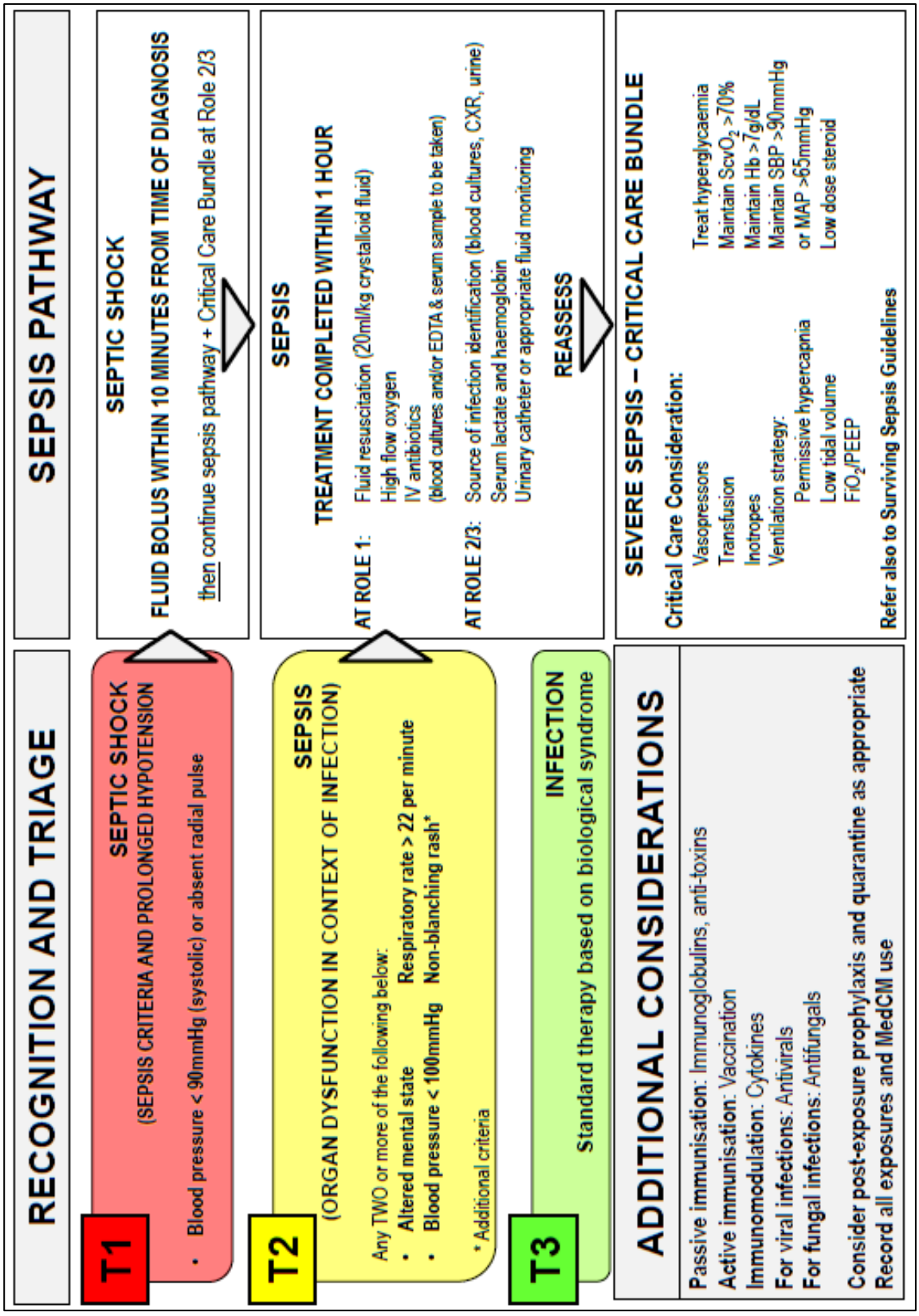


Figure 29-1: Sepsis Pathway for T1 and T2 Biological Casualties.

ANNEX 29A – THE HANDLING OF DIAGNOSTIC SAMPLES

1. When specialised diagnostic systems are available specimens of blood and other body fluids can be taken to allow rapid agent identification. Swabs of contaminated areas such as the nose or throat can also be employed. The sample taken depends on the laboratory apparatus deployed. General principles of the collection and processing of medical samples include the following, notwithstanding the risk from the infective disease:

2. **Specimen Collection**. Human and animal samples may be collected to aid in definitive diagnosis. They must be taken by an adequately trained person; in the case of invasive samples this will be a medically-qualified person. Examples include:

a. Blood culture with routine media will readily detect many bacterial agents, although specialised media may be required for some. Both aerobic and anaerobic cultures should be obtained routinely. If blood culture media are not available at least a 10 ml EDTA blood sample should be taken and forwarded to a microbiological laboratory. An initial patient specimen should always be collected before the beginning of an antimicrobial chemotherapy. If this is not the case, the antimicrobial agent used shall be clearly indicated on the sample documentation.

b. Cultures and impression smears should be taken from involved lymph nodes, sputum, pleural fluid, cerebrospinal fluid (CSF), and spleen when possible. Appropriate swabs should be taken, the minimum being from the upper respiratory tract.

c. Acute serum (at least 3 ml for suspected infectious agents, and at least 20 ml for suspected intoxications) should be collected as early as possible after onset of symptoms. Blood samples should also be obtained from exposed persons who are not yet symptomatic. Convalescent sera from survivors and non-affected unit members should be obtained 3-4 weeks later.

d. Samples for isolation of suspected viral agents should be obtained from organs and tissues as described above, and placed in specialised transport media and frozen for shipment to specified reference laboratories.

e. Tissue samples obtained at autopsy should be collected in multiple aliquots: minimally, one (25-50 grams) to freeze for microbial or toxin analysis and one in formalin for histopathology should be obtained. Where possible additional native specimens (without any additives) should be obtained for testing by polymerase chain reaction assays. Organs sampled should include lung, mediastinal lymph nodes, spleen, and liver. Obvious lesions and adjacent normal tissue should be taken from affected areas in any organ. Post-mortem blood (up to 20 ml) should be obtained.

3. **Specimen Labelling:**

a. Each container should be labelled with name, numerical identities, type of specimen, and date of collection. Included should be a brief description of the illness and gross autopsy findings; location, date, and time of death; location, date, and time of collection; medical officer and unit.

b. All serum samples should be completely labelled with patient's name, numerical identifier, unit, date, originating medical facility, and medical facility to receive results (if different from submitting facility). Routine laboratory slips should be included with each sample. Data on laboratory slips should include number of days since onset of

symptoms, the reason that samples were obtained and a geographical location of collection.

c. Clinical and operational data should be included for all samples, together with a form to establish chain of custody. This requirement must be strongly and clearly delineated since evidence may well be politically or militarily disputed.

4. **Forensic Samples and Chain of Evidence**. In the case of SIBCRA the issue of informed consent must be addressed according to existing medical ethical regulations of the respective NATO nations; a special case is the consent for deceased victims. The need to satisfy international law and prove chain of evidence will be paramount after a BW attack. There may be a requirement to retain a second sample and at least the following information must be provided:

a. When was the sample collected?

b. Who maintained custody of the sample?

c. What was done with the sample at each change of custody?

ANNEX 29B – SHIPPING OF DIAGNOSTIC SAMPLES

1. From a logistical point of view infectious substances are considered as dangerous goods and must always be transported according to the appropriate national and international regulations. These regulations also apply to NATO forces. A nominated shipper should be identified and suitably qualified as this person will be in charge of the consigning process involving the handling, packaging and sending of any infectious materials. Regulations, issued by the International Air Transport Association (IATA), regulate materials transported by air and are generally the most restrictive. For these reasons, this Annex outlines the IATA protocols.

2. Human or animal microbiological specimen as well as all environmental CBRN samples should be generally rated as at least potentially infectious and be classified and treated accordingly. There is a distinction between known infectious substances (Class A) and diagnostic specimens (Class B).[1] The two classes are described as:

a. Class A is a pathogen which is transported by any mode in a form that, when exposure to it occurs, is capable of causing permanent disability, life-threatening or fatal disease to humans (UN2814) or animals (UN2900).

b. Class B is an infectious substance that does not meet the criteria for inclusion in Category A and must be consigned/shipped as UN3373.[2]

3. The packaging requirements are determined by the United Nations (UN) and are contained in ICAO and IATA regulations in the form of Packaging Instructions (PI) 602 and 650. The requirements are subject to change by these organisations. The current packaging requirements are described below. UN-approved packaging systems are available commercially.

4. The basic triple packaging system consists of three layers as follows:

a. *Primary receptacle*. A labelled primary watertight, leak-proof receptacle containing the biological sample. The receptacle is wrapped in enough absorbent material to absorb all fluid in case of breakage.

b. *Secondary receptacle*. A second durable, watertight, leak-proof receptacle to enclose and protect the primary receptacle(s). Several wrapped primary receptacles may be placed in one secondary receptacle. Sufficient additional absorbent material must be used to cushion multiple primary receptacles.

c. *Outer shipping package*. The secondary receptacle is placed in an outer shipping package which protects it and its contents from outside influences such as physical damage and water while in transit.

[1] These IATA categories A and B must not be confused with the U.S. Centres for Disease Control and Prevention (CDC) classification of biological agents.

[2] Professional judgment, preferably by a microbiologist, is used to determine if a biological sample contains pathogens and should be based on symptoms, local conditions and individual circumstances. If there is more than a “minimal likelihood” that a sample contains a biological agent, *it must be shipped as a Category A infectious substance*. Category B (UN3373) is generally used for a general diagnostic sample rather than for a suspected CBRN agent.

5. Sample data forms, letters and other types of information that identify or describe the sample and also identify the shipper and receiver should be taped to the outside of the secondary receptacle.

6. The maximum net quantity of infectious substances which can be contained in an outer shipping package IS 50 mL or 50g if transport is by passenger aircraft. Otherwise, the limit per package is 4L-4Kg for transport by cargo aircraft or other carriers. Primary receptacles e.g. 50 mL in combination packing must be oriented so the closures are upward, and labels (arrows) indicating the "UP" direction must be placed on two opposite sides of the package. The passenger aircraft quantify limits do not apply to blood or blood products for which there is no reason to believe they contain infectious substances, when in receptacles of not more than 500 mL each and with a total volume of not more than 4L in the outer package.

7. For all dangerous goods to be shipped by airfreight, specific hazard label(s) must be affixed to the outside of each package.

8. Labelling of the outer package for shipment of infectious substances must include the elements listed hereafter.

a. The International Infectious Substance Label.

b. An address label with the following information:

(1) The receiver's (consignee) name, address and telephone number

(2) The shipper's (consignor) name, address and telephone number

(3) The UN shipping name (Infectious Substances Affecting Humans or Animals as the case may be) followed by the scientific name of the substance

(4) The UN Number (Humans – UN2814, Animals UN2900)

(5) Temperature storage requirements (optional).

c. Required shipping documents – these are fixed to the outer package:

(1) The Shipper's Declaration of Dangerous Goods

(2) A packing list

(3) A proforma invoice which includes the receiver's address, the number of packages, detail of contents, weight and value might also be required for customs purposes

(4) an airway bill if shipping by air

d. An import and/or export permit and/or declaration if required.

9. If the outer package is further packed in an over pack (with dry ice for instance) both outer pack and over pack must carry the above information, and the over pack must have a label stating "INNER PACKAGES COMPLY WITH PRESCRIBED SPECIFICATIONS".

10. If the outer package contains primary receptacles e.g. 50 mL in combination at least two "Orientation labels" (arrows) must be placed on opposite sides of the package showing correct orientation of the package.

AMedP-7.1 PART 5: MANAGEMENT OF RADIOLOGICAL / NUCLEAR CASUALTIES

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CHAPTER 30: INTRODUCTION TO RADIOLOGICAL / NUCLEAR HAZARDS AND THREATS

30.1. INTRODUCTION

1. The change from NBC to CBRN defence reflects the recognition of threats and hazards from radiological and nuclear materials and weapons. Radiological threats are usually associated with a single radioisotope and varying levels of radiation. The nuclear threat is associated with the fission process of splitting the atom. This generates a massive amount of energy in the form of blast, heat and ionising radiation including neutrons as well as multiple radioisotopes as fission products. Chapter 34 describes specifically the effects from a nuclear incident including nuclear detonation and weapons accidents.

2. Expected casualty types depend on the scenarios described below. Casualty types include the following types or combinations:

a. External contamination.

b. Internal contamination (including intake, uptake and deposition).

c. Wound contamination.

d. Irradiation.

e. Combined injuries (irradiation and trauma).

3. Nuclear casualties due to a nuclear detonation may be a combination of external, internal and wound contamination, blast and thermal injuries and irradiation.

30.2. RADIOLOGICAL HAZARDS AND THREATS

Radiological hazards and their possible deliberate use as a weapon can be overt or covert. Effects may be contamination and/or irradiation. The presence of trauma depends on the method of delivery i.e. ‘dirty bomb’. A summary of the more significant radionuclides and isotopes and their uses is in Table 30-1.

30.2.1. POINT SOURCE / RADIATION EXPOSURE DEVICE

1. Radioactive material, in a sealed source or within a container, can be encountered unintentionally at any time. Nearly all of the reported instances of significant radiation injury since 1945 have occurred as a result of accidents. Discarded or lost (‘orphan’) sources and other radioactive material are most likely to be found in cities, abandoned industrial/medical facilities and isolated waste dumps. Unless an area has been completely surveyed for radioactive contaminants, military personnel on routine deployed operations may encounter radiological hazards at any time. Medical providers must be alert to the possibility of radiation exposure as a complicating factor in nearly all operational environments. This will be true especially in the event of civil disorder or failure of the usual civic infrastructure.

2. A point source may be used to expose people intentionally to a high external radiation dose rate. A covert attack using a *radiation exposure device* (RED) could be used as a terrorist tactic. Industrial radiography sources constitute the most prevalent REDs in the civilian sector. Brachytherapy and industrial sterilisation sources are also common.

Note. As long as the source remains sealed then exposed persons will be irradiated only. There will be no contamination risk once source has been confirmed as intact or the exposed persons screened for contamination.

30.2.2. FOOD/WATER CONTAMINATION

1. The covert contamination of the food or water chain with a radionuclide is another threat, although a single isotope is likely to be used. This will result in personnel with internal contamination and varying degrees of irradiation depending on the isotope and dose ingested (intake).

2. Absorption from the gastrointestinal tract (uptake) depends on the chemical properties of the radionuclide and the form of the compound (organic/inorganic/metallic or soluble/insoluble). For example, radiocaesium in its salt form is rapidly absorbed, but heavy metal radionuclides in their metallic form are not. An example of a potent and absorbable alpha-emitter is Polonium-210 (^{210}Po).

3. The gastrointestinal tract is considered the target organ for ingested insoluble radionuclides. The large intestine receives the greatest radiation exposure due to its slower transit time. The clearance time for the gastrointestinal tract is approximately 24 hours for individuals who maintain a high-fibre diet. Individuals on a low-fibre diet have a slower transit time that may extend up to 5 days. Even in these individuals, insoluble alpha-emitting isotopes, such as uranium and plutonium, with long half-lives will not cause significant damage because the exposure time within the critical organ is relatively short.

30.2.3. RADIATION DISPERSAL DEVICE

1. A *radiation dispersal device*, or RDD, is defined as “any device, including any weapon or equipment, other than a nuclear explosive device involving a nuclear chain reaction (known as “criticality”), specifically designed to employ radioactive material by disseminating it to cause destruction, damage, fear, or injury by means of the radiation produced by the decay of such material.” RDDs can be used to deny access to an opposing force / terrorist by contaminating an area, compounding a conventional explosive incident with contaminated casualties and wounds, or creating illness or panic by contaminating food or water supplies (see above). An RDD may be used for the psychological impact it may have on troops as well as civilian populations, however the hazard is detectable and quantifiable.

2. Typically RDDs are low-technology devices that do not involve the massive release of energy of a nuclear chain reaction. Similar to RED, medical sources, industrial irradiators and radioactive waste can be used in a RDD. Such a weapon can be easily developed and utilised with conventional or improvised explosives (‘dirty bomb’). Reactors can be used to produce specific radionuclides, although this may require more complex technology and sophisticated techniques or be targeted directly.

3. The explosive charge used in an RDD will cause injuries associated with blast. These conventional casualties will have external and potentially internal contamination with wounds embedded with radioactive material. This may complicate medical evacuation. However, traumatic injuries take priority as they are more likely to be life-threatening than the radiological component.

Table 30-1: Examples of Common Radionuclides and Uses.

Medical facilities and research	**^{3}H (tritium), ^{14}C, ^{32}P, ^{99m}Tc, ^{103}Pd, ^{125}I, ^{131}I, ^{133}Xe, ^{137}Cs, ^{201}Tl** ^{137}Cs is used as a gamma source for external beam therapy (radiotherapy) usually where a linear accelerator (LINAC) is not available. ^{103}Pd, ^{125}I and ^{192}Ir are used for brachytherapy implants. ^{131}I is used for the treatment of thyroid cancer and thyrotoxicosis. ^{99m}Tc, ^{133}Xe and ^{201}Tl are both used for nuclear medical diagnostics. A number of radioisotopes of common elements in biology (e.g. hydrogen, carbon and phosphorous) are used to trace biological processes and pharmacokinetics.
Science institutions	**^{3}H, ^{14}C, ^{241}Am, ^{252}Cf** ^{3}H and ^{14}C are common radioactive tracers. ^{241}Am and ^{252}Cf are both used as neutron emitters.
Industrial radiography & irradiators (gamma-emitters)	**^{60}Co, ^{137}Cs, ^{192}Ir** ^{192}Ir is widely used in industrial radiography (non-destructive testing) to image large objects such as pipes, airplane wings and infrastructure. ^{60}Co is a potent gamma source used for the sterilisation of medical material and food substances. ^{137}Cs is less commonly used due to its powdered form and solubility.
Nuclear power	**^{235}U, ^{239}Pu** These are the most common radionuclides used as nuclear fuel.
Nuclear weapons	**^{3}H, ^{235}U, ^{239}Pu** These radionuclides are the main components of a nuclear weapon. ^{235}U and ^{239}Pu are both fissile metals that can be shaped into forms that allow criticality to be achieved. ^{3}H provides a fusion (thermonuclear) enhancement using the energy generated by the fission reaction.
Fission products	Products of a nuclear (fission) process, either as a result of weapon detonation or power reactor accident. Significant fission products include the radioisotopes of strontium, caesium and iodine.
Radioisotope thermoelectric generators (RTG)	**^{90}Sr**, **^{238}Pu**. Both radioisotopes have been used to power radioisotope thermoelectrical generators usually in remote locations or satellites. ^{90}Sr is a beta emitter and ^{238}Pu is an alpha emitter.
Miscellaneous	Tritium (^{3}H) is used widely to produce luminous paint.
	^{210}Po has been used as a radiological poison as well as used for static electricity elimination.
	^{241}Am is commonly found in small amounts (<1µg) in ion-chamber smoke detectors.
	Depleted uranium (DU) has proportionally more ^{238}U than natural uranium and is a very weak alpha and x-ray emitter. As a battlefield hazard, DU should be considered a chemical hazard with renal toxicity rather than a significant radiological hazard. (See Annex 35D for medical guidance)

30.3. NUCLEAR HAZARDS AND THREATS

Nuclear devices are associated with the fission process (see Figure 30-1). This leads to significant amounts of ionising radiation (including neutron radiation) as well as heat and potentially blast. A nuclear incident may release many *fission products / fragments (FP/FF)* consisting of multiple radionuclides (e.g. radioiodine, radiocaesium), and isotopes of each nuclide (^{129}I,^{131}I,^{132}I). In addition, the release of neutron radiation will *induce* radioactivity in surrounding lighter nuclides such as sodium and iron to form a radioactive isotope that will then decay to emit further ionising radiation. The main nuclear threats come from nuclear weapons of varying size (yield) and an attack or accident at a nuclear power reactor.

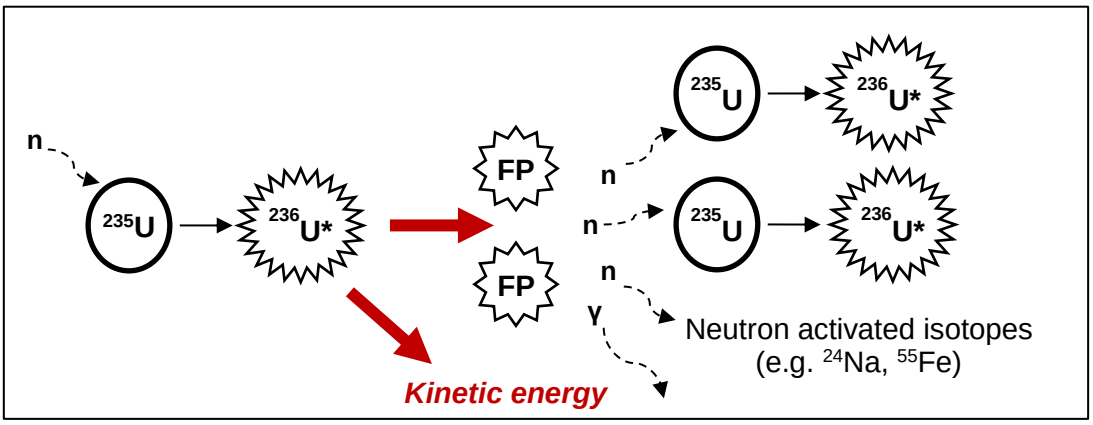


Figure 30-1: The Fission Process (showing supra-criticality chain reaction).

30.3.1. NUCLEAR WEAPON

1. The detonation of a nuclear weapon leads to a number of effects. A detonation exposes people to high levels of external radiation, trauma, external contamination, and inhalation and ingestion of radioactive materials especially in the radioactive fallout. The actual observed effects depend on the yield of the weapon and type of attack (air, ground, subterranean or submerged). The effects of a nuclear detonation are:

   a. Flash.

   b. Blast.

   c. Thermal energy.

   d. Ionising radiation, both initial and residual.

   e. Electromagnetic pulse (EMP).

2. The first nuclear weapon detonated over Hiroshima had an estimated explosive force (yield) of 15 – 18 kilotons (kT) of TNT. The yield of modern nuclear weapons varies but the larger devices are measured in megatons (MT). Small-yield tactical nuclear weapons might also be used on the battlefield.

3. Following a nuclear detonation, large numbers of casualties with combined injuries would be generated and local resources are likely to be overwhelmed or compromised as well. Initial medical management at the periphery of the lethal zone is focused on triaging and distributing causalities to multiple MTFs (regional and international). This type of catastrophic event would require the rationing of resources and the provision of palliative care. The expectant (T4) triage category for high dose exposures with or without concurrent trauma (combined injury) is likely to be used.

30.3.2. IMPROVISED NUCLEAR DEVICE

1. The proliferation of nuclear material and technology has made the acquisition and use of radiation device weapons more probable, particularly as a mode of asymmetric warfare and terrorism. An *improvised nuclear device* (IND) is a device that could be designed by terrorists to produce a nuclear detonation. At full or partial yield, an IND is physically the same as a nuclear weapon generating blast, burns and irradiation.

2. If an IND fails to detonate properly and thereby not achieve a supercritical nuclear chain reaction, the high explosives will disseminate the fissile material into the environment and effectively becomes a RDD. In some circumstances, a finite period of criticality ('fizzle') might occur but is not sustained long enough to cause a supercritical nuclear chain reaction but causes an intense release of gamma and neutron radiation, and the generation of activation products. This is similar to *criticality accidents* previously seen in the nuclear fuel and weapon manufacturing industry.

30.3.3. NUCLEAR POWER REACTOR

NATO forces may be operating in a theatre that has nuclear reactors with varying degrees of security, safety and containment. Tactics may require NATO forces to deploy downwind of these reactors. The nuclear reactors and their containment facilities may be critical nodes of enemy artillery or bombing targets because of both the disruption of electric power and the radionuclide release, possibly in large amounts. The resources then diverted to the crisis and consequence management of contaminated and irradiated NATO and host nation personnel may be overwhelming. Nuclear reactors are also vulnerable to collateral damage and, particularly during governmental instability, may suffer from degraded security, maintenance, management, and operation, thereby increasing the risk of accidents.

Table 30-2: Radiological and Nuclear Scenarios.

Scenarios	Targets	Casualties (number and types)	Radioiodine internal risk?	Types of radiation	EMP present?
RED: hidden source intended to expose people in a busy place, and late discovery	COY, FOB, civilian location	ARS 10s – 100s	No	Single isotope (α), β, γ	No
Contamination of food and water supplies	Civilians & military	IC 1,000s – 100,000s	If used	Single isotope α, β, γ	No
RDD: dispersal attack ± IED (dirty bomb)	SEC	IC-EC 10-100 Immediate 0 – 10s Delayed 0 – 10s	If used	Single isotope α, β, γ	No
	FOB	IC-EC 100s -1000s Immediate 0 – 10s Delayed 0 – 100s			
Attack on transport of radioactive material that could be used in RDD or RED	Civilians & military	Variable	If spent fuel rods or radioiodine and released*	α, β, γ	No
Criticality event	Weapon/ IND handler	Highly localised	Unlikely	γ, n (& secondary)	No
Nuclear detonation (0.1kT)	FOB	IC-EC ≥1000 ARS 10s – 100 Immediate 0 – 100s Delayed 0 – 100s	Yes, more significant for responders due to radioiodine in fallout*	Fission products α, β, γ, n	Yes
Nuclear detonation (100kT)	Civilians & military	> 100,000s ARS, IC, EC			
Attack on a nuclear power reactor	Civilians & military	IC-EC ≥1000 ARS 10s – 100 Immediate 0 – 100s Delayed 0 – 100s	Yes*	Fission products α, β, γ, (n)	No

* The use of stable iodine as a MedCM will depend on the predicted internal dose, risk:benefit and age of population at risk.
ARS: acute radiation syndrome; EC: external contamination; COY: company; EMP: Electromagnetic Pulse; FOB: forward operating base; IC: internal contamination; IED: improvised explosive device; RDD: radiological dispersal device; RED: radiological exposure device; SEC: section.

30.4. TYPES OF IONISING RADIATION

There are four biologically significant types of ionising radiation that may be encountered in military operations including those derived from a nuclear detonation: alpha particles, beta particles, x and gamma rays (high energy photons) and neutrons (Fig. 30-1). The types of ionising radiation are discussed in the following paragraphs and summarised in Table 30-3.

30.4.1. ALPHA PARTICLES

Alpha particles are made up of two protons and two neutrons (helium nucleus) and are typically emitted from heavy nuclei including U, Pu, and Am. There may also be an associated high energy photon emission and this may be exploited for detection. The alpha particle is relatively heavy and carries a double positive charge. The energy of this form of ionising radiation is fully absorbed within the first 20 micrometres of exposed tissue causing significant ionisation, and as such is described as having a high *linear energy transfer* (LET). This increases alpha radiation's biological damage (*relative biological effectiveness* (RBE)) and is associated with a *radiation weighting factor* (W_R) of 20 times greater than gamma or beta radiation. For external contamination, all of the alpha radiation is absorbed in the superficial layers of dead skin cells, and therefore is not considered an external hazard. Alpha particles can therefore be shielded by paper, including overalls and masks, to prevent internal and wound contamination. If alpha-emitting radioactive material is introduced internally usually by inhalation or ingestion, significant damage can occur. This damage includes sensitive tissue along the respiratory and gastrointestinal tract as well as distributed to other target tissues based on the material's physical and biochemical properties. Internalise alpha-emitting radionuclides are more associated with cellular damage rather than ARS. The exception to this is ^{210}Po which has a relatively short half-life and a large volume of distribution when absorbed internally, leading to significant effects including ARS.

30.4.2. BETA PARTICLES

Beta particles are energetic electrons emitted from neutron rich atomic nuclei, including fission products, by *beta decay* causing a neutron to convert to a proton and electron. The beta particle carries a single negative charge, is significantly smaller than an alpha particle and can only penetrate a few millimetres of tissue. If the beta-emitting material is on the surface of the skin, the resulting beta irradiation causes damage to the epithelial basal stratum (*beta burn*). If the radionuclide is introduced internally, the distribution depends on the physical and biochemical properties of the radioactive material. Beta particles can be shielded by sheets of layers of plastic or a sheet of metal e.g. aluminium.

30.4.3. GAMMA (HIGH ENERGY PHOTON) RADIATION

Gamma radiation is a form of electromagnetic radiation (photons) emitted by reactions in the nucleus such as alpha / beta decay or nuclear fission. It is a non-particulate high energy photon, with a short wavelength and high frequency, and thus is capable of creating ionisation. X-rays are similar but are emitted from the electron shell due to electron energy shifts created by electricity rather than nuclear decay and on average have less energy.[1] High energy photons are highly penetrating and a large fraction may pass through the human body without interaction. Energy deposition can occur anywhere in the body along the gamma photon's path. A significant portion of the body may be exposed to gamma radiation during a nuclear

[1] Due to the overlap of x-ray and gamma wavelengths and energy, low energy gamma (as associated with americium or plutonium alpha decay) are sometimes referred to as x-rays.

detonation or by a potent gamma source such as a cobalt-60 industrial irradiator. This is in contrast to the highly-localized exposure pattern that occurs with alpha and beta radiation. High-energy gamma sources near, on or within the body may result in a both local radiation injury and whole-body irradiation. Dense materials such as lead or concrete are used to shield (attenuate) gamma radiation.

30.4.4. NEUTRONS

Neutrons are uncharged particles and are usually associated with the nuclear fission process. Unlike the first three types of radiation, neutron radiation is not uniform and neutrons will have significantly different energies and interactions with nuclei in their path. This also alters their RBE. 'Slow' or 'thermal' neutrons are more likely to interact with atomic nuclei (neutron capture) and may create new isotopes as well as gamma radiation; this is also called *neutron activation*. This is an important form of secondary radiation following a nuclear detonation. Any resulting unstable radioisotope may undergo beta decay. Collisions with nuclei may also cause ionisation indirectly by elastic recoil which may cause ionisation in other atoms. 'Fast' uncharged neutrons may pass straight through matter unless they strike a nucleus directly, they are therefore very penetrating. Because thermal neutrons have greater interaction with low-weight nuclei including hydrogen, they have a variable and relatively high RBE (up to 20 times) compared to gamma and beta radiation. Neutron radiation will result from a criticality accident. Example include a weapon core accident or uranium processing, where a critical mass of fissionable material is handled in such a way as to allow a high enough neutron flux to sustain a brief chain reaction without explosive detonation.

Table 30-3: Types of Ionising Radiation with Examples.

Particle	Symbol	Components	Charge	Range		Weighting Factor
				Air	Tissue	
Alpha	α $^{4}_{2}He$	Helium nucleus (2 protons, 2 neutrons)	**++**	Millimetres	< 20 micrometres	20
Examples of alpha emitters: **^{235}U, ^{238}U, ^{239}Pu, ^{241}Am, heavy radionuclei**						
Beta	β e^{-1}	Electron	**-**	Centimetres	Several millimetres	1
Examples of beta emitters: **^{90}Sr, ^{131}I, fission products, neutron activated radioisotopes**						
Gamma	Γ	Electromagnetic radiation (very high frequency / low wavelength)	Nil	Kilometres	Penetrating	1
Examples of gamma emitters (associated with beta): **^{60}Co, ^{137}Cs, ^{192}Ir**						
X-ray*	X	Electromagnetic radiation (high frequency / low wavelength)	Nil	Kilometres	Penetrating	1
Neutron	N $^{1}_{0}n$	Neutron	Nil	Kilometres	Penetrating	Up to 20
Examples of neutron emitters: **^{252}Cf** (spontaneous), **^{235}U, ^{239}Pu** (during fission process following neutron capture), **^{241}Am** (combined with Be)						

* X-rays originate from the electron shell while the other forms of ionising radiation including gamma are emitted from the nucleus.

30.5. RADIATION UNITS AND MEASUREMENTS

A number of radiation units are used.

a. *Radioactivity (Becquerel)*. The Becquerel (Bq) is one nuclear disintegration (decay event) per second; the unit of radioactivity is often expressed in millions (MBq). One Curie (Ci) = 37GBq.

b. *Radiological half-life ($t_{½}$)*. The half-life is the time required for the activity of a given radioactive species to decrease to half of its initial value due to radioactive decay. The half-life is a characteristic property of each radioactive species and is independent of its amount or chemical properties.

c. *Effective half-life*. The effective half-life of a given isotope is the time in which the quantity in the body will decrease to half as a result of both radioactive decay and biological elimination. This period of time can be reduced by increasing the elimination of the isotope using MedCM such as blocking and chelating agents.

d. *Absorbed dose (gray)*. For acute effects of ionising radiation, absorbed dose is measured by the gray (Gy). One gray equals 1 joule per kilogram. The acute effects of radiation are directly related to absorbed dose and are called *deterministic*. In general, deterministic effects are not seen until over a dose *threshold*. A non SI unit is the rad; where 100 rad = 100 cGy = 1Gy.

e. *Equivalent and effective (sievert)*. The measurement of long-term and late effects of radiation corrects for the more damaging effect of some forms of radiation such as alpha and neutron (equivalent dose) as well as the organ-specific radiosensitivity (effective dose) – Table 30-4. This is used to predict effects termed *stochastic* because these effects are based on probability. For stochastic effects, it is assumed that there is no dose threshold for injury and that with higher doses, the probability of injury increases proportionally. A non SI unit is the rem where 100 rem = 1Sv. A summary of the relationship of each type of dose is in Figure 30.2.

f. *Free-in-air Dose*. Free-in-air Dose reflects the radiation that would be measured in air at a certain point. Military tactical dosimeters measure free-in-air doses.

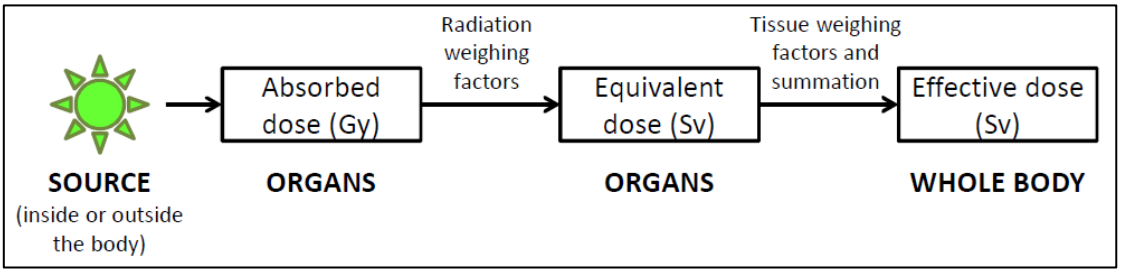


Figure 30-2: Radiation Doses.[2]

[2] Adapted from ICRP Publication 121.

Table 30-4: Tissue Weighting Factors.

Tissue	Tissue weighting factor (W_T)	Sum of W_T
Bone-marrow (red), colon, lung, stomach, breast, remainder tissues[a]	0.12	0.72
Gonads	0.08	0.08
Bladder, oesophagus, liver, thyroid	0.04	0.16
Bone surface, brain, salivary glands, skin	0.01	0.04
Total		1.00
[a]Remainder tissues: Adrenals, extrathoracic (ET) region, gall bladder, heart, kidneys, lymphatic nodes, muscle, oral mucosa, pancreas, prostate (♂), small intestine, spleen, thymus, uterus/cervix (♀).		

30.6. TYPES OF RADIOLOGICAL EXPOSURE

The prevention and management of radiological casualties including nuclear depends on the type and severity of any exposure. The type of radiological exposure and some of the medical considerations include:

a. Contamination.

(1) External contamination.

(2) Internal contamination.

(2) Wound contamination.

b. Irradiation.

(1) Low dose whole body irradiation, below the threshold for deterministic effects, although an increased risk of stochastic effects may remain.

(2) High dose whole body irradiation, above the threshold for deterministic effects as well as stochastic effects.

(3) Local radiation injury including beta burns.

c. Trauma including blast and burns from conventional explosives or nuclear.

d. Psychological effects.

Note: Casualties that have been irradiated only do not pose a risk to responders. Contamination may be a secondary hazard from the casualty (casualty hazard) either directly from external and wound contamination, indirectly from internal contamination due to excretion products and even as an irradiation hazard due to high dose rate shrapnel.

30.6.1 TYPES OF RADIOLOGICAL CASUALTIES

Casualties classified by the clinical effects are:

a. *Radiological casualty*. A casualty caused as a result of exposure solely to ionising radiation either due to high dose irradiation or treatment for internal contamination.

b. *Nuclear casualty*. A casualty caused by a nuclear detonation. These casualties are likely to also have concurrent trauma due to flash, thermal burns and blast (combined injuries).

c. *Combined casualty*. A high dose radiological casualty with concurrent traumatic injuries.

d. *Psychological casualty* (see section 2.9).

30.6.2. RADIOLOGICAL CASUALTY MANAGEMENT CONSIDERATIONS

The prevention and management of radiological casualties including nuclear may be complex with a number of considerations to both provide optimal care for the casualty while also maintaining personnel safety. The operational considerations include:

a. Implementation of radiological pre-exposure MedCM for the deployed force.

b. Implementation of radiological post-exposure MedCM for the deployed force.

c. Management of life-threatening trauma.

d. Assessment and removal of any external radiological contamination.

e. Assessment and removal of any internal radiological contamination.

f. Assessment and removal of any wound contamination.

g. Assessment (or estimation) of any acute radiation dose.

h. Assessment and treatment of acute radiation syndrome (ARS).

i. Assessment and treatment of any local radiation injury including burns.

j. Provision of reverse barrier nursing for immunosuppressed ARS casualties.

k. Assessment and risk communication of any long term health risk.

l. Public health risk communication to deployed force.

m. MASCAL management including the use of the Expectant (T4) triage category.

CHAPTER 31: RADIOLOGICAL DETECTION & PRINCIPLES OF RADIOLOGICAL PROTECTION

31.1. INTRODUCTION

Radiation detection is vital for both force protection and responding to a radiological or nuclear threat. While most biological agents and some chemical agents are not easily detected or monitored, ionising radiation is very detectable at levels below significant health effects. With effective detection and monitoring, it is possible to not only respond within safe working limits using the principles of radiological protection but to estimate potential health outcomes based on personal and location dosimetry. The biological effect of ionising radiation can also be detected using biodosimetry and this is described in Chapter 33.

31.2. DETECTION METHODS

1. Management of radiological or nuclear casualties includes the use of multiple methods of radiation detection and measurement. In the field, hand-held radiation survey instruments and personal dosimeters are most likely to be used to determine radiation and contamination levels including personnel contamination. Field equipment will also be used to quality assure the decontamination process. At a fixed base, where there is electrical power available, a portal monitor will allow a much larger number of personnel to be screened for contamination in a short period of time and larger laboratory based analytical instruments can be used to do clinical and environmental sample analysis. Biological markers can also be used to estimate absorbed dose (bioassay). Operational contamination and dosimetry applications include:

a. Area contamination and dose rate survey.

b. Airborne contamination monitoring.

c. Personal dosimetry.

d. Personnel and equipment contamination survey.

e. Radioisotope identification, leading to appropriate decorporation therapy.

f. Portal monitoring.

g. Biodosimetry (see Chapter 33).

2. Detectors and monitors may be real-time or retrospective. In the case of dosimeter, they may provide a real-time dose rate and/or cumulative dose. The choice of detector should be discussed with a radiation protection advisor.

31.2.1. AREA CONTAMINATION AND DOSE RATE SURVEY

Scene assessment is a vital part of force and radiation protection in order to both detect the radiological hazard and quantify the risk. The type of instrument used will depend on the type of radiation and information required. Measurements and information will vary also depending on the type of survey:

a. *Contamination survey*. This survey will be used to detect and measure the presence of radioactive material usually measured as counts per second (cps) or calculated for a type of radiation as activity per unit area ($Bq.m^{-2}$) or disintegrations per

minute per centimetre square ($dpm.cm^{-2}$). Survey monitoring for contamination is often based on a threshold of twice above background radiation. The detector used and measurement calculation or correction may depend on the type of radiation (alpha, beta, gamma and neutron) as well as the specific radioisotope and energy level.

b. *Dose rate survey or fixed point monitoring*. This detection will usually provide real-time dose rate monitoring of an external source usually beta, gamma or neutron. Measurements will be based on dose per unit time (such as µSv/hr). This may be in the form of a hand held device for surface or directional monitoring. An alternative mode of operation is stationary monitoring at a fixed point usually to monitor a radiation source or reactor potentially with variable shielding such as a gamma irradiator.

31.2.2. AIRBOURNE CONTAMINATION MONITORING

This detection method also called *airborne particulate monitoring* is used for airborne contamination or gas. This is normally at a fixed point although detectors may be mounted on a mobile platform especially following an incident. Information may be used for plume modelling, in accordance with ATP-45, and / or as a trigger for initiating protective measures such as sheltering, evacuation or MedCM use.

31.2.3. PERSONAL DOSIMETRY

1. Personal dosimetry usually consists of a small device worn on a person to measure the radiation dose arising from an external source. Personal dosimeters generally fall into two types:

a. *Direct (active) reading dosimeter*. This form of dosimeter provides real-time dose information either by an electronic personal dosimeter (EPD) or quartz fibre dosimeter. The EPD may be programmed to provide dose rate, total dose and accumulated dose if personal issue and re-worn. A quartz fibre dosimeter usually provides an indication of total dose before being reset.

b. *Indirect (passive) reading dosimeter*. This form of dosimeter provides retrospective dose information usually as a thermoluminescent dosimeter (TLD) or film badge dosimeter. These dosimeters are usually used for relatively low level occupational radiation exposures, such as medical personnel and radiation workers, not expected to exceed annual limits.

2. Dosimetry may be applied either to an individual or to a group (cohort).

a. *Individual dosimetry*. This form of dosimetry requires each person to carry a dosimeter usually for occupational and medico-legal reasons. Either type of dosimeter (direct or indirect) may be used, although the TLD is a common dosimeter for this use.

b. *Group (cohort) dosimetry*. This form of dosimetry uses a dosimeter carried by one member of a group (minimum of one per ten persons). Any dose accrued is recorded for all members of the group. This use of dosimetry may be applied to an operational environment as *operational dose management* as part of collective protective measure or for an incident response team.

c. *Local area or cordon dosimetry*. This form of dosimetry uses a fixed point dosimeter in the same way as group dosimetry but for a fixed location or cordon reflecting the maximal dose rate point to a known hazard.

d. *Internal dosimetry*. This form of dosimetry calculates or estimates the total internal dose deposited within the body of an individual due to internal contamination. This may be based on whole body dosimetry or calculated from samples such as nasal swabs or urinary excretion (see Chapter 35).

31.2.4. PERSONNEL AND EQUIPMENT CONTAMINATION SURVEY

This survey is carried out once personnel, including casualties, and equipment have left a potentially or confirmed contaminated area. The type of monitor will depend on the type of hazard and radiation but are similar to those used for area contamination surveys. Further details of casualty external contamination monitoring is given in Chapter 35.

31.2.5. RADIOISOTOPE IDENTIFICATION – GAMMA SPECTROSCOPY

Each radioisotope emits an energy spectra associated with gamma ray emission that can be used for identification. Identification of the radionuclide is important for the management of internal contamination including the selection of the correct decorporating agent. The use of gamma spectrometers is possible on scene or following decontamination, using contaminated items such as clothing.

31.2.6. PORTAL MONITORING

Monitors at point of entry and exit can be used to screen persons that may have been contaminated with radioactive material. The detectors monitor for gamma, and in some cases neutron radiation and are either pre-deploy at critical infrastructure, deployed on scene or at medical facility entrances as a rapid screen depending on the portal's sensitive and radiation sources of concern.

Note: Portals and other dosimeters will not detect persons that have been irradiated but are not contaminated.

31.2.7. RECORDING OF IONISING RADIATION EXPOSURES

The recording of contamination and dosimetry information is required in cases of suspected and confirmed radiological exposures as described in AMedP-7.8: *Recording of Operational Ionizing Radiation Exposure for Medical Purposes and Management of Dosimeters*.

31.3. RADIOLOGICAL PROTECTION

ALARA (As Low As Reasonably Achievable)[1] is the underlying philosophy associated with protecting people from ionising radiation. It requires that personnel should not be exposed unnecessarily to radiation and the benefit should outweigh the risk (justification), dose limitation and optimisation (ALARA). Time, distance, and shielding (TDS) are the primary methods of radiation protection with administrative controls. The principles are also described in the Internal Committee for Radiation Protection (ICRP) report series including recommended dose limitations for occupational exposure and emergency response. The ICRP guidance does

[1] Some nations use the phase of 'as low as reasonably practicable' (ALARP).

not provide guidance on operational exposures for military purposes, these can be found in STANAG 2084 and 2474 for war scenarios and non-war scenarios respectively.

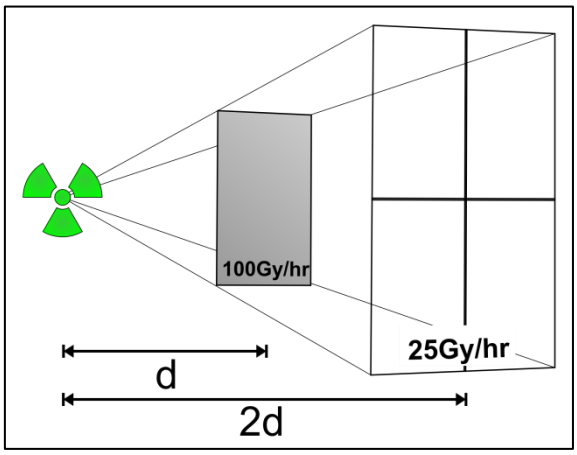
31.3.1. TIME

The absorbed dose is proportional to the time spent near the radiation source. The dose is therefore minimised by reducing exposure time.

Example. A person is 100 metres away from a radiation source and is exposed to a dose rate of 20mGy per hour. After an hour, the person will have received 20mGy absorbed dose. If the exposure time is halved to 30 minutes, the absorbed dose will be 10mGy.

31.3.2. DISTANCE

The dose absorbed is reduced by increasing the distance from the source. The reduction in dose and dose rate follows the inverse square rule. This rule states that the dose will fall inversely by the square of the relative distance i.e. doubling the distance will reduce the dose rate by a quarter ($1/2^2$), tripling the distance will reduce the dose rate by a ninth ($1/3^2$).



Figure–31-1 – Inverse Square Rule.

Example. A person is 100 metres away from a radiation source and is exposed to a dose rate of 20mGy per hour. After an hour, the person will have received 20mGy absorbed dose. If the person was at twice the distance (200 metres) for an hour, the absorbed dose will be 5mGy (one quarter of the dose).

31.3.3. SHIELDING

Shielding may be an effective protection, when operating within a zone where external ionising radiation is a hazard. The principle may be applied directly to the source to neutralise the hazard, as personal protective equipment against contamination or around a potentially exposed population (shelter, hardened infrastructure) to mitigate the external effects. Skin provides adequate protection against alpha as will paper. Aluminium or layered plastic shields against beta. Concrete, steel and lead are the best type of shielding to attenuate gamma, but is not always possible operationally. The protection factor varies depending on the type of shelter and building materials used; a concrete basement under a large building may provide a PF of up to 200. A summary of the various shielding materials is in Figure 31-2. These materials may also be used to help identify an unknown source as either an alpha, beta or gamma source with a non-specific detector.

Example. A person is outside a building 100 metres away from a radiation source and is exposed to a dose rate of 20mGy per hour. After sheltering behind a thick wall at the same distance (providing a protection factor of 2), the dose rate is now 10mGy per hour (1/PF).

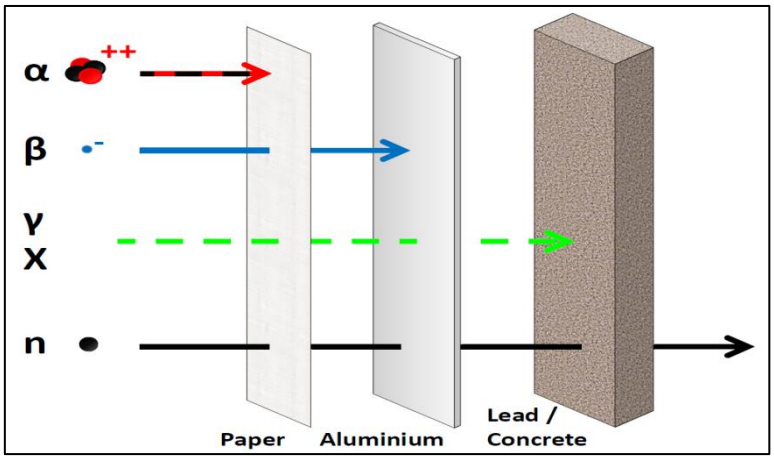


Figure 31-2: Radiation Shielding.

31.3.4. THE MEDICAL APPLICATION OF RADIATION PROTECTION

The scientific principles of radiation protection apply to casualty management also. The principles apply to the direct treatment of casualties especially with internal and wound contamination, and to the safety considerations of medical personnel.

a. *Time*. Any radiological contamination external, internal or wound should be removed as soon as possible to reduce the effects of exposure. However, this should not be to the detriment of the management of life saving conditions (emergency medical treatment). The removal of internal contamination (decorporation) aims to reduce the biological half-life of internalised radioisotopes by increasing elimination and thereby minimising the time an isotope is inside the body.

b. *Distance*. Personnel responding to a radiological incident should be aware of dose rates at critical points within the hot zone. The exclusion zone cordon may be based upon a set dose rate while the inner cordon may use a dose rate of 100µGy per hour. When operating on casualties with radiological shrapnel, the surgeon may consider maintaining a distance until the procedure to remove the fragment is ready to be performed.

c. *Shielding*. Medical personnel should select PPE appropriate to the radiological hazard. Light and disposable PPE may allow for a more rapid and medical dexterous response. Shielding to protect the responder should not prolong any medical procedure as time is also a factor as described above and benefits responder and casualty.

31.3.5. PERSONAL PROTECTIVE EQUIPMENT

1. Protective clothing is used to protect the medical provider from external contamination and any internalisation. The purpose of protective clothing is simply to keep the radioactive material off skin or personal clothing / uniform. Paper coveralls, cloth coveralls and surgical gown with respiratory protection are acceptable forms of protective clothing and maintain medical dexterity. Concerns for heat stress should be taken into account and the number of internal layers reduced as required. Medical personnel should be monitored for contamination

and, if necessary decontaminated following treatment and decontamination of contaminated patients.

2. Standard-issue particulate protective masks or respirators afford excellent protection from inhalation and ingestion of particulate radioactive material as respiratory protection devices are usually designed to filter particulates. However, radon and tritium gas will pass through the filters although short exposures to these nuclides are not usually medically significant. After use, filters should be changed and monitored for any significant radioactivity levels due to potential concentration in the filters.

CHAPTER 32: BIOLOGICAL EFFECTS OF IONISING RADIATION

32.1. EFFECTS OF IONISING RADIATION ON BIOLOGICAL TISSUE

1. The mechanisms for the clinical effects of ionising radiation are at the molecular level. Radiation may interact with DNA either directly or indirectly through the formation of free radicals by ionisation. Damage caused by ionising radiation includes single- or double-strand breaks in DNA. A break in a DNA strand is not compatible with cellular life as the cell cannot properly replicate its DNA and prevents the transferring of identical genetic material to its daughter cells. Breaks in DNA strands must therefore be repaired and mammalian cells have a number of repair systems that can repair radiation damage.

a. *Single-strand DNA breaks*. Single-strand breaks can be repaired by a relatively simple enzymatic mechanism. DNA repair is possible because the genetic information has been preserved in the complementary strand. Repair mechanisms therefore complete the original base sequence by reading from the complementary strand. The break is then closed by enzymes.

b. *Double-strand DNA breaks*. Some double-strand breaks can be repaired to a certain degree. This kind of repair comprises several enzymatic steps. It not only takes place after exposure to radiation but also after replication errors following the normal DNA synthesis required for cell division. During this process, repair errors may occur and either cause the cell to fail to replicate again or function, or to function differently.

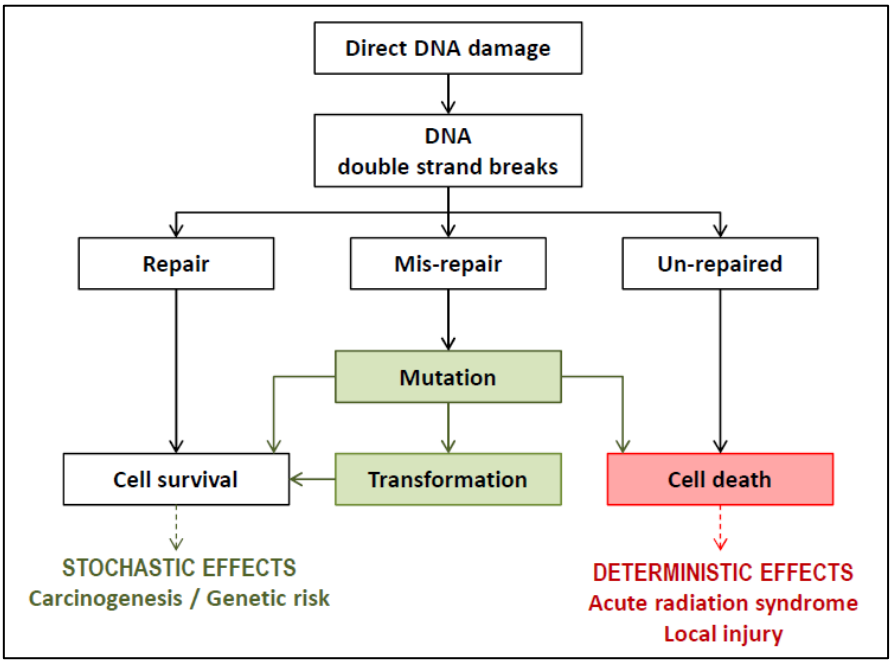
2. DNA damage involving the clustering of lesions and double-strand breaks is associated with high linear-energy transfer (LET) radiation such as alpha particles and some neutron interactions. The damage may remain or be mis-repaired. This form of clustering is now considered the likely source for the formation of chromosomal aberrations exploited for cytogenetic biodosimetry. The impact of this damage and/or mis-repair is summarised in Figure 32-1 and includes:

a. *Cell death*. Cell reproductive death may occur as well as cell dysfunction. The effects are more likely to be seen in tissues with rapidly dividing cells such as those within the gastrointestinal tract, skin and bone marrow; they are therefore said to be *radiosensitive*. Cell death forms the basis for the deterministic effects of acute radiation exposure.

b. *Mutation*. If the cell survives but is mis-repaired, it may pass on changed hereditary information in the DNA to its daughter cells. The long term risk follows a probability (stochastic) effect i.e. the greater or more frequent the radiation exposure, especially to high LET, the greater the risk of a long term consequence. The result of the mutation will depend on the cell type:

(1) *Cancer risk (carcinogenesis)*. If the damage is to a single somatic cell, this can form the basis for malignant transformation of the cell and the development of a malignant disease.

(2) *Genetic risk*. Damage to the DNA of a germ cell can cause mutations in the following generations.



Figure–32-1 – Radiation Effects on Cells.[1]

32.2. DETERMINISTIC EFFECTS

Deterministic (tissue) effects are those that are directly dose related and include acute and delayed effects. They result from nucleic acid damage and from systemic inflammatory reaction of the organism. *The greater the radiation exposure, the greater the magnitude of the effect.* While individual variations may occur due to individual sensitivity, the severity of the effect is predictable based on the dose received. Deterministic effects include:

a. *Acute radiation syndrome (ARS)*. ARS describes the phased chronology and clinical presentation of illness directly attributed to doses of ionising radiation. While it is assumed that whole body irradiation has occurred, in some cases there may be preservation of localised tissue such as bone marrow.

b. *Local radiation injury*. Highly-radioactive material held in close proximity to tissue will produce acute local tissue damage and necrosis. While beta radiation will mainly effect the skin (cutaneous syndrome), gamma radiation may be highly penetrating and damage deep tissue as well as the skin.

c. *Delayed effects*. Tissue fibrosis, chronic immune system suppression, reproductive tissue dysfunction and selected ocular problems including cataract formation are some of the more common (and serious) late-onset deterministic pathologies.

32.3. ACUTE RADIATION SYNDROME

1. ARS is characterised by the body's multi-system response to a high dose radiation exposure. The dose above which the signs of ARS are seen is referred to as the *threshold* (~0.75Gy). ARS has been encountered after the detonation of nuclear weapons, criticality accidents, industrial radiation accidents, radiotherapy patients, in the emergency workers who

[1] Adapted from Prise KM. New advances in radiation biology. Occ Med. 2006; 56: 156-161.

responded to the nuclear disasters, and may be encountered in case of malevolent radiological exposure device (RED).

2. ARS has been broken down into four phases with the third being further sub-divided into sub-syndromes which are dose and time dependent (Figure 32-2). The four phases and sub-syndromes are:

a. An initial or *prodromal phase* occurring during the first few hours after exposure.

b. A *latent phase*, which becomes shorter with increasing dose.

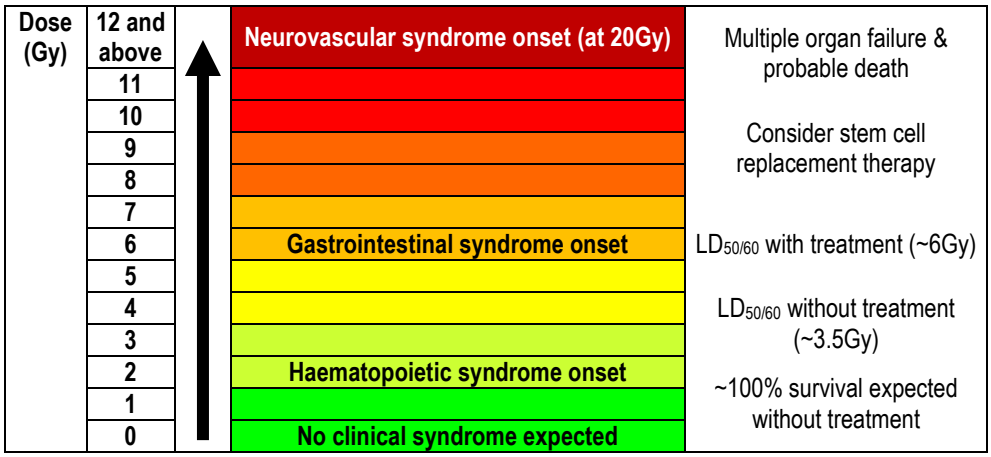
c. The *manifest illness (clinical) phase* including:

(1) Hematopoietic sub-syndrome (ARS-H).

(2) Gastrointestinal sub-syndrome (ARS-G).

(3) Neurovascular sub-syndrome (ARS-N).

d. Recovery or death.



Figure–32-2 – Acute Radiation Effects.

3. The time of onset and degree of the transient incapacitation of the prodrome phase, the duration of the latent period, and the time of onset and severity of the clinical phase and ultimate outcome are all dependent upon total dose, dose rate and individual radiation sensitivity. Commanders and military personnel need to know the basic course of the disease and the necessity for treatment. Medical planners must be aware of the time-phased logistical requirements for triage, treatment areas, MEDEVAC, medical personnel and supplies. The severity of symptoms will increase and the duration of each phase will shorten with increasing acute radiation dose.

4. At higher doses, more systems are involved. In the hematopoietic system, acute effects do not become manifest until after approximately 10 to 15 days. The higher the radiation dose received, the more pronounced and earlier the lymphopenia and thrombocytopenia. The gastrointestinal syndrome will result in high mortality, but a few individuals may respond to therapy. The neurovascular syndrome appears shortly after exposure to lethal dosages and is probably not amenable to medical therapy. The pulmonary sequelae of radiation injury have recently been described following rare criticality accidents where the casualties survived the more common sub-syndromes but received direct irradiation to the thorax causing fatal

pulmonary fibrosis. The cutaneous syndrome is also described as a sub-syndrome in ARS but is more likely to be seen due to local radiation injury.

32.3.1. PRODROMAL PHASE

1. This is a non-specific clinical response to an acute radiation exposure. The initial phase of prodromal symptoms is characterised by the relatively rapid onset of nausea, vomiting, malaise, headache, fever and, in the most severe cases, diarrhoea. The cause for the prodrome is thought to be due to neurotransmitter such as serotonin ($5\text{-}HT_3$), and histamine release. The rapidity of onset and severity of symptoms are associated with severity of the acute exposure, however alone are not diagnostic of ARS (see Table 32-1). Vomiting should not be confused with psychogenic vomiting which results from the stimulation by the sight / odour of blood, mutilation, vomitus or excrement. Symptoms however may inform the triage process as an early but limited discriminator before more accurate methods are available.

2. *Symptomatic management*. Management of casualties during the prodrome is symptomatic and include $5–HT_3$ oral anti-emetics such as granisetron and ondansetron. Anti-emetics may reduce subsequent traumatic injuries and allow self-extraction after irradiation by reducing or preventing the early incapacitating effects. Prophylactic anti-emetics do not change the degree of irradiation and are therefore not radioprotectants (pre-exposure) or mitigators (post-exposure).

Note. Anti-emetics will reduce the reliability of nausea and vomiting as indicators of radiation exposure for triage and multi-parametric biodosimetry.

Table 32-1 – Prodromal Phase Features.[2]

	ARS degree and the approximate dose of acute whole body irradiation (Gy)				
	Mild (1-2Gy)	**Moderate (2-4Gy)**	**Severe (4-6Gy)**	**Very severe (6-8Gy)**	**Lethal (>8Gy)**
Vomiting – Onset	2 h after exposure or later	1-2 h after exposure	Earlier than 1 h after exposure	Earlier than 30 min after exposure	Earlier than 10 min after exposure
% of incidence	10-50	70-90	100	100	100
Diarrhoea	None	None	Mild	Heavy	Heavy
Onset		-	3-8 h	1-3 h	Within minutes or 1 h
% of incidence		-	< 10	> 10	Almost 100
Headache	Slight	Mild	Moderate	Severe	Severe
Onset	-	-	4-24 h	3-4 h	1-2 h
% of incidence	-	-	50	80	80-90
Consciousness	Unaffected	Unaffected	Unaffected	May be altered	Unconsciousness
Onset	-	-	-	-	Seconds/minutes
% of incidence	-	-	-	-	100 (at > 50Gy)
Body temperature	Normal	Increased	Fever	High fever	High fever
Onset	-	1-3 h	1-2 h	< 1 h	< 1 h
% of incidence	-	10-80	80-100	100	100

[2] IAEA. Diagnosis and treatment of radiation injuries. Safety report series No.2. Vienna: IAEA 1998.

32.3.2. LATENT PHASE

1. Following recovery from the prodromal phase, there will be a latent phase during which the exposed individual will be relatively symptom free. The length of this phase varies with the dose and the nature of the later manifest illness phase. The latent phase is longest preceding the bone-marrow depression of the hematopoietic syndrome and may vary between 2 and 6 weeks. It is somewhat shorter prior to the gastrointestinal syndrome, lasting from a few days to a week. It is shortest of all preceding the neurovascular syndrome, lasting only a matter of hours. These times are variable and may be altered by the presence of other disease or injury.

2. Because of prodrome variability, it is not practical to hospitalise all personnel suspected of having radiation exposure during the latent phase. Outpatient (or radiation assessment unit) monitoring should be instituted as an alternative especially if multiple casualties.

3. The latent period is the ideal time to MEDEVAC at risk radiological casualties from the operational theatre as the operational situation permits. It is also the optimal time to perform any primary surgery before any significant coagulopathy or immunosuppression manifests.

4. Hospital care should wait until the onset of any clinical illness or the development of significant bone marrow suppression as indicated by the individual peripheral blood profile. The latter may require reverse barrier nursing, as well as replacement therapy and prophylactic antibiotic, antifungal and antiviral MedCM. Consider cytokine stimulation therapy within the first 24 hours if the early estimated dose is > 3 Gy.

32.3.3. MANIFEST ILLNESS – OVERVIEW (METROPOL)

1. The manifest illness phase presents with the clinical symptoms associated with the cell death of the major organ system exposed (bone marrow (haematopoietic) (H), gastrointestinal tract (G), neurovascular system (N)). ARS is a direct result of exposure to doses of ionising radiation above 0.75Gy. Its sub-syndromes are dose dependent. As described above, the higher the dose, the sooner the onset of symptoms and shorter the latency phase. Casualties who develop the extremely high-dose neurovascular syndrome will succumb to it prior to the development of the lower dose hematopoietic syndrome. A summary of features of each syndrome and the doses at which they are expected to be seen in young healthy adults exposed to short, high-dose single exposures is described below. These features also provide criteria for dose assessment and guide medical management using the *Medical Treatment Protocol* (METROPOL).

2. For the purpose of radiological injury assessment, the METROPOL system, using biodosimetry, uses the symptoms and signs that make up each of the four ARS sub-syndromes as well as acute local radiation (cutaneous) injury to apply a Response Category (RC) to each of them. This gives a Neurovascular (N), Gastrointestinal (G), Hematopoietic (H) and Cutaneous (C)[3], grading system with RCs of 1 to 4 for each sub-syndrome with the highest score giving the overall ARS or local radiation injury severity.

[3] The cutaneous sub-syndrome (C) is used as part of the ARS assessment as one of four sub-syndrome, but is also a key feature of local radiation injury.

32.3.4. MANIFEST ILLNESS – HEMATOPOIETIC SUB-SYNDROME (ARS-H)

1. Patients who have received doses of radiation in the potentially low to mid-lethal range (2 to 6 Gy) will have bone marrow depression with cessation of blood-cell production leading to pancytopenia (Figure 32-3). The hematopoietic clinical consequences are due to:

a. Haemorrhage.

b. Immunosuppression, followed by infection.

c. Anaemia.

2. Changes within the peripheral blood profile will occur as early as 12 hours post irradiation. The exact time sequence of the depression of various circulating cell lines will vary. Nucleated lymphocytes will be depressed most rapidly and enucleated erythrocytes least rapidly. Other leukocytes and thrombocytes will be depressed but less rapidly than lymphocytes potentially following a brief surge in the case of neutrophils. The time of onset of the cell production depression in the marrow will vary considerably. The onset of the clinical effects range from 10 days to 6 to 8 weeks after exposure and average 2 to 3 weeks. However, even lethal cases of bone-marrow depression may not occur until 6 weeks after exposure. Death occurs from overwhelming infection and/or haemorrhage unless bone marrow function is restored either by cytokine (stimulation) therapy, or replacement therapy.

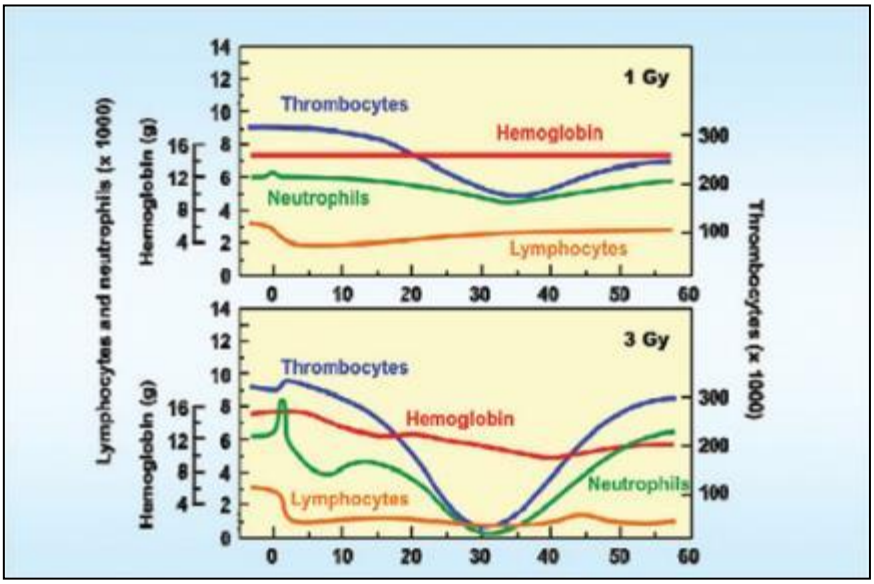


Figure 32-2 – Haematopoietic Effects of Ionising Radiation.

3. The presence of other injuries will increase the severity and accelerate the time of maximum bone-marrow depression. If the exposures leading to bone-marrow depression are multiple, the time of onset of depression will be very difficult to estimate. The clinical picture, however, once bone marrow depression is present, will be identical regardless of the sequence of exposure.

4. Important hematopoietic cell line consequences include:

a. *Lymphocytes*. Lymphocytes are the most radiosensitive aspect of the hematopoietic system. The fall in circulating lymphocytes can be used as a crude biodosimetry tool to

estimate the effective radiation dose received. The steeper the curve, the higher the dosage and the more severe the injury will be. For example, if a casualty's lymphocyte count drops to 25% of baseline at 24 hours, then a severe injury has occurred, and bone marrow resuscitative measures should be instituted as soon as is practical including cytokine therapy.

b. *Thrombocyte (platelets)*. The thrombopoietic cell-renewal system is responsible for the production of platelets. Platelets have a lifespan of 8 to 9 days and are produced by megakaryocytes in the bone marrow. Both platelets and mature megakaryocytes are relatively radioresistant. However, platelet renewal is influenced by any mechanism that alters megakaryocyte maturation. Thrombocytopenia is reached by 3 to 4 weeks after mid lethal-range doses due to the killing of radiosensitive stem cells and immature megakaryocytes precursors. Regeneration of thrombocytopoiesis after sub-lethal irradiation normally lags behind other bone marrow cell lines. Elevated platelet numbers, overshooting the pre-irradiation level, have been observed during the regenerative phase in human nuclear accident victims. The mechanism for the rapid recovery of platelet numbers may be explained by the surviving and regenerating stem cell pool responding to a feedback stimulus due to the acute thrombocytopenia.

c. *Granuloctyes (including neutrophils)*. Following a significant radiation dose, there will be a transient stress-type rise in circulating neutrophils. This transient rise compared to the lymphocyte fall can be exploited as a ratio as an early diagnostic marker for acute radiation exposure. At sub-lethal levels, the neutrophil count returns to normal. At higher doses, there is a fall in neutrophils due to a failure of the radiosensitive myeloblasts. The fall in neutrophils leads to a significant risk of bacterial (as well as other opportunistic) infections (10-30 days). A severe neutropenia (<0.5 x 10^9 cells/L) is a medical emergency with high risk of neutropenic sepsis especially with concurrent gastrointestinal syndrome and associated bacteraemia.

d. *Erythrocytes (red blood cells)*. Erythrocytes are radioresistant as they lack a nucleus. They have a biological half-life of 120 day and are the last cell line to be depressed. This is due to failure of erythropoiesis due to the radiosensitive precursors. Anaemia is more likely in the presence of haemorrhage but can be managed with replacement (transfusion) therapy.

Table 32-2 – Hematopoietic (ARS-H) Sub-syndrome Response Categories.

CRITERIA (Reference)	Degree 1 (H1)	Degree 2 (H2)	Degree 3 (H3)	Degree 4 (H4)
Lymphocyte changes (1.4-3.5 x 10^9 cells/L) at 1-2 days	≥ 1.5	1 - 1.5	0.5 – 1	< 0.5
Granulocyte changes (4-9 x 10^9 cells/L) at 3-7 days	Day 1-2 sees a transient rise in neutrophil count. The neutrophil:lymphocyte ratio can be used for early estimated dose calculation			
	≥ 2	1 - 2	0.5 – 1	< 0.5
Thrombocytes changes (140-400 x 10^9 cells/L) at 3-7 days	≥ 100	> 50 - 100	> 20 – 50	< 20
Blood loss	Petechiae, easy bruising, normal Hb level	Mild blood loss with < 10% decrease in Hb level	Gross blood loss with 10-20% decrease in Hb level	Spontaneous blood loss with > 20% decrease in Hb level

Infection	Local, no antibiotic therapy required	Local; only local antibiotic therapy required	Systemic; oral antibiotic treatment sufficient	Sepsis; iv antibiotics necessary

5. *Clinical assessment*. Clinical features suggesting bone marrow suppression include:

a. *Haemorrhage*. Patients will show increased evidence of haemorrhagic disease. This may be petechiae, purpura, bruising or overt bleeding.

b. *Infection*. Conventional signs of infection may be present including fever. Some atypical or opportunistic infections may be present including fungal infections. In some cases, there may be little clinical response because of a depressed inflammatory response (*neutropenic sepsis*).

c. *Anaemia*. While this is a late sign, the patient may present with pallor.

6. *Clinical investigation*. The most useful clinical investigation to evaluate bone marrow depression is the peripheral blood count.[4] A pancytopenia with severe depression of lymphocytes, granulocytes and thrombocytes (with erythrocyte sparing) is strongly indicative of radiation-induced bone marrow depression. Bone marrow-based diagnostic tests are unlikely under field conditions and will add little extra useful information.

32.3.5. MANIFEST IILLNESS - GASTROINTESTINAL SUB-SYNDROME (ARS-G)

1. The acute radiation doses that result in the gastrointestinal syndrome are higher than those that cause hematopoietic syndrome. The acute dose that will cause this syndrome is greater than 6Gy. Gastrointestinal syndrome has a very serious prognosis because it is almost always accompanied by bone-marrow suppression. The onset of the clinical phase of the gastrointestinal syndrome occurs earlier than that of the hematopoietic syndrome. After a short latent period of a few days to a week, the characteristic severe fluid losses, haemorrhage and diarrhoea begin. The basis for this syndrome is damage to the rapid turnover and radiosensitive epithelial cells leading to severe loss of intestinal mucosa and injury to the submucosa vasculature. Mucosal damage allows translocation of gut bacteria causing a bacteraemia, the effects are compounded by immunosuppression. Patients with gastrointestinal syndrome may be salvageable but are at high risk of infection, fluid loss and haemorrhage.

2. *Clinical assessment*. There are no specific symptoms or signs of radiation-induced gastrointestinal damage. A challenge for diagnosis may arise discriminating patients with sub-lethal radiation-induced hematopoietic depression and concurrent gastrointestinal infection from patients with lethal ARS and gastrointestinal sub-syndrome.

3. *Clinical investigation*. A peripheral blood count will show an early onset of a severe pancytopenia due to bone marrow depression. Microscopic examination of any diarrhoea may reveal inflammatory cells, which is suggestive of dysentery. Radiation enteropathy is not likely to result in an inflammatory response. Plasma citrulline is recommended as a biomarker for radiation-induced small intestinal damage.

[4] Often referred to as a full blood count (FBC) or a complete blood count (CBC) with white cell count differential.

Table 32-3 – Gastrointestinal (ARS-G) Sub-syndrome Response Categories.

CRITERIA	Degree 1 (G1)	Degree 2 (G2)	Degree 3 (G3)	Degree 4 (G4)
Diarrhoea:				
Frequency; stools/day	2-3	4-6	7-9	≥ 10; refractory
Consistency	Bulky	Loose	Very loose	Watery
Bloody stools / melena	Occult	Intermittent	Persistent	Persistent; large amount
Abdominal cramps / pain	Minimal	Moderate	Intense	Excruciating

32.3.6. MANIFEST ILLNESS – NEUROVASCULAR SUB-SYNDROME (ARS-N)

This syndrome is associated with very-high acute doses of radiation greater than 20Gy. The latent period is very short, varying from several hours to 1-3 days. The subsequent clinical picture is a steadily deteriorating state of consciousness with eventual coma and death due to acute microvascular changes and membrane permeability leading to cerebral oedema, hypotension, and cerebrovascular insufficiency. Hypotension may be seen at lower doses. Convulsions may or may not occur. There may be little or no indication of increased intracranial pressure. Because of the very high doses of radiation required to cause this syndrome, personnel close enough in a nuclear detonation to get this dose would generally be located well within 100% lethality radius for blast and thermal effects. The syndrome could be seen potentially in air burst detonation or if shielded against blast. Radiation doses in the >10Gy range could also result from a reactor or weapons facility, or fuel reprocessing plant due to a criticality event especially if localised to the head region.

Table 32-4 – Neurovascular (ARS-N) Sub-syndrome Response Categories.

CRITERIA	Degree 1 (N1)	Degree 2 (N2)	Degree 3 (N3)	Degree 4 (N4)
Nausea	Mild	Moderate	Intense	Excruciating
Vomiting	Occasional (one per day)	Intermittent (2-5 times per day)	Persistent (6-10 times per day)	Refractory (> 10 times per day)
Headache	Mild	Moderate	Intense	Excruciating
Anorexia	Able to eat and drink	Intake decreased	Intake minimal	Parenteral nutrition
Fever	< 38°C	38-40°C	> 40°C for < 24 h	> 40°C for > 24 h
Hypotension	Heart rate > 100/m; BP > 100/70mmHg	BP < 100/70mmHg	BP < 90/60mmHg	BP > 80/?mmHg; persistent
Neurological deficits	Barely detectable	Easily detectable	Prominent	Life threatening. Loss of consciousness
Cognitive deficits	Minor loss	Moderate loss	Major impairment	Complete impairment
Fatigue / weakness	Able to work	Interferes with work or normal activity	Needs assistance with self-care	Prevents daily activities

32.3.7. MANIFEST ILLNESS – CUTANEOUS SUB-SYNDROME (ARS-C)

The skin symptoms and signs form the cutaneous sub-syndrome to ARS following whole body irradiation. Because of the overlap with local radiation injury, this syndrome is described below in the next section.

32.3.8. RECOVERY OR DEATH

The primary cause of death from acute radiation syndrome is most likely to be infection or haemorrhage. The neurovascular syndrome is rapidly fatal even in isolation before other syndromes are seen. Survival of hematopoietic syndrome will be based on early diagnosis,

replacement therapy, infection prevention and bone marrow restoration. Below 10Gy, death is not inevitable and for low casualty numbers all patients should be assessed and treated early, and transferred to a specialist unit as soon as possible. The early detection of biomarkers for radiation-induced multi-organ dysfunction syndrome (RI-MODS) or multi-organ failure (MOF) may improve recovery with appropriate Rad MedCM.

32.4. CUTANEOUS SYNDROME AND LOCAL RADIATION INJURY

Damage to the skin can present in three ways:

a. Cutaneous syndrome, following whole body irradiation and as part of ARS.

b. Local radiation injury due to close contact high dose gamma radiation exposure.

c. Beta burns.

32.4.1. ARS CUTANEOUS SUB-SYNDROME (ARS-C)

The cutaneous sub-syndrome to ARS is usually seen at doses associated with both hematopoietic and gastrointestinal syndromes. Although doses may also be high enough to cause neurovascular syndrome but death is likely to have occurred before significant skin features are seen. The investigation of the syndrome follows the same general requirements as for ARS but may also include assessment for any local features depending on the underlying cause for the radiation exposure. For ARS, the RC criteria can be used as shown in Table 32-5. Local irradiation is classified differently although may still be associated with ARS.

Table 32-5 – Cutaneous (ARS-C) Sub-syndrome Response Categories.

CRITERIA	Degree 1 (C1)	Degree 2 (C2)	Degree 3 (C3)	Degree 4 (C4)
Erythema	Minimal, transient	Moderate (< 10% body surface area)	Marked (10-40% body surface area)	Severe (> 40% body surface area)
Pruritus (itching)	Sensation of itching	Slight or intermittent pain	Moderate and persistent pain	Severe and persistent pain
Oedema	Persistent, asymptomatic	Symptomatic, tension	Secondary dysfunction	Total dysfunction
Blistering	Rare, sterile fluid	Rare, haemorrhage	Bullae, sterile fluid	Bullae, haemorrhage
Desquamation	Absent	Patchy dry	Patchy moist	Confluent moist
Ulcer and necrosis	Epidermal only	Dermal	Subcutaneous	Muscle / bone involvement
Hair loss	Thinning, not striking	Patch, visible	Complete, reversible	Complete, irreversible
Onycholysis	Absent	Partial	Partial	Complete

32.4.2. LOCAL RADIATION INJURY

1. Local tissue irradiation occurs when highly radioactive (usually gamma emitting) material such as an industrial radiography source is placed in close proximity to tissue including skin. As radiation intensity decreases in proportion to the square of the distance, the tissue immediately adjacent to the source receives a huge dose. Radiological shrapnel may be associated with deep tissue radiation damage and spare the skin; in this situation even beta radiation may still have a significant tissue damaging effect. For external exposures, the total body dosage may be only 2Gy, but the local skin surface dose can be 10^2-10^3Gy. Initial skin changes will be similar to those of the acute cutaneous radiation syndrome and the dose,

appearance and timeframe is shown in Table 32-6. Deep tissue effects may either be due to the direct damaging effect of the radiation causing cell death, or may be secondary to inflammatory changes, vascular occlusion and avascular necrosis, and infection.

Table 32–6 - Local Radiation Injury Effects and Thresholds.

Dose (Gy)	Sign	Time post exposure	Comments
3	Epilation	2-3 weeks	Simple hair loss
6	Erythema	Minutes (transient)	Known as primary erythema
		2-3 weeks	Known as secondary erythema and may be associated with cutaneous syndrome
10-15	Dry desquamation	3 weeks	Dry flaky skin due to damage to the germinal epidermal layer
15-25	Wet desquamation	2-3 weeks	Partial thickness radiation burn with oedema, blistering and moist dermal surface
>25	Radionecrosis, deep ulceration	Weeks	Overt radionecrosis, ulceration, fibrinoid vascular damage and avascular necrosis

2. Following a close contact skin exposure to penetrating gamma radiation, deep tissues will show signs of damage and necrosis over time. Development of deep-base ulcers with marked erythema at the margins is common. Granulation tissue develops poorly, and months will be required for healing. Gradual expansion of the surface ulcer will occur, as the lesser damaged tissues will ulcerate later. Skin flaps will often ulcerate at the margins of attachment. Deep tissues respond in a similar fashion if the source is placed in their immediate proximity. Radiotherapy literature is the best source of information concerning injury to specific tissues and anatomic structures.

3. The exact estimation of local radiation injury is difficult and is often based on dose reconstruction using health physics data and patient or witness recollection of distance and timings and can be inaccurate. A modification of the CDC (I, II and III) grading in Table 32-7 uses both estimated or confirmed cutaneous dose, and clinical features.

Table 32-7: Local Radiation Injury Grading.

Local radiation injury grade	Dose (Gy)	Possible observed (clinical) effects	Comments
I	2 - 15	Erythema and dry desquamation	Monitor effects and dress wounds
II a	5 - 25	Wet desquamation only	Monitor effects and dress wounds
II b	5 - 40	Wet desquamation ± deep tissue damage*	Consider debridement of wounds
III	> 40	* Overt radionecrosis, ulceration, fibrinoid vascular damage and avascular necrosis	

32.4.3. BETA BURNS

Beta burns are shallow radiation burns. However, they are notable as gross skin contamination with beta-emitting isotopes may cause harm by both irradiation and large surface area loss of skin integrity. Beta burns are a significant feature following skin contamination with fission products either in fallout or as seen in firefighters responding to the Chernobyl nuclear power reactor accident.

32.4.4. KELOID SCARRING

This is a form of severe hypertrophic scarring and complicates skin healing following any injury but especially burns. Simple nuclear flash burns may be complicated by this type of scarring and require reconstructive surgery with a significant risk of reoccurrence.

32.5. CATARACT FORMATION

A late effect of eye irradiation is cataract formation. It may begin anywhere from 6 months to several years after exposure. While all types of ionising radiation may induce cataract formation, neutron irradiation is especially effective in its formation, even at relatively low doses. Cataract formation begins at the posterior pole of the lens and continues until the entire lens has been affected. Growth of the opacity may stop at any point. The rate of growth and the degree of opacity are dependent upon dose as well as the type of radiation. Recent studies support lowering the previous threshold for detectable cataract formation to 0.5Gy for both acute and protracted photon radiation doses. A 50% cataract risk has been estimated at acute doses of ~3Gy. Higher doses tend to increase the degree of opacity and shorten the period of latency.

32.6. EXPOSURE IN UTERO

Exposure to high dose radiation in utero may be associated with spontaneous abortion, deterministic effects in the new-born, increased infant mortality, and microcephaly with associated mental retardation.

32.7. STOCHASTIC EFFECTS OF IONISING RADIATION AND CANCER RISK

1. A stochastic (cancer and heritable disease) effect is a consequence based on statistical probability. Radiation stochastic effects are calculated based on equivalent and effective dose, both measured in sieverts. Equivalent dose takes into account the more damaging effect (radiation weighting factor) of some types of radiation such as alpha (W_R 20) or neutrons. Effective dose takes into account the tissue sensitivity of a specific tissue or organ system (tissue weighting factor (W_T)) as a proportion of the whole body e.g. bowel (W_T 0.12) is twelve times more sensitive than brain (W_T 0.01).

2. Carcinogenesis is the most important long-term sequelae for a radiation dose < 1Sv but there is also the potential for genetic mutations to be passed on to offspring. Unlike deterministic effects it is generally thought that there is no dose threshold for stochastic effects and that any radiation dose however small has a change of causing harm. Stochastic effects are also cumulative and do not have the same benefit of fractionated dose exposure that are seen in high dose exposures and exploited during brachytherapy (radiotherapy).

32.7.1. CARCINOGENESIS AND CANCER RISK

1. Most of the data utilised to construct risk estimates are taken from radiation doses > 1Sv and is then extrapolated down for low-dose probability estimates assuming a linear relationship. Significant direct data is not available for absolute risk determination of doses <50mSv and there remains some debate on whether there is truly a linear relationship at the low dose range due to repair mechanisms and confounding factors.

2. The overall life time risk of a fatal cancer is approximately 25%. In general, the excess risk of cancer is associated with 5% per Sv. Therefore, a 1 Sv dose of radiation increases the

lifetime risk of fatal cancer from 25% to 30% whereas a 0.1 Sv dose increases the risk from 25% to 25.5%.[5]

3. Irradiation of almost any part of the body increases the probability of cancer. The type formed depends on such factors as body-area irradiated, radiation dose, radionuclide, route of exposure and age. Irradiation may either increase the absolute incidence of cancer or accelerate the time of onset of cancer appearance or both. There is a latent period between the exposure and the clinical appearance of the cancer. In the case of the various radiation-induced cancers seen in man, the latency period may be several years but varies with the type of cancer. A leukaemo-genic effect was expected and found among Hiroshima and Nagasaki survivors. The peak incidence occurred 6 years after exposure and was less marked for chronic granulocytic leukaemia than for acute leukaemia. The incidence was inversely related to distance from the hypocentre.

4. Cancer is not a single disease but a complex of diseases comprising both cancers of the blood (leukaemia's) and cancers of solid tissues of both epithelial and mesothelial origins. The radiogenic nature of these specific cancers differs. Bone tumours (osteosarcomas) serve as a good example, as they are prominent late-arising pathologies associated with internally deposited, bone-seeking radionuclides (^{90}Sr) but are rarely associated with the cancers that stem from exposure to external radiation sources (^{60}Co). Thyroid cancer is a particular organ of concern due to the concentration of iodine, including radioiodine, and can be prevented by the used of stable iodine as a blocking agent.

5. Cancer types that are unequivocally inducible by ionising radiation are most lymphohematopoietic cancers and cancers of the lung, mammary tissues, liver, kidney, thyroid, colon, stomach, pancreas, and salivary glands. Cancers with either a low incidence or a low probability of induction include cancers of the larynx, nasal sinuses, parathyroid, nervous tissue, and connective tissue. Cancers that are probably not inducible include the chronic lymphocytic leukaemias, Hodgkin's lymphoma, and cancers of the uterus, cervix, prostate, testis, mesentery, and mesothelium.

6. Increased health risks have been associated with high intakes of internalised radionuclides. These risks are largely long-term in nature and dependent not only on the concentration of nuclide absorbed to specific tissues but also on the distribution, metabolism and elimination of the chemical form the radionuclide is in. Cancers of the lung, liver, and bone are principal organs of concern. However, it is thought that very large doses are generally required to induce these cancers. Other factors may also play a role in how likely a person exposed to radiation is to develop cancer. Age is an important factor with children being more radiosensitive than adults. Additional cancer risk taking such as smoking, diet and further radiation exposure will also increase the risk of cancer.

32.7.2. GENETIC RISK

Genetic abnormalities are also theoretically stochastic, as their occurrence is based on genetic material damage in reproductive DNA. Although expected, no significant genetic effects have been in the next generations of atomic bomb survivors. Congenital effects may also be seen and they include low birth weight, congenital defects and infant deaths. Congenital effects are also seen in other non-radiological conflict populations.

[5] International Committee on Radiation Protection Publication 103: *The 2007 Recommendations of the International Commission on Radiological Protection*.

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CHAPTER 33: DOSE ASSESSMENT AND BIODOSIMETRY

33.1. INTRODUCTION

1. Rapid acute radiation dose assessment is vital to early and appropriate casualty management. In addition, accurate low dose assessment is important for risk communication, reassurance and enabling a return to duty. Initial dose assessment will be an estimation using early indicators. These indicators may in isolation be non-specific and have low statistical significance but as part of a multi-parametric system may be reliable enough to support clinical decisions.

2. Biodosimetry is the use of a biological response as an indicator of absorbed dose. Biodosimetry is not intended to replace other methods of dose assessment, such as early health physics dose estimation and formal dose reconstruction. Dose assessment is often based on a combination of physical dosimetry (personal and area dosimetry), dose estimation based on location and biodosimetry.

3. A number of dose assessment tools are available and the QR link provides an example of biodosimetry record sheets (☑) (courtesy of AFRRI).

Note: Avoidance and real-time monitoring remains the most effective tool to minimising radiation exposure while biodosimetry is retrospective.

33.2. ACUTE RADIATION DOSE ASSESSMENT

Dose assessment is based on:

a. Personal and area dosimetry (see Chapter 31).

b. Presence of prodromal symptoms (see Chapter 32).

c. Biodosimetry methods.

d. Early-response multi-parametric biodosimetry.

e. Dose reconstruction.

f. Dose estimation due to internal contamination.

33.3. BIODOSIMETRY (BIOASSAY) METHODS

There are currently four methods of biodosimetry:

a. Leucocyte (white cell) profile.

b. Molecular biomarkers in body fluids and tissues.

c. Cytogenetic biodosimetry (not available as a deployed capability).

d. Electron paramagnetic resonance (EPR) (not available as a deployed capability).

33.3.1. LEUCOCYTE (WHITE CELL) PROFILE

The early fall in lymphocyte count (L) is a significant parameter for early dose estimation in the first 24 hours. This decay rate for the fall in lymphocytes is a function of the radiation dose and is classically shown in the Andrews curve (Figure 33-1 & Table 33-1). During this period there is also a transient dose-related rise in neutrophil count (N). These two parameters either in isolation or as a N:L ratio are useful indications for a predicted dose of > 3Gy and triaging for early cytokine therapy and subsequent infection prophylaxis. Because of the early neutrophil effect, absolute leucocyte count (ANC) is not useful in the first few days following an incident. Haematological and biomarker that may be useful are shown in Figure 33-2.

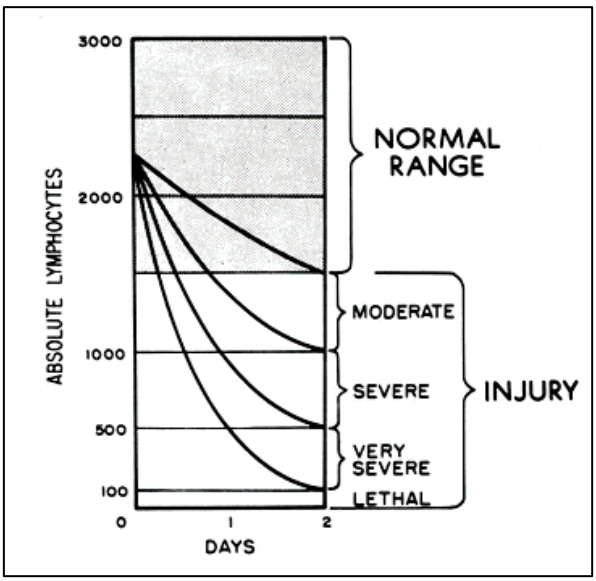

Figure 33-1: Fall in Lymphocyte Count and Dose Severity.[1]

33.3.2. MOLECULAR BIOMARKERS IN BODY FLUIDS AND TISSUES

1. Molecular biomarkers represent underlying changes to human physiology and biochemistry following exposure to ionising radiation. Causes for these changes include physical damage (e.g. cell lysis and the release of intracellular proteins into the circulation, oxidation by-products or DNA breakage), underlying changes in biochemistry (e.g. the presence of new metabolites or changes in levels of key gene products), and indicators of organ injury and/or changes in cellular composition of tissues. Markers may also include the presence or increase in DNA and microRNA, and other repair mechanisms and enzymes and be as diverse as proteins and small molecule metabolites.

2. Within minutes to hours after exposure to ionising radiation, proteins are modified and activated. Changes occur in the gene expression profiles involving a broad variety of cell-process pathways. There are presently about 90 known proteins that show changes after exposure to ionising radiation. Some of these changes are dose-dependent.

3. Multi-parameter dose estimation may be able to use deployed assays that are in general medical use already, including:

[1] Moderate (3.1 Gy), severe (4.4 Gy), very severe (5.6 Gy) and lethal (7.1 Gy).

a. Differential white cell count, as described above.

b. Amylase (specifically salivary amylase).

c. C-reactive protein (CRP).

d. Citrulline, as a marker for gastrointestinal sub-syndrome.

4. Haematological and biomarker that may be useful are shown in Figure 33-2.

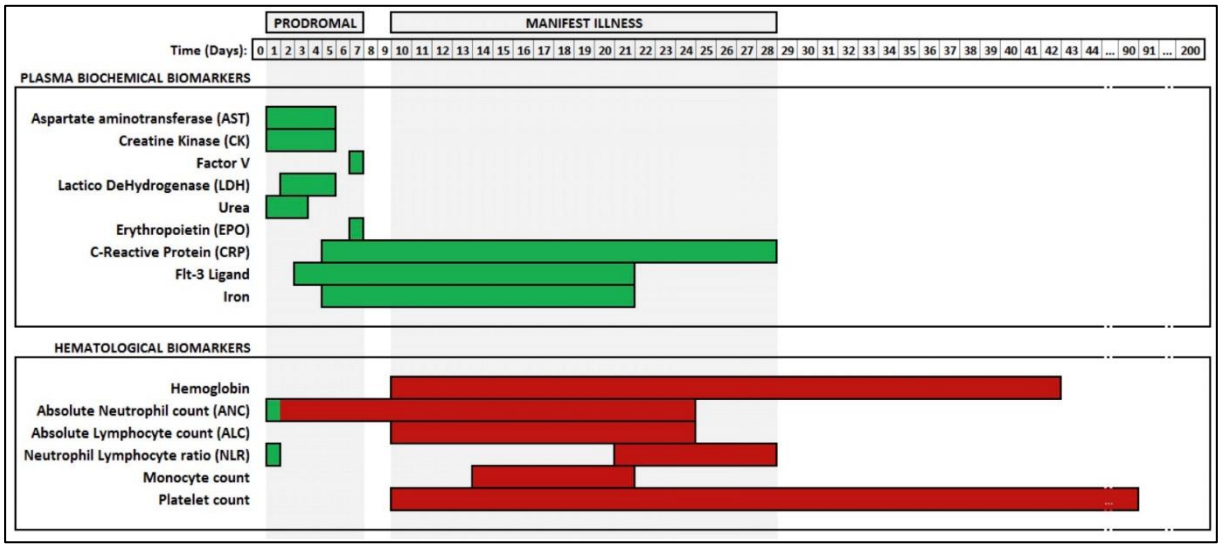

Figure 33-2 – Potential Haematological and Molecular Biomarker Indicators of ARS.[2]

33.3.3. CYTOGENETIC BIODOSIMETERY

1. Cytogenetic biodosimetry is considered the gold standard for the determination of whole-body radiation dose. Cytogenetic biodosimetry has been used for decades to estimate dose on the basis of radiation-induced chromosome aberrations in dividing lymphocytes. The gold standard is the metaphase-spread dicentric chromosome aberration assay (Figure 33-3). The test is normally performed on a full (complete) blood count sample taken at 24 hours. On deployments this is likely to require Reach Back support and may include international collaboration.

2. This assay is mainly applicable to recent whole-body acute radiation exposures. Due to the low background level of dicentric chromosomes in lymphocytes the assay's sensitivity is comparatively high, with a threshold whole-body dose of 0.1 to 0.2Gy (based on analysis of 1000 cells). The assay shows strong dose dependence up to 5 Gy for acute photon exposures. This assay is generally accepted as the most specific and sensitive method (> 0.2Gy) for determining doses from recent exposures to ionising radiation within days to about six months.

3. The reproducibility, relative specificity of radiation-induced dicentric aberrations, and its sensitivity to doses below acute medical significance have allowed the assay to become the gold standard in radiation biodosimetry. Additionally, statistical techniques are available that can determine if the body received a homogeneous (whole body) dose distribution or if the dose was delivered in a non-homogeneous distribution.

[2] STO-TR-HFM-222.

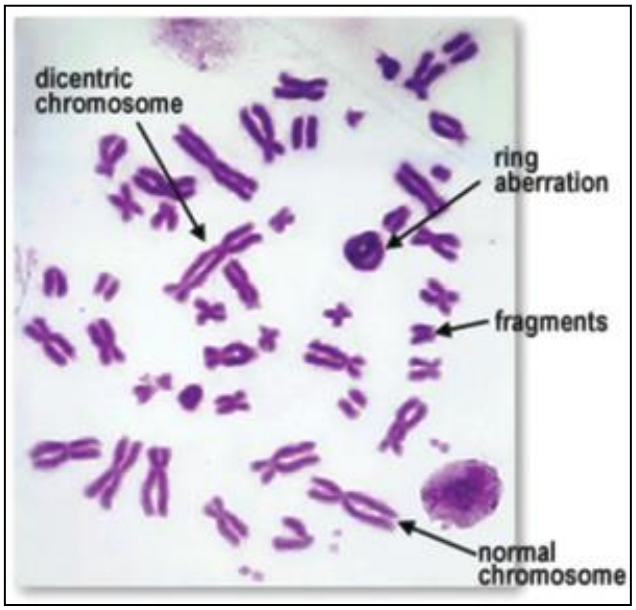

Figure 33-3 – Chromosomal Aberrations in the Metaphase Stage of Lymphocyte Mitosis.

4. The usefulness of this assay is limited for measuring doses received more than six months ago because of the half-life of lymphocytes. Another disadvantage of the assay is the time it takes to process a large number of samples. For this reason, international networks have been established to support a surge capacity for this diagnostic capability. More rapid assays for larger doses analysing less cells, as well as automation are being assessed.

5. Other methods currently used in cytogenetic dosimetry include the translocation, premature chromosome condensation (PCC), and the cytokinesis-block micronucleus (CBMN) assays (Table 33-1), which have applications to various radiation exposure scenarios.

Table 33-1 – Cytogenetic Chromosome Aberration Assays.

	Dicentric, including rings	Translocation	PCC	CBMN
Photon equivalent, acute dose (Gy) range for whole-body exposure	0.1-5	0.25-4	0.2-20	0.3-5
Partial-body exposure assessment	Yes	No	Yes	No
Typical exposure applications	Low level; acute, protracted; prior exposures (<6 months)	Protracted, prior exposures (>6 months)	Acute (including high dose) exposures	Acute

33.3.4. ELECTRON PARAMAGNETIC RESONANCE

1. Human exposure to ionising radiation results in radiation-induced free radical changes that can be measured and, depending on the absorbed dose, quantified. The use of EPR for biodosimetry is based on the capability of the technique to provide specific and sensitive measurement of unpaired electrons in solid tissue which are created in proportion to the absorbed dose.

2. The lifetimes of these electrons are very short (nanoseconds) in aqueous systems, such as most biological tissues, but can be extremely stable in non-aqueous media including teeth, bone, fingernails, and hair. EPR has been used for in vitro analyses of exfoliated teeth to measure doses in populations form Japan and the former Soviet Union. There is a practical problem of signal preservation linked to nail sampling.

33.4. EARLY-RESPONSE MULTI-PARAMETER BIODOSIMETRY

1. No single bioassay is sufficiently robust to address all potential radiation scenarios, including management of mass casualties and the diagnosis for early medical treatment. Recommendations for the use by first responders and first receivers involve a prioritised multiple-assay biodosimetric-based strategy. Multi-parameter triage involving time to emesis, lymphocyte kinetics, and other biodosimetry and biochemical indices as the current best early assessment of a victim's absorbed dose. Rapid dose assessment by haematological biodosimetry, using triage scoring, and injury assessment based on clinical symptoms and signs are critical to provide useful diagnosis to inform medical management decisions. An early estimated dose at 24 hours is recommended to support early cytokine therapy.

2. Consensus biodosimetric guidelines currently include the measurement of:

   a. Signs and symptoms (vomiting, diarrhoea and onset).

   b. Haematology (fall in lymphocyte count and neutrophil: lymphocyte ratio).

   c. CRP.

   d. Serum amylase.

   e. Citrulline (for gastrointestinal ARS sub-syndrome).

   f. Cytogenetics.

   g. Electron paramagnetic resonance.

3. Using multiple parameter biodosimetry (see Table 33-2), it is evident that vomiting within 1-2 hours with a drop in lymphocyte count by 33 - 55% of baseline values within 24 hours suggests a potentially lethal dose of > 4 Gy. Vomiting within 4 hours with a noticeable fall in lymphocyte count and rise in amylase suggest may be a predictor for hematopoietic syndrome (≥ 3Gy) and an indication for cytokine therapy.

33.5. DOSE RECONSTRUCTION

1. Following a radiation incident with known parameters, it may be possible to reconstruct the event and therefore predict the absorbed dose to those persons present. Parameters include quantity of material, radioactivity, yield or a single dosimetry reading at a known distance.

2. The dose calculation will depend on the type of exposure:

   a. *Continuous high (gamma) dose rate exposure*. The calculation will include the estimated (or known) source dose rate with a known duration and distance, and potential shielding.

b. *Prompt gamma / neutron radiation*. This may be due to a criticality event or nuclear detonation. The calculation uses the estimated gamma and neutron initial radiation dose with a known distance, and potential shielding. Following a criticality event, neutron activated radioisotopes such as ^{24}Na and ^{55}Fe provide neutron dose information and can be used to calculate concurrent gamma dose.

Table 33-2 – Multiple Parameter Biodosimetry for Rapid (24 hour) Dose Estimation.[3]

Dose (Gy)	Vomiting		% of absolute lymphocyte count (at 24 h)	Relative increase in serum amylase (at 24 h)	Number of dicentrics		Full chart is shown via link below
	%	Onset (h)			per 50 metaphases	Per 1000 metaphases	
0	-	-	100	1	0.05 - 0.1	1 – 2	
1	19	-	88	2	4	88	
2	35	4.6	78	4	12	234	
3	54	2.6	69	6	22	439	
4	72	1.7	60	10	35	703	
5	86	1.3	53	13	51	1034	
6	94	1.0	47	15			
7	98	0.8	41	16.5			
8	99	0.7	36	17.5			
> 8	100	< 0.7	< 36	> 17.5			

33.6. DOSE ESTIMATION FROM INTERNAL CONTAMINATION

1. Total dose following an internal radiation exposure includes the long term effective dose as irradiation continues while the contamination is still present in the body. The total dose over the calculated period of 50 years for adults is called the *committed effective dose*. The calculation takes into account the quantity of the radionuclide, type(s) of radiation emitted (and radiation weighting factor) and the sum of the effective dose to each tissue / organ most likely to be exposed based on tissue weighting factor.

2. Estimation of internal contamination and therefore potential committed effective dose can be based on:

a. Nasal swab contamination counts.

b. Urinalysis, including 24 hour collection.

c. Stool samples.

d. Whole body monitoring.

[3] Adapted from STO-TR-HFM-99 and Waselenko *et al*. Strategic National Stockpile Radiation Working Group (2004). Medical Management of the Acute Radiation Syndrome: Recommendations of the Strategic National Stockpile Working Group. *Annals of Internal Medicine*, 140(12): 1037-1051, 2004.

33.7. PRE-HOSPITAL MEDICAL REPORTING FOR BIODOSIMETRY

As the medical response to a CBRN incident is a continuum of care, it is vital that information about radiological casualties are reported and recorded. Initial data to support dosimetry, casualty and inform casualty management decision making includes:

a. Exact location.

b. Personal dosimetry readings.

c. Any site dosimetry and location in relation to the casualty.

d. Any radioisotope(s) identified.

e. Any personal protective equipment worn.

f. Risk / presence of external contamination and any decontamination.

g. Risk / presence of wound contamination and any decontamination.

h. Risk / presence of internal contamination and any sampling.

i. Onset and severity of vomiting.

j. Any anti-emetic given and when.

k. Onset and severity of diarrhoea.

l. Onset and severity of headache.

m. Onset and severity of rash, or bruising (if late signs).

n. Any MedCM use including iodine blocking therapy (see Chapter 35).

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CHAPTER 34: MEDICAL ASPECTS OF A NUCLEAR INCIDENT

34.1. INTRODUCTION

1. In order to understand the medical implications of a nuclear event, it is essential to understand how it differs from conventional high-explosive incidents and radiological incidents involving one radioisotope. Nuclear events include:

   a. Nuclear warfare with single or multiple strategic warheads.

   b. Improvised nuclear device or tactical yield weapon use.

   c. Nuclear power reactor accident or attack.

   d. Nuclear weapon accident.

   e. Criticality event.

2. A nuclear weapon produces a combination of effects including:

   a. Flash.

   b. Blast.

   c. Thermal.

   d. Ionising radiation.

   e. Electromagnetic pulse.

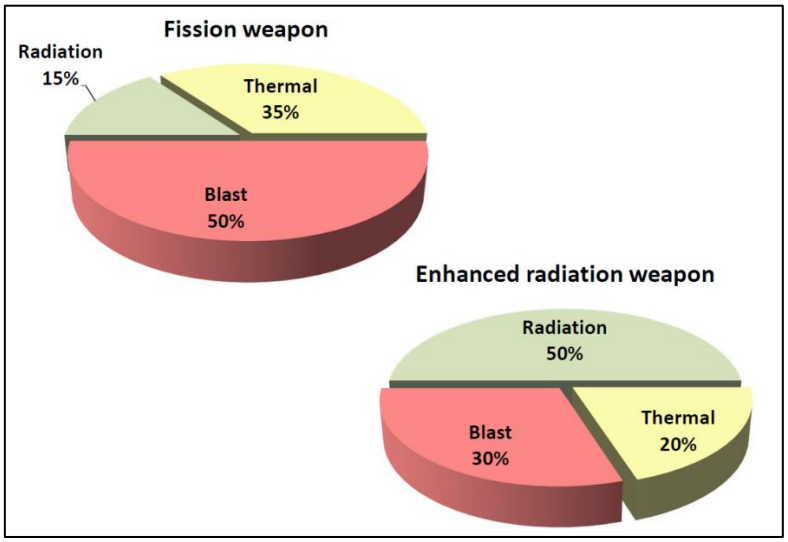

Figure 34-1 – Weapon Energy Distribution.

3. The size and proportion of the effects depends on the weapon design. Fission (atomic) weapons are based purely upon fissile material including ^{235}U and ^{239}Pu, while a fusion (thermonuclear) weapon or hydrogen (H) bomb combines nuclear fission with nuclear fusion for a greater yield. A variation of the latter is an enhanced radiation weapon (ERW) or neutron bomb which delivers proportionally greater nuclear radiation as neutrons (see Figure 34-1).

The effects generated by a detonation result in biological effects greatest at the hypocentre or 'ground zero', and declining progressively with distance (Figure 34-2). The effectiveness of each component as a proportion of the energy release from a nuclear detonation vary with distance and weapon yield measured in kilotons (kT) of trinitrotoluene (TNT) (see Figure 34-2).

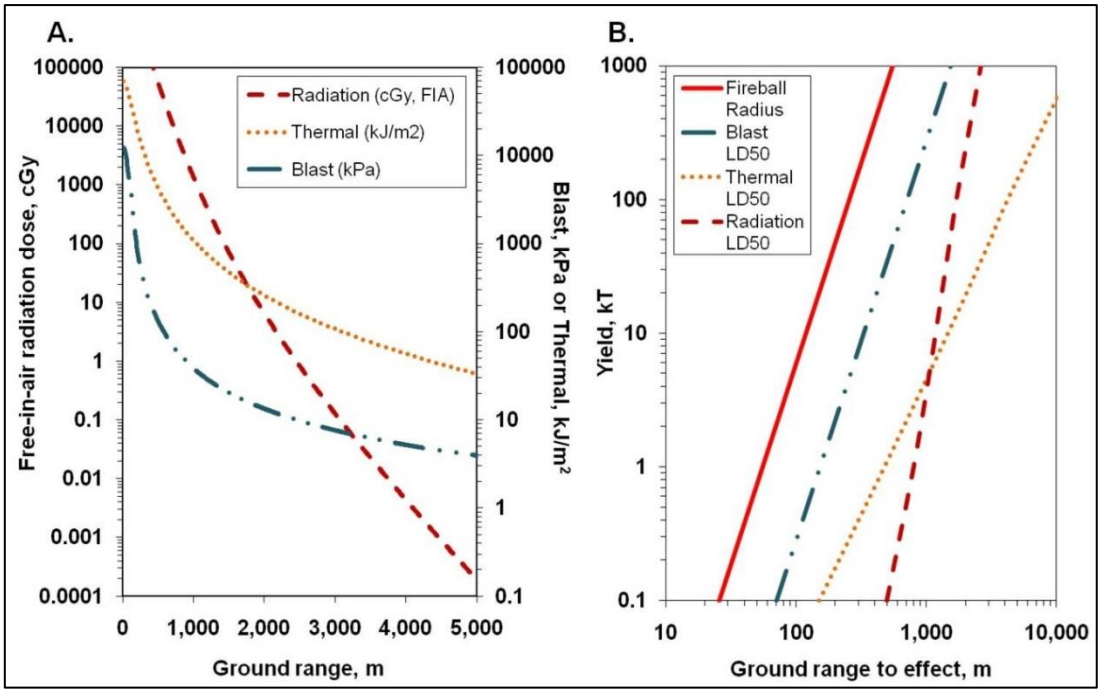

Figure 34-2 – Nuclear Weapon Effects.[1]

Table 34-1 – Comparison of Estimated Nuclear Weapon Effects (the highlighted cells indicate the greatest radii for LD50 effects)**.**

Effect	1kt	10kt	100kt	1000kt (1Mt)
Ionising radiation (50% immediate transient ineffectiveness)	600m	950m	1400m	2900m
Ionising radiation (50% untreated lethality, LD_{50})	800m	1100m	1600m	3200m
Blast (50% casualties)	140m	360m	860m	3100m
Thermal radiation (50% casualties, second-degree burns under fatigue uniform)	369m	1100m	3190m	8020m

4. The effect of a nuclear detonation by the four components on the environment also depends on the location of the detonation:

[1] A) Radiation dose, blast, and thermal effects for a 10-kT low-air burst generic fission device without accounting for effects due to buildings or geography from ground zero. B) LD_{50} for fireball, blast, thermal and initial radiation effects alone from ground zero for low-air burst fission devices of various yields. (Source: AMedP-7.5 (formerly AMedP-8) and refer for methodology)

a. Air burst.

b. Ground (surface) burst.

c. Subsurface (underground).

d. Submerged (under water).

5. Table 34-1 provides a summary of the LD50 radii for an air burst detonation in order to compare the effect of increasing yield on the component effects. The highlighted cells show the most significant component of the detonation for a number of yields as a function of the greatest radii of effect from the hypocentre.

34.2. BLAST EFFECTS

1. A large part of the destruction caused by a nuclear detonation is due to blast effects. The blast consists of:

a. Blast (shock) wave.

b. Positive blast wind.

c. Negative blast wind.

2. The effect of the blast component of a detonation depends on the location:

a. *Surface and subsurface bursts*. A sizable portion of the yield is transmitted in the form of ground or water shock waves. In the case of a surface burst on land, a crater is formed at the hypocentre. Its size depends primarily on yield. Relatively little damage beyond a distance of approximately 3 crater radii will occur due to ground shock. Most damage will be due to the accompanying air blast wave. In subsurface bursts, the crater will be formed either by ejection of material as in a shallow detonation or by the collapse of ground into the cavity formed by a deeper detonation. Since the overpressure in a ground shock wave decreases very rapidly with distance, blast damage will again be confined to a region close to the point of detonation.

b. *Air burst*. Ground shock waves will also be induced as a result of an air burst. For a very large overpressure in the blast wave, the ground shock will penetrate some distance into the ground and may damage underground structures and buried utilities.

c. *Submerged*. Underwater or water surface detonations will cause much greater subsurface shock waves. Because of water's density and relative incompressibility, the shock waves have very high peak overpressures and velocities of propagation. The peak overpressure at a distance of 1 kilometre from a 10kt underwater burst is approximately 6080 kilopascals (60 atmospheres). The peak overpressure in air at the same distance from an airburst is only 111.4 kilopascals (1.1 atmospheres). The resulting surface waves at this distance will be approximately 10 meters in height. The shock front will also travel at approximately 5 times the speed of the blast wave in air. Severe damage to naval vessels may result. Although the major portion of the shock energy is propagated in the water, a significant amount is also transferred through the surface as a typical air blast. This blast wave would probably be the principal source of damage to land targets if the detonation occurred in a coastal area.

34.2.1. BLAST WAVE

1. As a result of very high temperatures and pressures at the point of detonation, hot gaseous weapon residues move outward from the centre of the detonation at very high velocities. Most of this material is contained within a relatively thin, dense shell that acts like a piston pushing against and compressing the surrounding medium. This front transfers energy to the atmosphere and generates a steep-fronted, spherically expanding blast (or shock) wave. This wave initially lags behind the surface of the developing fireball. However, immediately after detonation, the rate of expansion of the fireball decreases and the shock wave catches up with it. The blast wave then moves ahead of the fireball. For a fraction of a second, the dense shock front will obscure the fireball, accounting for the characteristic double peak of light seen with a nuclear detonation. As it expands, the peak pressures of the blast wave diminish and the speed of propagation decreases from the initial supersonic velocity to that of sound. However, upon reflection from the earth's surface, the pressure in the wave will be reinforced by the combination of the incident and the reflected wave (see Figure 34-3).

2. Objects within the path of the blast wave are subjected to severe, sharp increases in atmospheric pressure and to extraordinarily severe transient winds. Most buildings, with the exception of reinforced or blast-resistant structures, will suffer moderate to severe damage when subjected to overpressures of only 35.5 kilopascals (0.35 atmospheres). The range for blast effects increases significantly with the explosive yield of the weapon. In a typical air burst, the values of overpressure and wind velocity described above will prevail at a range of 0.7 kilometres for 1 kiloton yield, 3.2 kilometres for 100 kilotons, and 15.0 kilometres for 10 megatons.

3. When a blast wave strikes the surface of a hard target, such as a building, the reflected wave will reinforce the incident wave, and the face of the building will be subjected to overpressures 2 to 8 times that of the incident wave alone. The severity of this additional stress depends on many factors, including the peak overpressure of the incident blast wave and the angle at which the wave strikes the building. As the shock front advances, it bends or diffracts around the building, and the pressure on the front wall decreases rapidly.

4. A surface burst results in the highest possible overpressures near ground zero. In such a burst, the blast wave is hemispherical in form. Essentially all objects are subjected to a blast wave similar to that in the Mach region described above. A subsurface burst produces the least air blast. Most of the energy is dissipated in the formation of a crater and the production of a ground shock wave.

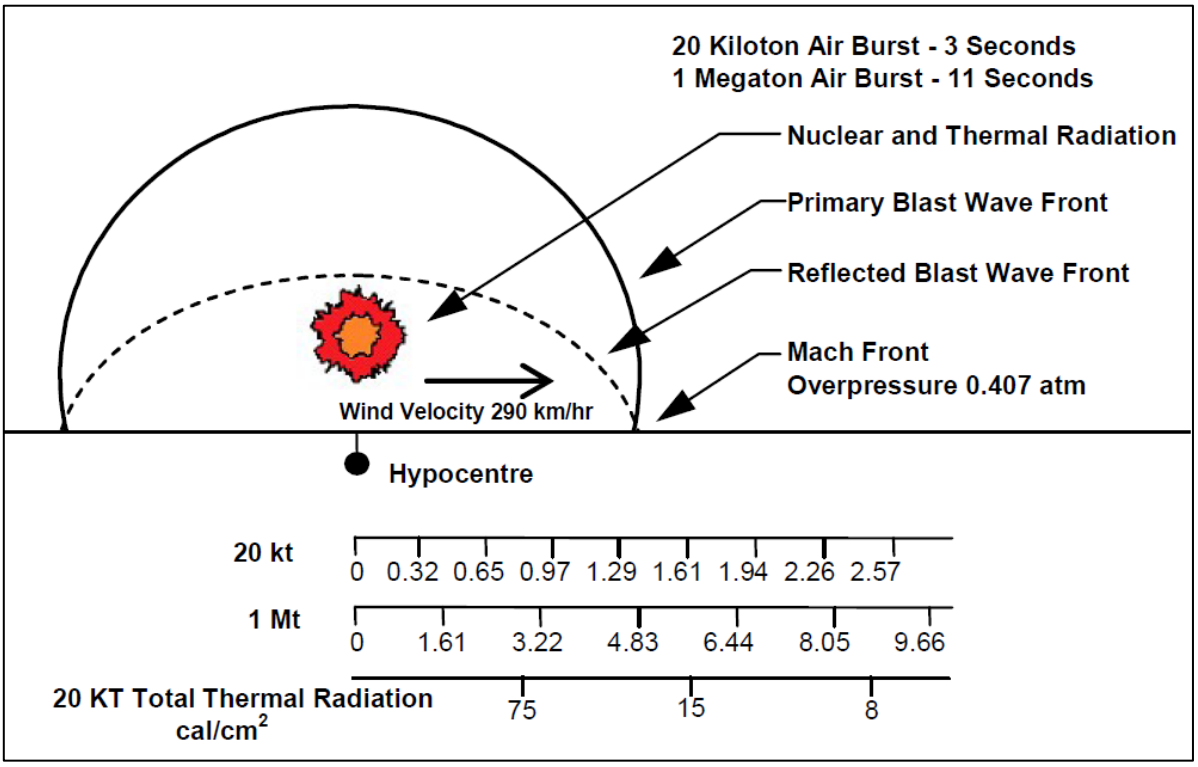

Figure 34-3 – Blast Properties of a Nuclear Air Burst Detonation.

34.2.2. BLAST WINDS

1. In passing through the atmosphere, the blast wave imparts its energy to the molecules of the surrounding air, setting them into motion in the direction of the advancing shock front. The motion of these air molecules is manifested as severe transient winds, known as blast winds. Two phenomena are associated with the blast wave in air:

a. *Static overpressure*. This is the sharp increase in pressure due to compression of the atmosphere. This positive pressure is exerted by the dense wall of air that comprises the wave front. The magnitude of the overpressure at any given point is directly proportional to the density of the air in the wave.

b. *Dynamic pressures*. These are the drag forces exerted by the strong transient blast winds associated with the movement of air required to form the blast wave. The pressures may be positive or negative due to the compression and displacement of air molecule.

2. As the blast wave moves out from the hypocentre, the peak (*positive phase*) overpressure of the front diminishes, while the decay of overpressure behind the front becomes more gradual. After traveling a sufficient distance from the fireball, the pressure behind the front drops below normal atmospheric pressure and is the so-called *negative phase* of the blast. The blast winds produce a large number of objects that are highly destructive (see Figure 34-4).

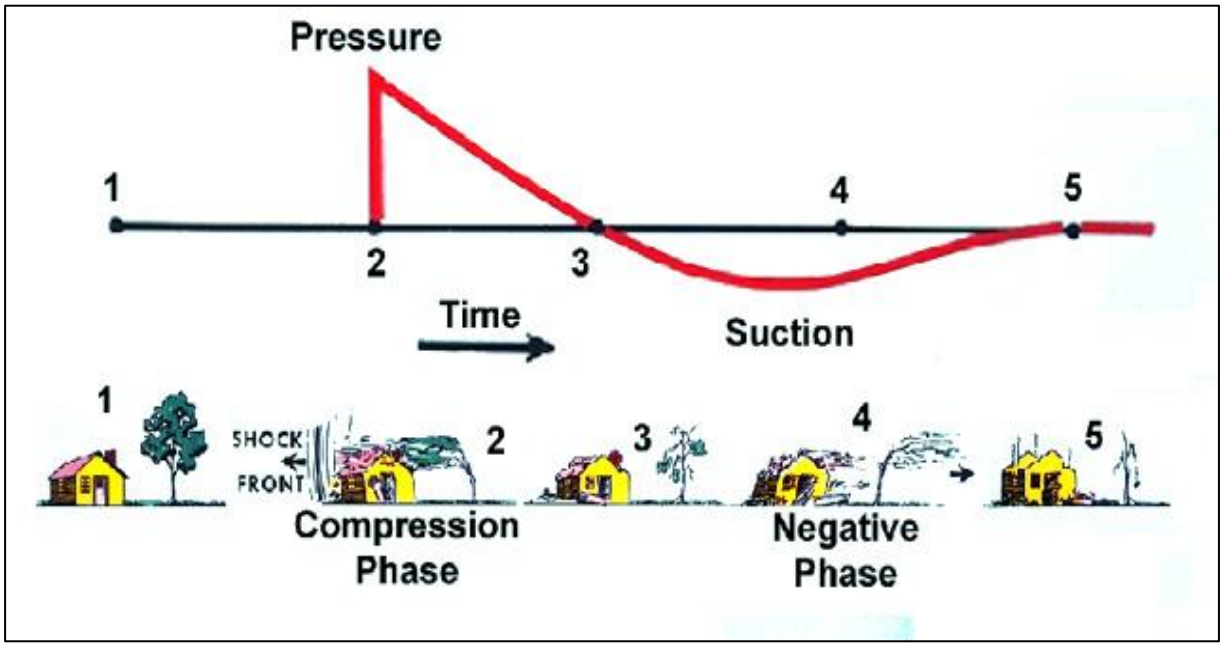

Figure 34-4 – Example of Blast Wave and Wind Progression.

3. For a short period after a nuclear air burst, there will be no increase in pressure since it takes a finite time for the shock front to reach a given point. This arrival time may range from a few seconds to minutes and will depend primarily on the distance of the location from the centre of burst and to a lesser extent on the yield of the detonation. Initially, the speed of the shock front is many times the speed of sound because it is traveling through superheated air, but as it travels away from the fireball, it slows down to the speed of sound.

4. Most blast damage will be experienced during the positive or compression phase of the wave. The relatively long duration of the compression phase of the blast wave is also significant in that structures weakened by the initial impact of the wave front are torn apart by the forces and pressures which follow. Forces many times greater than those in the strongest hurricane are present. These persist even through the negative phase of the blast wave when a partial vacuum is present because of the violent displacement of air. Damage sustained during the negative phase is generally minor, however, because the troughs of underpressure and wind velocity are relatively low.

5. The implications of the blast wind components depend on the types of objects in its path. Flat or recessed surfaces offer great resistance and hence are subjected to increased impact pressure and probability of damage. While the dynamic pressure at the face of a building is generally less than the peak overpressure due to the blast wave and its reflection, the period of dynamic loading is much longer than that of diffraction loading. Hence, the damage to frame-type buildings, bridges and other structures will be considerable. Equipment and personnel are relatively resistant to static overpressures but highly vulnerable to dynamic pressures. For example, military vehicles, from jeeps to tanks, are most likely to suffer damage when pushed, overturned, and thrown about by blast winds (see Figure 34-4 and Table 34-2).

Table 34-2: Blast Effects on Building and Casualties.[2]

Peak overpressure	Maximum wind speed	Effect on structures	Effect on the human body
1 psi (6.9 kPa)	38 mph (61 kph)	Window glass shatters	Light injuries from fragments occur
2 psi (13.8 kPa)	70 mph (113 kph)	Moderate damage to houses (windows and doors blown out and severe damage to roofs)	People injured by flying glass and debris
3 psi (20.7 kPa)	102 mph (164 kph)	Residential structures collapse	Serious injuries are common, fatalities may occur
5 psi (34.4 kPa)	163 mph (261 kph)	Most (non-reinforced) buildings collapse	Injuries are universal, fatalities are widespread
10 psi (68.9 kPa)	294 mph (470 kph)	Reinforced concrete buildings are severely damaged or demolished	Most people are killed
20 psi (137.9 kPa)	502 mph (803 kph)	Heavily built concrete buildings are severely damaged or demolished	Fatalities approach 100%
While humans can withstand higher overpressures that cause primary blast injuries in isolation, most casualties will die as a result of penetrating and blunt injuries.			

34.2.3. MEDICAL EFFECTS OF BLAST

1. The effects of blast on the human body following a nuclear detonation are similar to those of any other high-energy explosion (detonation). Blast winds are the cause of most blast injuries compared to primary blast injuries due to direct overpressure and blast wave.

2. Blast injuries are classified as:

a. *Primary*. This injury pattern is due to the blast wave and its direct interaction on the body, especially at air-tissue interfaces and long bones. These interfaces include the tympanic membrane, lungs and gastrointestinal tract. The impact on long bones with the blast wave results in mid shaft traumatic amputations with associated catastrophic bleeding and coagulopathy.

b. Secondary. These injuries result due the debris and fragments carried on the blast wind and are generally penetrating (fragmentation) injures.

c. *Tertiary*. These injuries are due to either the displacement of the body then striking blunt objects or the ground, or due to blunt injuries from falling or displaced objects such as falling masonry.

d. *Quaternary* (miscellaneous). These injuries are those that are not described above and include crush, burns, CBRN and psychological. For a nuclear detonation, there is a significant contribution from thermal exposure and irradiation.

3. Mechanical injuries due to missiles (secondary blast injuries) sent into motion by the winds or to violent bodily translation (tertiary blast injuries) will outnumber or be concurrent with any primary blast injuries.

[2] Glasstone and Dolan, 1977 and Sartori, 1983.

34.3. THERMAL EFFECTS

Large amounts of electromagnetic radiation in the visible, infrared and ultraviolet regions of the electromagnetic spectrum are emitted from the surface of the fireball within the first minute after detonation. This thermal radiation travels outward from the fireball at the speed of light. The main consequences of thermal radiation is the production of burns and eye injuries. The range of thermal effects increases with weapon yield. For high yield devices, thermal injuries may occur at distances where blast and initial nuclear radiation effects are minimal. Absorption of thermal radiation will also cause the ignition of combustible materials. Fires then spread rapidly among the debris left by the blast and potentially cause further burn casualties. The radius of the thermal effects as a function of yield is shown in Figure 34-2.

34.3.1. FIREBALL

Most of the energy released in the fission or fusion process, in addition to ionising radiation, is in the form of the kinetic energy. This energy generates a plasma ball with a temperature of several tens of million degrees centigrade. The transfer of energy to the surrounding atmosphere causes blast effects due to expanding air molecules and emits enormous quantities of energy in the form of visible light, infrared and ultraviolet radiation. The transfer of heat energy causes a fireball to be formed which expands rapidly. The fireball consists of two concentric regions, a hot inner core known as the isothermal sphere and an outer layer of luminous shock-heated air. Thermal emission from the fireball is governed by its apparent surface temperature which after an air burst will occur in two pulses. The initial pulse consists primarily of ultraviolet radiation and contains only about 1% of the total radiant energy of the detonation. The initial pulse is then terminated as the shock front moves ahead of the fireball. The thermal radiation emitted from the fireball surface during the second thermal pulse is responsible for most of the thermal effects but includes radiation in the infrared, visible and ultraviolet regions of the electromagnetic spectrum.

34.3.2. THERMAL RADIATION

1. The fireball can be considered a point source of radiation and most thermal radiation is emitted during the second thermal pulse. Thermal radiation travels at the speed of light and its mean free path (distance between point of emission and point of absorption) is relatively long. Thermal exposure (measured in joules per unit area of exposed surface) will be less farther from the centre of the detonation although the inverse square rule does not apply exactly because thermal radiation, particularly ultraviolet, will also be absorbed and scattered by the atmosphere. The thermal radiation intensity at a given point will also depend on the altitude and type of burst. In general, the thermal hazard is greatest in the case of a low-altitude airburst. General thermal effects will be less for surface bursts and frequently non-existent for subsurface bursts due to ground or water absorption. In surface bursts, a large part of the thermal energy is absorbed by the ground or water around the hypocentre as well as shielding due to terrain and absorption by dust, moisture and various gases in the air near the surface of the earth.

2. Since thermal radiation travels in straight lines from the fireball, any opaque object interposed between the fireball and the target will act as a shield and provide significant protection from thermal radiation. If a significant amount of scattering is present, as is the case when visibility is poor, thermal radiation will be received from all directions and shielding will be less effective. Thermal damage and injury is due to the absorption of large amounts of thermal energy within relatively short periods of time. The absorbed thermal radiation raises the temperature of the absorbing surface and results in scorching, charring, and possible

ignition of combustible organic materials, such as wood, paper, and fabrics (see below). If the target material is a poor thermal conductor, the absorbed energy is largely confined to a superficial layer of the material.

34.3.3. NUCLEAR CONFLAGRATION / SECONDARY FIRES

1. Ignition of materials exposed to thermal radiation is highly dependent on the duration of the thermal pulse, which is dependent on weapon yield, and the nature of the material, particularly its thickness and moisture content. At locations close to the hypocentre where the radiant thermal exposure exceeds 125 J/cm^2, almost all ignitable materials will flame, although burning may not be sustained. At greater distances, only the most easily ignited materials will flame, although charring of exposed surfaces may occur.

2. The probability of significant fires following a nuclear detonation depends on the density of ignition points, the availability and condition of combustible material (whether hot, dry, or wet), wind humidity, and the character of the surrounding area. Blast effects such as broken gas lines and overturned stoves and furnaces will compound the incendiary effects.

3. Depending on thermal and wind effects, a firestorm may develop. A firestorm burns in upon itself with great ferocity and is characterised by gale force winds blowing in toward the centre of the fire from all directions. In Hiroshima, a firestorm developed within 20 minutes of detonation. However, it is not a phenomenon specific to nuclear detonations. Firestorms are observed frequently in large forest fires and occurred following conventional incendiary bombing raids in both major theatres during World War II.

34.3.4. MEDICAL EFFECTS OF THERMAL RADIATION

The type of thermal injuries seen depend on the intensity and sustainability of the thermal exposure as well as protection factors. The main effects are:

a. *Close proximity to the fireball*. Close to the fireball, the thermal output is so great that everything is incinerated. Immediate lethality would be 100% within this range. The actual range within which overall lethality would be 100% will vary with yield, position of burst, weather and the environment.

b. *Direct (flash) burns*. Since the thermal pulse is direct infrared radiation, burn patterns will depend on direct line of sight, distance and clothing pattern absorption. The exposed person will absorb the infrared radiation in a variable pattern with burns on the side facing the detonation. The attenuating effect of heavy cloud cover is relatively small. Light coloured clothing will reflect the infrared, while dark portions will absorb it causing

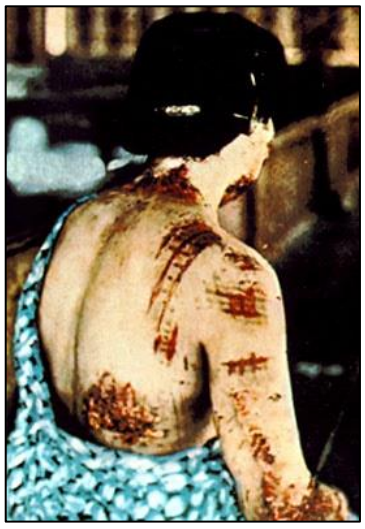

Figure 34-5 – Flash Burn.

patterned burns (see Figure 34-5)[3]. Persons shaded from the direct light will be protected from this form of injury. At temperatures below those required to ignite clothing or directly on naked skin, it is possible to transfer sufficient thermal energy to produce flash burns. Clothing significantly reduces the effective range producing partial thickness burns, thus affording significant protection against thermal flash burns.

c. *Indirect (flame) burns*. Indirect or flame burns result from exposure to fires caused by the thermal effects in the environment, particularly from ignition of clothing. Firestorm and secondary fires will cause typical flame burns, but they will be compounded by closed space, fire-associated injuries. This could be the predominant cause of burns depending on the number of and characteristics of flammable material in an environment. This is particularly true for the large-yield weapons, which can cause conflagrations and fire storms over extensive areas. Clothing made of natural fibres provide more protection next to the skin than manmade fibre which may melt. The probability of indirect burns cannot be quantified with range as well as can that of direct burns due to the variables of environmental flammability. Depending on the flammability of the material, blast winds can either extinguish or fan the burning material. Airway burns and lung damage are also more likely due to flames and smoke inhalation especially in enclosed environments and downwind. Early endotracheal intubation is advisable whenever airway burns are suspected and respiratory system complications are associated with severe morbidity and high mortality rates.

34.4. IONISING RADIATION EFFECTS

The ionising radiation effects due to a nuclear detonation are classified as:

a. Initial radiation.

b. Residual radiation.

(1) Fission products.

(2) Neutron activation products.

(3) Unused fuel (uranium, plutonium (and tritium)).

34.4.1. INITIAL RADIATION

1. About 5% of the energy released in a nuclear air burst is transmitted in the form of initial neutron and gamma radiation. The neutrons result almost exclusively from the energy-producing fission and fusion reactions. The initial gamma radiation includes that arising from the initial detonation and the decay of short-lived fission products (less than one minute). The intensity of the initial radiation decreases rapidly with distance from the point of burst. This is due partially to the inverse square rule and to absorption, scattering and capture by the atmosphere. The character of the radiation at a given location also varies with distance. Near the point of the detonation, the neutron intensity is greater than gamma. The neutron-gamma ratio decreases away from the centre, until the neutron component becomes negligible.

[3] The photo shows a Hiroshima victim with flash burns in a pattern similar to the clothing due to the reflection of thermal radiation by the lighter parts.

2. As the weapon yield increases, the range for significant levels of initial radiation does not increase markedly compared to the blast and thermal effects. Therefore, the initial radiation becomes less of an issue with increasing yield, as individuals close enough to be significantly irradiated are killed by the blast and thermal effects. With larger weapons (>50kt), blast and thermal effects are so much greater that initial radiation effects can be ignored although residual radiation must still be considered and mitigated against.

3. For small yield nuclear events (<10kt), including INDs, criticality events and an incomplete detonation of a larger weapon, there is the potential for proportionally more irradiated casualties compared to blast and thermal predicted for larger yields.

34.4.2. RESIDUAL RADIATION

1. The residual radiation hazard from a nuclear detonation is in the form of radioactive fallout (fission products and unused fuel) and neutron-induced activity. Residual radiation arises from a variety of sources:

a. *Fission products*. Fission products are intermediate-weight isotopes which are formed when a heavy uranium or plutonium nucleus is split during the nuclear fission process. There are approximately 300 different fission product isotopes of 36 elements that may result from a fission reaction. Many of these are radioactive, with widely differing half-lives. Some fission products have half-lives which are very short (fractions of a second). Others can be hazards for months or years. Their principal mode of decay is by the emission of beta and associated gamma radiation. Approximately 60g of fission products are formed per kiloton of yield. The estimated activity of 60g of fission products 1 minute after detonation is 1.1×10^{21} Bq. This level of activity is equal to that of 30 million kg of radium in equilibrium with its decay products.

b. *Neutron activation products*. If atomic nuclei are exposed to a flux of neutron radiation and capture neutrons, they will, as a rule, become radioactive (neutron-induced activity). They will then decay by the emission of beta and gamma radiation over an extended period of time. Neutrons emitted as part of the initial nuclear radiation will cause activation of the weapon residues, atoms of environmental material such as soil, air and water, depending on their composition and distance from the burst. For example, a small area around the hypocentre may become hazardous as a result of exposure of the minerals in the soil to initial neutron radiation. However, this is a usually negligible hazard because of the limited area involved and greater hazard from the fireball and blast.

Note. This form of irradiation is important during criticality events and neutron capture by sodium and phosphorous, in biological tissue, can be used for dose estimation.

c. *Unused fuel*. Nuclear weapons are relatively inefficient in their use of fissionable material, and much of the uranium and plutonium is dispersed by the detonation without undergoing fission. Such unfissioned nuclear material decays primarily by the emission of alpha particles and is of relatively minor importance as long as it remains outside of the body.

34.4.3. NUCLEAR FALLOUT

1. Nuclear fallout is a major contribution to the residual radiation described above and is a significant hazard following a detonation and extends beyond the immediate area. It consists mainly of vaporised fission products, unused fuel and weapon residues. In addition, depending

on the type of device, ground material and water may also be raised into the atmosphere. This additional material may also include neutron activated products. The suspension of residual radiation particles will depend on the yield, type of device (airburst and surface compared to sub-surface) and meteorological conditions. The future effects of the fallout will depend on the timing of the deposition back to the ground as this determines the radioisotope mixture, concentration and location. Early deposition in the first 24 hours will be concentrated, highly radioactive due to the presence of radioisotopes with short half-lives. Following the atomic bombings in 1945, the phenomenon of 'black rain' was described and was a significant contribution to the number of radiological casualties outside the lethal thermal and blast zone. Late deposition may be months and even years later and will have a wider more global distribution.

2. *Meteorological conditions*. Meteorological conditions will greatly influence fallout, particularly local fallout. Atmospheric winds are able to distribute fallout over large areas. For example, as a result of a surface burst of a 15 Mt thermonuclear device at Bikini Atoll on 1 March 1954, an approximate cigar-shaped area of the Pacific extending over 500 km downwind and varying in width to a maximum of 100 km was severely contaminated. Snow and rain, especially if they come from high altitude, will accelerate local fallout. Following these conditions, local rain showers ('black rain') originating above the radioactive cloud will form limited areas of heavy contamination.

3. *Types of detonations*. After a high airburst detonation, particularly if the explosive yield exceeds 10 kT, residual material is vaporised by the heat of the fireball and will condense into a fine suspension of very small particles 0.01 to 20 µm in diameter. These residual particles may be quickly drawn up into the stratosphere. They will then be dispersed by atmospheric winds and will gradually settle back to the Earth's surface over an extended period of time, even years, as worldwide fallout. In a surface or low-altitude airburst, over land or water, large amounts of earth or water may be vaporised by the heat of the fireball and drawn up into the radioactive cloud.[4] This material has a wider range of particle sizes from less than 0.01 micrometre to several millimetres in diameter. The larger particles will not rise into the stratosphere and will settle back to earth within 24 hours as early localised fallout far beyond the blast and thermal effects, particularly in the case of high-yield surface detonations. Subsurface bursts present an additional phenomenon called base surge. The base surge is a cloud that rolls outward from the bottom of the column produced by a subsurface detonation.

4. *Early (local) fallout*. Early or local fallout describes the residual radiation within the first 24 hours usually following a surface or low-altitude airburst detonation. The fallout cloud will contain highly radioactive small to large residual particles. The larger particles will return to the ground especially in precipitation such as rain or snow.

5. The key features of early fallout are:

   a. Usually within 24 hours but depends on local meteorological conditions.

   b. Localised deposition although likely to be outside the thermal and blast lethal zone.

   c. Highly radioactive with a greater mix of radioisotopes including those with relatively short half-lives.

[4] The Hiroshima atomic bomb detonated only 600m over the hypocentre resulting in the classical mushroom cloud phenomenon and resulting 'black rain'.

d. Significant health risks due to:

(1) Internal contamination due to inhalation, ingestion or wound contamination.

(2) Direct gamma irradiation hazard.

(3) Beta burns.[5]

6. Hazard management to protect against the effects of early fallout includes sheltering, use of appropriate PPE (although this will not mitigate gamma radiation hazard), use of stable iodine MedCM, and delaying re-entry for recovery operations for three to five weeks or until dose rates are at safe levels. The dose rate of residual radiation follows the rule of seven, a ten-fold decrease for every seven-fold increase in time (see Table 34-3).

Table 34-3: Reduction in Residual Radiation Dose Rate (Rule of Seven).

Duration since detonation	Residual radiation
0 hours	100%
7 hours	10%
49 hours (2 days)	1%
343 hours (2 weeks)	0.1%

7. *Late (global) fallout.* Late or global fallout describes the more regional and global effects of fallout due to smaller residual particles in the stratosphere. The fallout will return to the ground at a time when most of the short half-life radioisotopes have decayed sufficiently not to contribute to the continuing radiological hazard. The radiobiological hazard of worldwide fallout is essentially long term due to the potential accumulation of long-lived radioisotopes, such as strontium-90 and caesium-137, in the body as a result of ingestion of foods or water that had incorporated these radioactive materials. Late fallout should not cause ARS.

8. The key features of late fallout are:

a. Beyond 24 hours, and may be months or years after the detonation.

b. Continental and global deposition.

c. Less in number but significant radioisotopes remain including strontium-90 and caesium-137 with relatively longer half-lives. Radioiodine is also an important consideration in the first few weeks and will be a regional (cross-border) issue.[6]

d. Significant health risks due to:

(1) Contamination of water and the food chain and resulting food restrictions.

(2) Internal contamination due to ingestion of contaminated water and food.

9. The hazard from late fallout though serious is less immediate than the hazard associated with early fallout. The radiological health effects will be stochastic rather than acute

[5] Beta burns were seen in the Marshall Islanders after a change in meteorological conditions during atomic bomb testing.

[6] Following the Chernobyl nuclear power reactor accident stable iodine was distributed to residents in neighbouring Poland.

deterministic. Early and localised fallout is of much greater immediate operational concern while late fallout will have global and strategic consequences including food restriction but are outside the scope of this publication.

34.4.4. MEDICAL EFFECTS OF NUCLEAR RADIATION

1. *Initial radiation*. For large nuclear detonations (> 10kt), initial radiation is unlikely to have a great impact on the medical response as the lethal effects of the blast and thermal components will be greater. INDs, tactical weapons and criticality incidents may see a higher proportion of significantly irradiated casualties including combined injuries. For criticality incidents, neutron radiation will be a significant contribution while potential assays for neutron-activated products including sodium-24 and phosphorous-32 will be needed.

2. *Residual radiation*. Residual radiation is of greater concern due to the risk of internal and wound contamination, gamma irradiation and beta burns especially in the first 24 hours ad locally. Exposure may lead to a range of effects including significant whole body irradiation, local skin effects including beta burns and internalisation of radioisotopes including caesium-137, strontium-90 and iodine radioisotopes. Significant long-term effects may include increased cancer risk, especially that of thyroid cancer in children and adolescents. Early medical management will be focused on preventing or reducing internalisation, casualty decontamination, blocking the absorption of radioiodine with stable iodine, decorporation and the early triage and management of casualties including surgery and burn management.

34.5. EYE INJURIES

1. Sudden exposures to high-intensity sources of visible light and infrared radiation can cause eye injury, specifically to the chorioretinal areas. Although the most common source of thermal energy injury to the eye is from directly viewing the sun, other sources of luminance including nuclear detonations can cause eye injuries. Factors that determine the extent of eye injury include pupil dilation, spectral transmission through the ocular media, spectral absorption by the retina and choroid, and duration of exposure.

2. Direct-vision optical equipment such as binoculars will increase the likelihood of damage. Optical devices such as night-vision equipment that provide electronically enhanced images cannot reproduce the intensity of the thermal pulse and will not cause injury. Eye injury is due not only to thermal energy but also to photochemical reactions that occur within the retina with light wavelengths in the range of 400 to 500nm. To fully protect against injury, the eyes should be covered or shielded with protective googles.

3. The two types of eye injury following a nuclear detonation are:

   a. *Flash Blindness*. Flash blindness occurs with sudden peripheral visual observation of a brilliant flash of intense light energy e.g. a fireball. This is a temporary condition that results from a depletion of photopigment from the retinal receptors. The duration of flash blindness can last several seconds when the exposure occurs during daylight. The blindness will then be followed by a darkened afterimage that lasts for several minutes. At night, flash blindness can last for up to 30 minutes and may occur kilometres away from the blast.

   b. *Retinal burns*. Direct observation of a brilliant flash of light in the wavelengths of 400 to 1400 nm can cause macular-retinal burns. Burns of the macula will result in permanent scarring with resultant loss in visual acuity. Burns of the peripheral regions

of the retina will produce scotomas (blind spots) but overall visual acuity will be less impaired. These burns can occur at distances of several miles under optimal conditions.

c. *Blast related*. 10% of blast casualties will have associated eye injuries. These may include penetrating injuries and foreign bodies, subconjunctivial injury, globe perforation and retinal detachments.

4. Following nuclear detonation or blast, early ophthalmological assessment and triage is recommended subject to casualty burden and medical resources available.

34.6. COMBINED INJURIES

1. Combined injury in the context of a radiological or nuclear incident describes a significant exposure to ionising radiation and significant traumatic injuries including crush, blast and burns. Nuclear detonations will cause combined injuries. Other nuclear events such as reactor accidents and criticality incidents may not always have combined injuries. It is generally accepted that combined injuries have a worse prognosis than for irradiation alone.

2. Tactical and strategic MEDEVAC assets should be made available as combined injury patients should be evacuated to hospital care including Role 4 as soon as possible. In the first 2 days, combined casualties may be triaged for early surgery so that it is completed within 48 hours of the radiation injury before the onset of immunosuppression and coagulopathy. Surgery delayed past this timeframe may be associated with increased mortality and morbidity. However certain procedures such as skin grafting and other reconstructive surgery could be deferred.

3. In nuclear warfare scenarios and other mass casualty events, the use of the expectant (T4) triage category may consider the severity of combined injury including injury to the torso, severity of burns, radiation exposure and the resources available. However, this approach may be delayed due to advances in radiological triage, the treatment of the haematopoietic ARS sub-syndrome, and availability of international mutual aid.

4. Further management considerations of these effects are given in Chapter 35.

34.7. ELECTROMAGNETIC PULSE AND THE MEDICAL RESPONSE

A high-altitude detonation can produce an electromagnetic pulse (EMP) that could adversely affect medical capability. EMP is an electromagnetic field generated from the detonation that produces a high-voltage surge. This voltage surge can impact electronic components that it reaches. The EMP phenomenon is a major effect for bursts at very high altitude, but it is not well understood how it radiates outward from a surface level detonation and to what degree it will damage the electronic systems that permeate modern society. Many critical medical, electronic and transport equipment systems are not hardened against EMP effects. Consequently, medical operations including MEDEVAC could be severely compromised. Because the extent of the EMP effect is expected to occur relatively close to the hypocentre, other effects of the explosion (such as blast and thermal effects) are expected to dominate over the EMP effects. Equipment brought in from unaffected areas should functional normally if electronic infrastructure and services remain functioning.

34.8. MEDICAL PLANNING FOR NUCLEAR INCIDENTS

1. Medical operations in contaminated or radiation environments impose additional constraints and logistical requirements. The radiological hazards and force protection issues

are complex with a requirement to protect and treat those exposed to very high levels of radiation, while also limiting the occupational exposure of responders to as low as reasonably acceptable (or practicable). Prolonged treatment of radiation casualties at deployed sites is not practical, and there will be a significant pressure and re-evaluation of MEDEVAC resources depending on the severity and numbers of casualties especially during a MASCAL event.

2. The likely medical scenarios for nuclear consequence management include:

a. Medical response to a local / tactical level nuclear event (deliberate or accidental) with preserved medical infrastructure and personnel.

b. Medical response to a local / tactical level nuclear event (deliberate or accidental) with loss of local medical infrastructure and personnel.[7]

c. Medical response in support of a non-combatant evacuation (NEO) mission.

d. Medical response in response to a strategic nuclear attack.

34.8.1. MEDICAL RESPONSE PHASES DURING A NUCLEAR INCIDENT

1. Rescue operations, damage control, and medical support operations are complementary and should be closely coordinated. This includes establishing consolidated staging, treatment and evacuation sites in areas of relative safety from residual radiation, secondary explosions, fires and potential civil disturbance. The medical load may be overwhelming, and every effort should be made to conserve resources so as to provide effective medical care for the maximum number of casualties. Therefore, medical personnel should not be taken from primary patient care duties and used to perform rescue and damage control operations.

2. The phases of a medical response following a nuclear detonation will reflect the varying casualty types and onset effects.

a. *Immediate phase (day zero)*. This phase will focus on initial extraction, rescue and early triage of casualties in and from the hot zone. The most effective evacuation method for casualties during this phase will be self-extraction supported by rescuers when available and as practicable due to hazards including residual radiation, fires and building instability. Casualty care at this point will be enhanced first aid for trauma only with the priority being evacuation. Rescue and damage control personnel should be designated, trained and equipped to render life-saving (enhanced) first aid. Rescue efforts may have to be conducted in the presence of fallout contamination or with the possibility of fallout arriving at a later time. Qualified radiation monitors should be available to evaluate radiation dose rates and provide specific recommendations to the commander as to the hazards present especially if there is also a high gamma dose rate that will require time on scene limitations.

b. *Casualty reception phase (week one)*. This phase relies on the early establishment upwind of peripheral casualty reception centres and casualty clearing stations away from residual radiation hazards including fallout. Medical care is focused on decontamination and trauma care with supportive management and documentation of any early prodrome symptoms. Triage will be based on the indications for trauma care

[7] Following the Hiroshima bomb, approximately 90% of medical personnel were killed or injured. 18 hospitals and 32 clinics were destroyed.

based upon severity of injuries, burnt percentage body surface area and other features. Most casualties significantly irradiated will have also been exposed to lethal thermal or blast effects. However, for low yield events or certain situations where a casualty survived the thermal and/or blast effects, early post-exposure radiation management should be the goal although realistically is unlikely to be immediately available. During a MASCAL, it is likely that the expectant (T4) triage category would be implemented at the casualty clearing station due to the finite MEDEVAC resources and overwhelmed receiving medical facilities.

c. *Trauma casualty response phase (week one to two)*. Management of casualties during the first two weeks will be focused on trauma care including surgery and supportive care. Where resources allow, casualties with risk of ARS and requiring surgery should be prioritised. However, if resources are limited surgery may be deferred in order to maximise care for casualties with a greater chance of survival. Trauma care will continue into the next phase and the recovery (rehabilitation) phase for patients that do not have contra-indications for further surgery. It is likely that trauma casualties will be MEDEVAC back to the next level of care in the medical evacuation chain either to a Role 3 or Role 4 MTF.

d. *Radiation casualty response phase (up to 60 days)*. This period after the initial event will see the manifestations of ARS including infections, coagulopathy and fluid loss. Due to the time delay, there is a potential window of opportunity to evacuate casualties to multiple specialist receiving units or forward deploy radiation specialists and medical treatments. Preparation for this phase will include radiological triage during the previous two weeks in anticipation.

e. *Recovery (rehabilitation) phase (month two to three onwards)*. Patients reaching this phase are likely to survive and medical care is now focused on the restoration of function, and prevention or treatment of complications. Treatment will include reconstructive surgery especially for burns. Potential complications include keloid scarring of the skin, eye disability and psychological reactions and disorders. Key medical personnel will include plastic surgeons, ophthalmologists and mental health care practitioners as well as friends and family. Formal counselling of the risk of stochastic effects due to any radiation dose received will also be required; this will include risk of cancer and potential genetic effects

f. *Long-term (health surveillance) phase*. Following any significant health event, it is likely that a health register will be established and include health surveillance. This will be both proactive for known effects such as cancer and cataracts, and reactive to any new features described in the exposed or injured cohort previously not documented. A health surveillance should include health education as well as periodical health screening / monitoring or self-examination. This should be supported by appropriate data collection methods and statistical analytical support.

34.8.2 NUCLEAR WEAPON AND REACTOR ACCIDENT CONSIDERATIONS

1. Annex 34A provides an outline of some of the considerations following a nuclear weapon accident with the emphasis on non-nuclear explosive risks and radiological and non-radiological hazards.

2. Annex 34B provides an outline of the classification of nuclear incidents and accidents whether due to combat, sabotage or industrial accident. Some of the considerations and routes of exposure are also considered.

ANNEX 34A – NUCLEAR WEAPON INCIDENTS

34A.1. INTRODUCTION

Nuclear weapon accidents could be peacetime or wartime events. Medical services should be prepared to provide the response required if they occur. Responders should be aware that the major hazard from a weapon accident is an explosive hazard and not a nuclear detonation. There are radiological hazards but these are weak x-ray and gamma emitters.

34A.2. EXPLOSIVE HAZARDS AND RISK OF ACCIDENTAL DETONATION

All nuclear weapons contain substantial amounts of high explosives. In any accident with explosive material, there can be risk of explosion and fire. Either may occur immediately or later if a weapon is severely damaged and fragments of high explosive and nuclear material are scattered. All personnel at an accident site must be aware of these hazards and conduct all operations and duties under the direction of experienced ordinance disposal personnel.

Note. A nuclear detonation is not a credible event. The mechanical physics of a nuclear weapon's design are such that accidental detonation will not occur.

34A.3. RADIOLOGICAL HAZARDS

The principal fissionable materials in nuclear weapons (^{235}U and ^{239}Pu) are basically alpha particle emitters. However, there are several weak (up to 185-kiloelectronvolt) x-ray and gamma emissions associated with the alpha-particle decay although the radiation intensity at an accident site is generally low and quantifiable with a dosimeter. The principal radiological hazard is from airborne alpha-particle emitters.

34A.3.1. PLUTONIUM

1. Plutonium (Pu) is a heavy metal (atomic number 94) which is artificially produced in reactions by bombardment of ^{238}U with neutrons. Most plutonium so produced is ^{239}Pu. However, relatively small quantities of other isotopes are also produced, and ^{241}Pu, ^{242}Pu, and ^{244}Pu will be present in small quantities in the plutonium used in weapons.

2. If plutonium particles are inhaled, they will be deposited at all levels of the respiratory system, depending on their size. The larger particles are deposited in the nasopharynx or high in the tracheobronchial tree. Only the very small particles, 10 microns in diameter or smaller, are deposited in the alveolar air sacs. The plutonium deposited down to the terminal bronchioles will be cleared from the lungs by the respiratory mucosa. These particles do not present any significant hazard. The possibility of any significant radiation damage while they are in transit out of the lungs or subsequently during their passage through the gastrointestinal system is almost non-existent. Any cells that are damaged by radiation would be replaced during the normally high rate of cell turnover that occurs in these tissues.

3. The plutonium remaining in the alveoli can cause damage, since much of it will remain there essentially for the lifetime of the individual. The rate of removal of plutonium deposited in the air sacs is difficult to estimate, but animal experimentation has indicated that it would take at least several years for significant amounts to be removed. Some of the plutonium particles are phagocytised and picked up by the lymphatic system, but they will not be transported far since a large proportion will be trapped in regional lymph nodes of the lung. Only very negligible quantities will reach the blood stream.

4. Radiation from plutonium and its daughter products trapped in the lung tissue can cause an inflammatory response and eventual fibrosis. The degree of fibrotic scarring will depend upon the amount of plutonium deposited and time. The overall reserve capacity of the lungs is so great that this would only rarely become a serious problem. Carcinogenesis must also be considered the main stochastic hazard, but it is of small relevance in nuclear war. In operations short of actual war, long-term consequences will be of concern to commanders, engaged personnel, and national populaces. Most cells damaged by alpha radiation will be lethally damaged. A very small percentage will be non-lethally damaged. However, there is some x-ray and gamma radiation associated with plutonium and its daughter products. The hazard of this radiation to an organ like the lungs is difficult to assess, but it is penetrating and must also be considered as a potential producer of both fibrosis and cancer. This x-ray and gamma radiation has a very low energy level (17 keV and 60 keV respectively) and is difficult to detect at low concentrations with standard x-ray sensitive instruments. Special probes are available as accessories to some alpha-sensitive detection instruments and are of value in monitoring contamination by plutonium.

34A.3.2. URANIUM

Uranium (U) is a heavy metal (atomic number 92) and is primarily an alpha emitter, but both gamma and beta radiation are also produced. On a gram for gram basis, the typical uranium alloy has only about 1/500 the radioactivity of an equivalent amount of ^{239}Pu. Therefore, the radiation hazard associated with uranium is much less than that associated with plutonium. Otherwise, the same factors governing deposition and retention in the pulmonary system apply to uranium. Uranium metal can cause chemical toxicity (heavy metal toxicity) at exposures of 0.1 milligram per kilogram body weight. This is seen as damage to the cells of the lower portion of the proximal convoluted tubules of the kidney. There is usually a lag period of 6 hours to several days followed by chemical necrosis. Even after levels that cause necrosis, the kidneys show evidence of regeneration within 2 to 3 days, depending on the severity of the initial exposure. Bicarbonate and alkalisation of the urine has been recommended to increase renal elimination.

34A.3.3. TRITIUM

1. Tritium (^{3}H) is an isotope of hydrogen with a nucleus composed of two neutrons and one proton. There are two other isotopes, normal hydrogen (^{1}H), which has one proton in its nucleus and no neutrons, and deuterium (^{2}H), which has one neutron in its nucleus in addition to the proton. Deuterium is not radioactive and occurs naturally in small quantities. Tritium is radioactive, emitting a low-energy beta particle. It is extremely rare in nature and must be made artificially in reactors to meet the requirements for its scientific, industrial and military uses. Tritium has a physical half-life of 12.26 years. While tritium gas readily dissipates into the atmosphere (like hydrogen gas), water formed from tritium can be readily absorbed into the body and becomes diffusely distributed throughout the body water. It may be inhaled, ingested, or absorbed transcutaneously. If a large amount is incorporated into the body accidentally, there is a serious risk of whole-body beta irradiation and acute radiation sickness as described earlier in Chapter 32.

2. If patients are seen with suspected tritium contamination, the best treatment is to shorten the turnover time of the body water with managed fluid intake and diuretics. This essentially flushes out the tritium and can materially reduce the exposure time and the total dose of radiation received. Fortunately, tritium would not be a hazard in the usual accident situation because it dissipates so rapidly. Generally, it will not accumulate in an outside area, although

tritium contamination of metal or other surfaces can be persistent. Tritium is usually only considered an internal contamination hazard if it is encountered in a very confined space.

34A.3.4. MAGNESIUM-THORIUM ALLOYS

Magnesium-thorium alloys should also be considered as radioactive hazards since radioactive thorium is an alpha emitter. Many aircraft and missile structures contain significant amounts of magnesium-thorium (up to 4% of thorium); therefore, in an accident, the radioactive thorium must be recovered and disposed of as radioactive waste.

34A.4. NON-RADIOACTIVE HAZARDOUS MATERIALS

In any modern weapon system, a fire involving the various components of airplanes, missiles, and so on will release a large variety of nonradioactive toxic materials into the atmosphere. Among these, beryllium is the most important because of the severity of the clinical effects that may be associated with any exposure to this quite hazardous material. This material is far more important than any others discussed below.

34A.4.1. BERYLLIUM

Beryllium is a light, grey-white, nonradioactive metal. However, it is one of the most toxic metals known. If beryllium in any form is inhaled, a particularly severe pneumonitis (berylliosis) may follow. This is primarily a Type IV reaction that results in a diffuse pulmonary granulomatosis. The resulting pulmonary disability is progressive and frequently fatal, either within a few months or after a period of several years. There is no specific treatment. In addition, beryllium can be a hazard if it contaminates wounds. Such contamination results in a severe local inflammatory response and the development of persistent granulomatous lesions requiring surgical excision. Since beryllium is not radioactive, its presence at an accident site cannot be detected readily with radiological monitoring instruments.

34A.4.2. LITHIUM AND PLASTICS

1. Lithium is the lightest of all metals and is used extensively in the field of nuclear technology, often in the form of a hydride. If lithium hydride is exposed to water and carbon in the presence of a fire, a chemical reaction occurs that produces acetylene gas and lithium hydroxide. The gas in turn increases the intensity of the fire, while the strong alkali is caustic and a strong pulmonary irritant.

2. A wide variety of plastics are used in modern military weapon systems. Many of these, if burned, produce toxic fumes including cyanide.

34A.5. ON SCENE PRIORITIES FOR THE MEDICAL RESPONSE

The priorities for the medical incident management follow the same principles as described in Part 2. As the radiological hazards are relatively weak, the mainstay of casualty management will be the management of any trauma and casualty hazard management. ARS is extremely unlikely. Contamination of the injured with varying amounts of radioactive material may be present, but this contamination should not be a serious or an immediate hazard to either the injured or to personnel caring for them.

34A.6. CONTAMINATION OF THE GEOGRAPHICAL AREA SURROUNDING AN ACCIDENT WITH POTENTIAL HAZARD TO LOCAL POPULATION

1. Medical personnel may be called upon to give advice as to the nature and degree of public hazard associated with a given type and level of contamination. This hazard will rarely be an acute one but may well be a significant long-term one. The advice given will be an essential factor in determining what methods are used to minimise and remove the hazard.

2. The most probable hazard will occur downwind from an accident site due to airborne particles of radioactive material which could be inhaled. Sheltering is the most efficacious first action. Early after an accident, adequate information may not be available to determine the exact degree of the hazard from airborne contamination. As a result, a decision to evacuate an area close to an accident location may have to be made by local authorities without waiting for radiation measurements. No precise guidance can be given for these types of situations.

3. A much less frequent hazard would be contamination of water supplies if an accident occurred near a river source or reservoir. Dilution and settling of insoluble materials would further reduce this small hazard, and simple monitoring measures by trained personnel could be obtained before condemning the water supply. Water from other locations can be used temporarily until adequate measurements are made to determine whether there has been contamination or not. However, drinking water contaminated by plutonium is an insignificant hazard due to the immense dilution factor and the relative insolubility of the material.

34A.7. DECONTAMINATION OPERATIONS

Exposure-level criteria used in accident-related operations are basically the same as peacetime industrial exposure limits, as defined by the laws of the country in which the accident occurs or by international agreement (wartime exposure limits may be higher). Emergency exposures exceeding specified limits must be restricted as much as possible to those truly critical operations such as rescue of the injured. Once the emergency phase of a nuclear accident is over, decontamination must be carried out under strictly controlled medical supervision. This is a separate operation from care of the injured and requires the presence of specially trained medical practitioners and health physicists.

ANNEX 34B – NUCLEAR REACTOR INCIDENTS

34B.1. INTRODUCTION

Any operation acting in a region with nuclear reactors should consider the possibility of a contingency plan in the event of a nuclear incident or accident. The impact of such an event has similar consequences as described in the main chapter:

a. Potential traumatic injuries either during the initial incident or in response.

b. High dose or dose rate exposures due to the nuclear process.

c. Exposure to a range of radioisotopes (fission products), many of which will be highly potent during the first few days following an event. Most will be beta emitters with associated gamma emissions.

d. Exposure to radioiodine (~2% of fission products) and potential for stable iodine to be used as a radiological MedCM.

e. Risk of internal contamination either by inhalation or ingestion either directly or through the water and food chain (see Figure 34C-1).

f. Risk of fallout from airborne nuclear material.

g. Risk of direct irradiation (shine) from settled nuclear material.

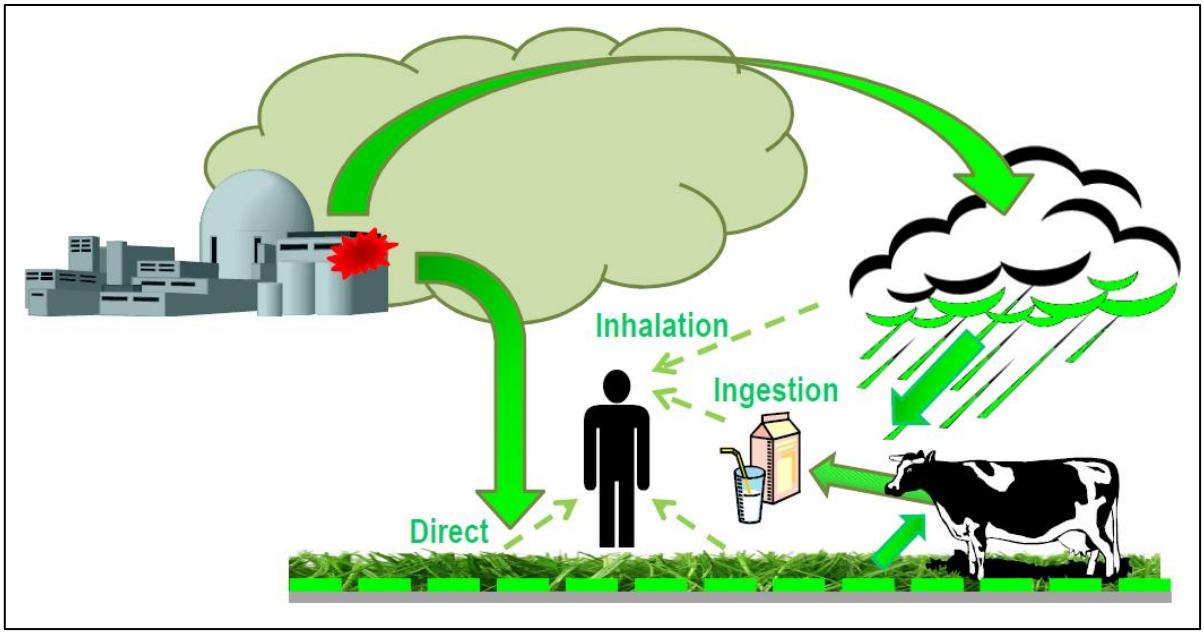

Figure 34B-1: Consequences of a Nuclear Reactor Incident.

34B.2. FISSION PRODUCTS

Significant fission products include the radioisotopes of strontium, iodine and caesium. Although there are many more these isotopes have relatively longer half-lives and are

absorbed into the body in locations such as the thyroid (iodine) and bones (caesium and strontium).

34B.3. INTERNATIONAL NUCLEAR EVENT SCALE (INES)

1. The International Atomic Energy Agency developed a 7 scale indicator of severity following a nuclear or radiological event (see Figure 34B-1). The scale is designed to be almost logarithmic with each level being 10 times worse than the previous. Events are differentiated into:

a. Incidents – Small scale events.

b. Accident – Large scale events.

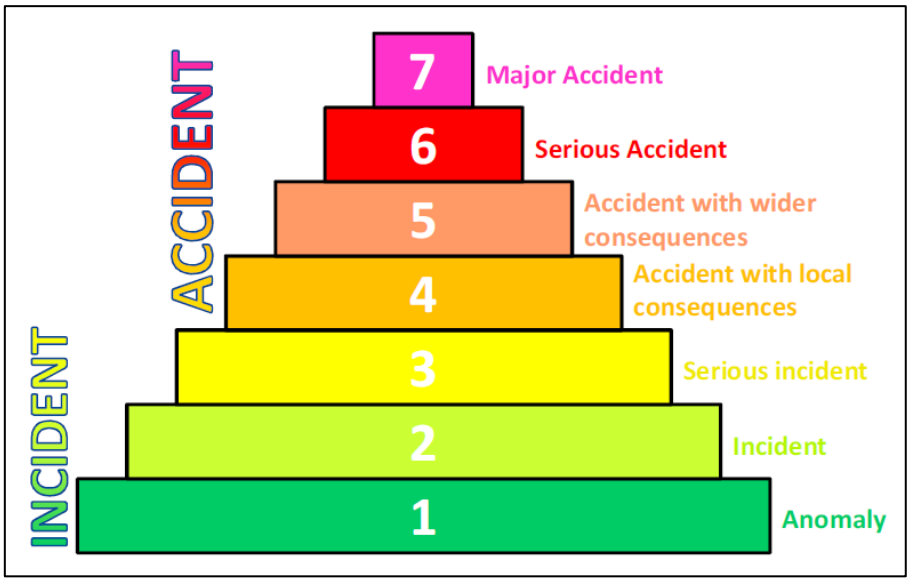

Figure 34B-2: IAEA Nuclear Accident Scale. (●)

2. The impact of each level is summarised below in Table 34B-1.

3. Examples of certain levels of nuclear or radiological events include:

a. Chernobyl (1986) – Level 7 reactor accident with widespread health and environmental effects. External release of a significant fraction of reactor core inventory.

b. Fukushima (2011) – Level 7 reactor accident following an earthquake and tsunami.

c. Windscale (1957) – Level 5 accident due to a graphite fire releasing radioactive material into the environment.

d. Three Mile Island (1979) – Level 5 reactor accident with reactor core damage.

e. Goiania (1987) – Level 5 radiation accident with the rupturing of an abandoned ^{137}Cs source leading to four deaths.

f. Tokaimura (1999) – Level 4 criticality event with fatal overexposures of workers.

Table 34B-1: INES Criteria.

INES LEVEL	People and Environment	Radiological Barriers and Control	Defence-in-Depth
Major Accident Level 7	• Major release of radioactive material with widespread health & environmental effects requiring implementation of planned and extended countermeasures.		
Serious Accident Level 6	• Significant release of radioactive material likely to require implementation of planned countermeasures.		
Accident with Wider Consequences Level 5	• Limited release of radioactive material likely to require implementation of some planned countermeasures. • Several deaths from radiation.	• Severe damage to reactor core. • Release of large quantities of radioactive materials within an installation with a high probability of significant public exposure. This could arise from a major criticality accident or fire.	
Accident with Local Consequences Level 4	• Minor release of radioactive material unlikely to result in implementation of planned countermeasures other than local food controls. • At least one death from radiation.	• Fuel melt or damage to fuel resulting in more than 0.1% release of core inventory. • Release of significant quantities of radioactive material within an installation with a high probability of significant public exposure.	
Serious Incident Level 3	• Exposure in excess of ten times the statutory annual limit for workers. • Non-lethal deterministic health effect (e.g. burns) from radiation.	• Exposure rates of > 1 Sv/h in an operating area. • Severe contamination in an area not expected by design, with a low probability of significant public exposure.	• Near accident at a nuclear power plant with no safety provisions remaining. • Lost or stolen highly radioactive sealed source. • Mis-delivered highly radioactive sealed source without adequate procedures in place to handle it.
Incident Level 2	• Exposure of a member of the public in excess of 10mSv. Exposure of a worker in excess of the statutory annual limits.	• Radiation levels in an operating area > 50 mSv/h. • Significant contamination within the facility into an area not expected by design.	• Significant failures in safety provisions but with no actual consequences. • Found highly radioactive sealed source, device or transport package with safety provisions intact. • Inadequate packaging of a highly radioactive sealed source.
Anomaly Level 1			• Overexposure of a member of the public in excess of statutory annual limits. • Minor problems with safety components with significant defence-in-depth remaining. • Low activity lost or stolen radioactive source, device or transport package.

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CHAPTER 35: MANAGEMENT OF THE RADIOLOGICAL CASUALTY

35.1. INTRODUCTION

1. The management of radiological casualties follows the same principles of CBRN casualty management. The priorities depend on the presence of trauma, contamination and external irradiation and is summarised as the radiation casualty management pathway adapted from the REAC/TS management algorithm (see Annex 35A). They include:

a. Starting radiological MedCM:

(1) Radiation protectants (pre-event MedCM).

(2) Radiation mitigators (post-event MedCM).

(3) Decorporating agents to mitigate internal contamination.

b. Triage for trauma and radiation effects.

c. Casualty assessment:

(1) Initial assessment 'quick look' for trauma.

(2) Further assessment for prodrome and other predictive features for ARS.

d. Emergency Medical Treatment (EMT) of T1 casualties.

e. Damage control resuscitation and surgery for life or limb threatening injuries[1].

f. Casualty hazard management:

(1) Management of external and wound contamination.

(2) Management of internal contamination (decorporation).

(3) Reverse isolation (barrier nursing) to protect a neutropenic patient.

g. Management of significant irradiation (local and whole body).

(1) Supportive management including prophylaxis, fluids and blood replacement therapy.

(2) Definitive management including early use of cytokine (stimulation) therapy, stem cell therapy, and management of neutropenic sepsis and opportunistic infections.

(3) Psychological support and risk communication.

h. Primary surgery following decontamination but before any manifest syndromes.

i. Rehabilitation include reconstructive surgery and risk counselling.

[1] Full decontamination (external & wound decontamination) should not delay life-saving treatment and surgery. Clothing removal is recommended before entry into a MTF. Wound debridement may be the only effective method of wound decontamination and required for conventional wound management.

j. Health surveillance.

Note. A patient that is not contaminated but is irradiated only is not a radiological hazard to medical personnel.

2. In order to quantify the radiological hazards and priorities for treatment, it is necessary to assess:

a. Predicted absorbed dose from physical dosimetry.

b. Assess external contamination risk by:

(1) Identifying hot spots of external contamination.

c. Assess wound contamination and:

(1) Sample any contaminated wound.

(2) Identify any radioisotope and presence of radiological shrapnel.

(3) Determine the dose rate from any shrapnel.

d. Assess any internal contamination and:

(1) Taking nasal samples within the first hour.

(2) Identify any potential internalised radionuclide / isotope.

(3) Conduct initial radiological urinalysis.

(4) Start 24 hour urine collection, as required.

(5) Consider faecal samples if ingestion risk.

e. Assess absorbed dose using biodosimetry (see Chapter 33):

(1) Take initial full (complete) blood count sample with white cell differential.

(2) Repeat full (complete) blood count every 6 hours.

(3) Take serum biomarkers including amylase, C-reactive protein.

(4) Cytogenetics at 24 hours.

f. Monitor for skin effects including cutaneous syndrome, beta burns and local radiation injury.

3. A combined casualty with major trauma is at greatest risk from death from both the complications of trauma, and the risk of infection and coagulopathy. However, life-threatening trauma should take precedence and where possible, the 10 minute – 1 hour – 2 hour timeline set by medical doctrine should be the goal for trauma management.

4. Following a nuclear detonation, casualties may be a combination of blast, burns and high dose irradiation with risk of external and internal contamination by highly radioactive contamination. The priorities remain the same as above. However not only are military but civilian and regional healthcare systems likely to be overwhelmed. This will result in resources

being rationed and reserved for salvable cases, and the potential use of the expectant (T4) triage category based on the severity of trauma (including burns) and absorbed dose.

35.2. RADIATION PROTECTANTS AND MITIGATORS

1. Drugs given before exposure to ionising radiation, as may happen in an emergency response in the nuclear scenario are called 'radioprotectants'. Drugs given after exposure but before the appearance of manifest symptoms are called 'radiation mitigators'.

2. Possible MedCM to ionising radiation (exposure and contamination) can be categorised into the following groups:

a. Drugs that alter the absorption, distribution and elimination of internal contaminants ('decorporating agents').

b. Drugs that prevent the initial radiation injury e.g. antioxidant free radical scavengers.

c. Drugs that modify the initial biological changes triggered by irradiation, such as programmed cell death.

d. Drugs that stimulate the proliferation of surviving stem and progenitor cells e.g. cytokine stimulation therapy.

3. A summary of some of these agents is found in Annex 35C. Specific MedCM use will be directed by national guidance, authorisation and/or licensing.

35.3. TRIAGE OF RADIOLOGICAL AND COMBINED CASUALTIES

Chapter 14 provides an outline of generic triage. Radiological triage can be based on a number of factors including:

a. Conventional triage for trauma.

b. Early onset of prodromal symptoms.

c. Dose assessment based upon physical and biodosimetry.

d. Body surface area of burns.

e. Presence of torso trauma in irradiated casualties.

f. Presence of radiological shrapnel.

35.3.1. CASUALTY TRIAGE

A summary of the synergistic effects of radiation and trauma is provided in Figure 35-1 to support the initial triage before clinical investigations, and where physical dosimetry made be available. This data may be collected using Annex 33A. Further guidance for burns is found later in this chapter.

35.3.2. MASCAL TRIAGE

1. Following a potentially catastrophic event, the triage of casualties during a MASCAL event is vital for the best care for as many casualties as possible. Not only will there be an overwhelming demand for medical care but also a finite supply of specialist drugs and critical

care. This may be in the operational environment of a failure of infrastructure or lack of host nation support.

2. Potential criteria for expectant triage include:

   a. Absorbed dose of 8 Gy[2].

   b. Combined injury including the torso.

   c. Penetration of torso with highly radioactive shrapnel.

   d. Absorbed dose and body surface area of burn.

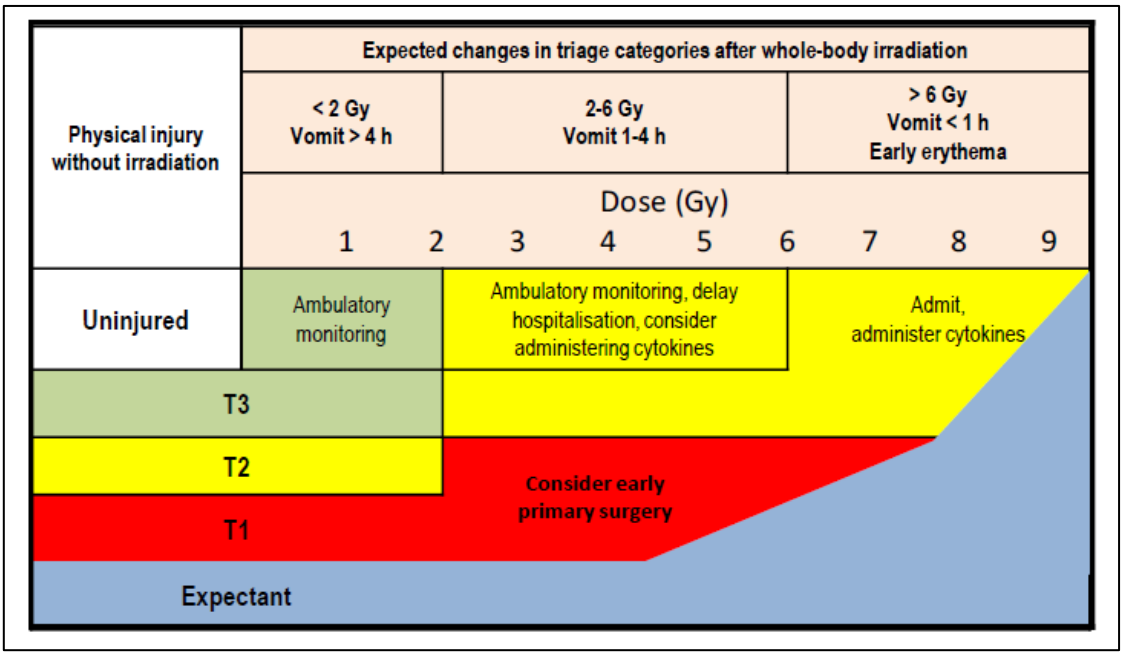

Figure 35-1: Initial Radiological Triage.[3]

35.4. RADIATION CASUALTY PATHWAY

A summary of the various considerations is at Annex 35A and described as parts of the pathway in the following sections. These parts include:

   a. Emergency Medical Treatment pathway (Figure 35-2).

   b. External and wound contamination pathway (Figure 35-3).

   c. Internal contamination pathway (Figure 35-4).

   d. Irradiation assessment and ARS pathway (Figure 35-5).

[2] The dose used as a threshold for limiting treatment and the provision of palliative care may be reduced depending on demand and resources. > 4.5-6Gy represents a less than 50% chance of survival with supportive management.

[3] Adapted from US AFRRI triage guidance. Further triage will be based on the METREPOL Response Category and this will inform the decision for cytokine therapy.

e. Non-damage control and combined injuries surgical pathway (Figure 35-6).

35.5. EMERGENCY MEDICAL TREATMENT

1. The principles of EMT described in Chapter 5 apply to a radiological and nuclear environment with the priority of managing T1 casualties with life and limb threatening conditions. For combined casualties trauma management takes precedence. Any radiation MedCM, such as anti-emetics, use should be recorded as should any assessment of dose and contamination.

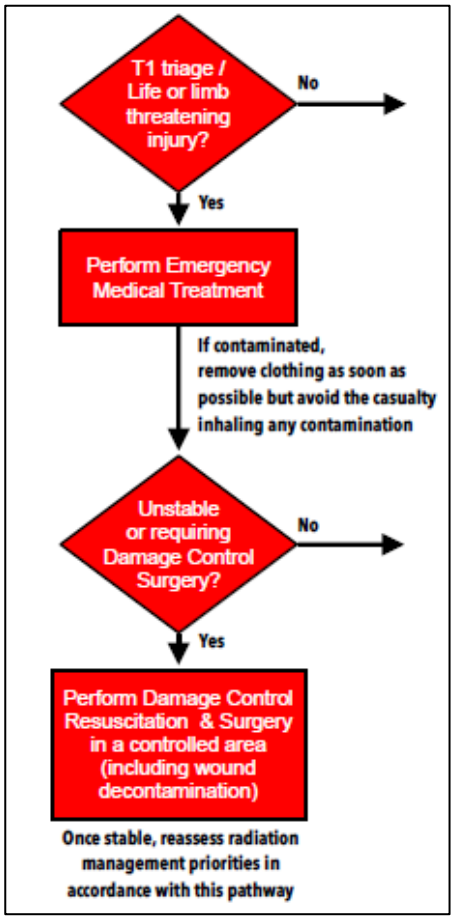

Figure 35-2: Radiation Emergency Medical Treatment.

2. Following the standardised approach, the priorities for treatment are:

a. *Catastrophic haemorrhage*. The management of catastrophic haemorrhage remains the priority and bleeding should be controlled by an initial trial of control by pressure dressing, followed as required by tourniquet application. In a contaminated environment, the use of haemostatic agents in a wound is relatively contraindicated but may be required for torso and junctional injuries.

b. *Airway*. The airway should be managed as for any CBRN environment with basic airway management. Where a respirator is not tolerated, it may be exchanged for a tight-fitting particulate face mask.

c. *Antidotes, fluids and access*. Parenteral access for drug and fluid administration is possible using both the intraosseous and intravenous route but requires a focused decontamination of the skin as recommended to prevent infection, and where possible confirmation that the skin is not significantly contaminated.

d. *Breathing, oxygen administration and chest injuries*. In a contaminated environment, oxygen may be given through nasal specula and covered with a tight fitting particulate face mask. While an oxygen mask provides higher inspired oxygen concentration, most still have an air mix during some of the inspiration phase. The management of chest injuries should also be considered with sucking chest wound being covered with a one way valve and thereby reducing both the introduction of contamination and the development of a tension pneumothorax. In the event of a tension pneumothorax being present or developing a needle decompression (thoracocentesis) is justified as a life-saving intervention even in a contaminated environment; however it is advised to decontaminate the skin before.

e. *Anti-emetics*. For intractable vomiting especially with potential airway compromise, an anti-emetic may be considered. Any use of an anti-emetic and the time of onset of vomiting should be clearly documented to support the initial dose assessment and subsequent triage. Selective serotonin (5-HT_3) antagonists are recommended and doses recommended are:

(1) *Ondansetron*: Initial adult dose (IV/IO) – 8mg followed by 4-8mg every 8 hours or 1mg/hour for the next 24 hours (oral dose – 8 mg every 8 hours).

(2) *Graniseton*: Initial adult dose (IV/IO) - 10µg/kg with oral dose of 1-2mg (every 12-24 hours – max dose 2mg per day).

35.5.1. UNSTABLE TRAUMA PATIENTS

1. For unstable trauma casualties immediate life-saving surgery in a controlled and monitored clinical environment may be required preceded by the removal of all clothing if there is a risk of contamination. Damage control surgery should not be delayed as surgical protective equipment will protect medical staff from most forms of radiological contamination on a live casualty. Any high dose rate fragments should however be identified and risk quantified.

2. Further details are given in the surgery section later in this chapter.

35.6. MANAGEMENT OF EXTERNAL AND WOUND DECONTAMINATION

The practical decontamination of externally contaminated patients is easily accomplished with 90-95% of the decontamination accomplished by simply removing the outer clothing and shoes and simple washing. This can generally be achieved before admission without interfering with medical treatment. Before removing clothing, if there is a risk of re-aerosolisation then casualties should be provided with face masks.

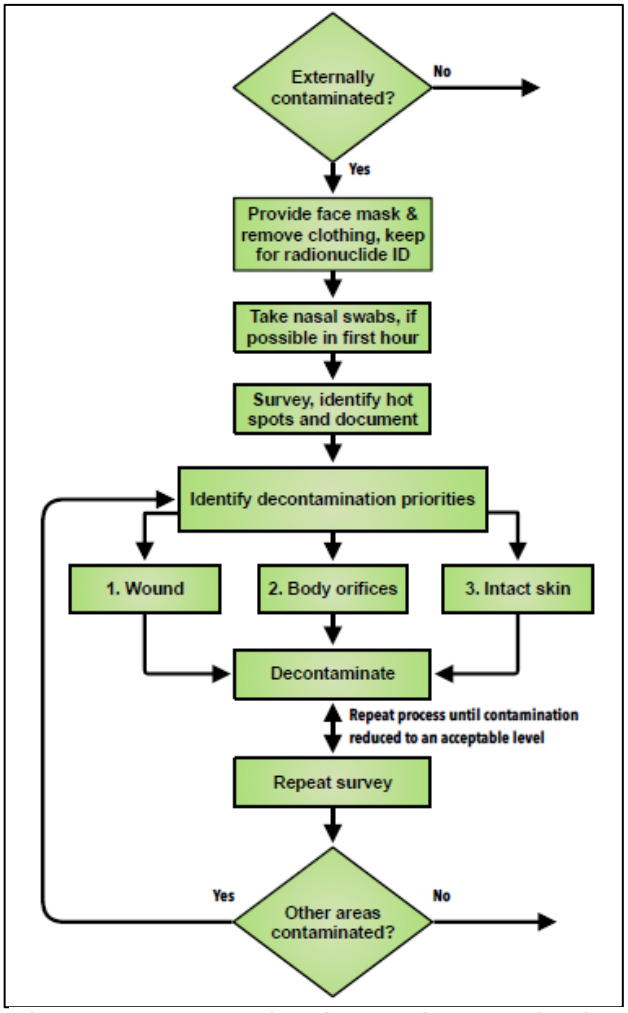

Figure 35-2: External and Wound Contamination Considerations.

35.6.1. DECONTAMINATION OF WOUNDS

The initial priority is haemostasis but other objectives include wound irrigation and potentially formal surgical debridement. Wound irrigation may be with water or crystalloid fluids but may include agents used for decorporation such as DTPA for americium or plutonium.

35.6.2. DECONTAMINATION OF FACE AND ORO-NASAL (BODY) ORIFICES

Nasal swabs should be taken as soon as possible, and ideally within the first hour following exposure. Nasal swabs can be used to estimate the internal dose burden based on the counts per second, radioisotope and radiation monitor efficacy. Health physics advice is recommended to assist with the calculation and dose estimation.

35.6.3. DECONTAMINATION OF INTACT SKIN

1. Once removed, contaminated clothing can be placed in bags, tagged and removed to a remote section of the medical facility to avoid creating a hazard due to concentration of such contamination. The contaminated clothing may also be used to identify any radioisotope using gamma-spectroscopy especially where internal contamination may have taken place and decorporation is being considered. The clothing can be decontaminated or disposed of by qualified personnel as time permits.

1. The second phase of decontamination consists of rewashing and wiping the casualty's face and hands and any other areas that may have been exposed. This should leave the casualty significantly decontaminated. This simple task can be accomplished prior to admission, later on the ward, or elsewhere in the medical facility as the situation dictates.

2. The third phase of decontamination consists of washing the hair or clipping the hair and washing the scalp. The third phase need only be accomplished if the casualty arrives without headgear or monitoring indicates that the hair is significantly contaminated.

3. Unprotected and externally contaminated casualties should be evaluated for internal contamination. Nasal swab specimens should be collected (as described before) before facial decontamination. Baseline stool and urine samples should be collected and analysed, or sent to reference laboratories. Personnel in intact IPE / PPE do not require this screening.

35.7. MANAGEMENT OF INTERNAL CONTAMINATION

1. The requirement for the treatment of persons with internal radionuclides is to reduce the absorbed radiation dose and hence the risk of possible future biological effects. The methods to mitigate internal contamination include:

a. The reduction of initial absorption and internal deposition;

b. The blocking of the uptake of a radioisotope by a stable isotope of the same nuclide;

c. The enhanced excretion of absorbed contaminants by redistribution (mobilising) and chelation; and

d. Physical removal, including gut decontamination methods, wound irrigation and debridement, and pulmonary lavage.

Note. Most of the techniques will exploit the radionuclide's physical or chemical properties and not the radiological properties.

2. MedCM that interfere with absorption and internal deposition, including blocking and chelation agents, are less effective when the radionuclide has already entered the target tissue(s). For relatively low risk procedures, the benefits of rapid administration should outweigh the absolute quantification of the contamination or dose burden. Treatment should be initiated as soon as practical when the suspected internal contamination is judged to be significant. The decision to stop therapy is not as crucial as the decision to initiate treatment and can be made after further assessment, including samples and reach back advice.

3. A list of decorporating agent(s) for each significant radionuclide is at Annex 35D.

35.7.1. REDUCING ABSORPTION

1. Gastrointestinal absorption of radioactive substances should be minimised if possible and practical. Gut decontamination is used widely following the ingestion of toxic substances. However, there use is limited if the contaminant is rapidly absorbed. Methods of gut decontamination include:

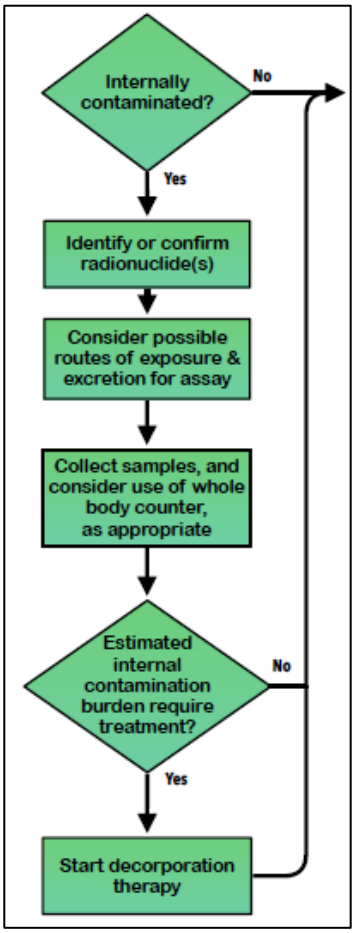

Figure 35-3: Internal Decontamination Considerations.

a. *Gastric lavage*. Gastric lavage has been used to empty the stomach after the ingestion of poisonous or radioactive materials although recent research has questioned their efficacy, and safety especially in an unconscious casualty. There is a risk of aspiration and use for radiological ingestions is not recommended.

b. *Emetics*. Emetics are contraindicated if the consciousness is impaired and after the ingestion of corrosive agents or petroleum hydrocarbons. The commonly used emetics are apomorphine and syrup of ipecac.

c. *Purgatives*. Purgatives or laxatives are widely used and generally safe. They include irritants, bulk-forming substances, lubricants and wetting agents. Purgatives used in bowel preparation for colonoscopy are used for whole bowel irrigation in toxic ingestions, especially slow release preparations. This method is not specific to the chemical properties of the radionuclide. It is rapid acting and decreases the transit time of slow or non-absorbable contaminants.

d. *Ion-exchange resins*. The use of ion exchange resins has been largely limited to decreasing gastrointestinal uptake of ingested or inhaled radionuclides. Even this use has been limited mainly to experimental tests in animals. Ferric ferrocyanide (Prussian Blue) and alginates have been used in man to accelerate faecal excretion of ^{137}Cs and are given orally shortly after ingestion of the radionuclide. Aluminium-containing antacids are effective agents for reducing intestinal uptake of radioactive strontium.

35.7.2. STABLE IODINE BLOCKADE

1. Radioactive iodine is a normal fission product following the fission of uranium or plutonium. The radioactive emissions are beta particles accompanied by multiple gamma emissions. ^{131}I (iodine), ^{132}I, ^{134}I, and ^{135}I will be found after nuclear detonations, reactor accidents and destruction of a nuclear reactor by hostile forces. Of these, only ^{131}I will pose a hazard to personnel after the first 2 days due to the short half-lives of the radioisotopes of iodine.

2. *Medical effects of radioiodine*. The primary target of injury for radioiodine is the thyroid gland. Thyroid uptake concentrates the radioiodine and allows local irradiation. Direct high-dose irradiation injury from absorbed radioiodine may cause acute thyroiditis, hypothyroidism and parathyroid damage similar to therapeutic thyroid ablation. There is significant risk of

thyroid carcinoma and benign nodules, both considered stochastic effects, even at lower levels in at risk populations. Medical planning and automatic MedCM guidance must include the prevention of both deterministic and stochastic effects from radioiodine both locally and within regional populations exposed to any residual fallout.

3. *Stable iodine*. Stable iodine, if given early enough, will saturate the thyroid gland with stable iodine (^{127}I) and prevent any further radioactive iodine to enter the thyroid. Stable iodine is usually given as potassium iodide or potassium iodate (see Table 35-1 for stable iodine dosing). Stable iodine should be given before or within 4 hours of exposure. A single dose will protect for 24 hours and should be sufficient unless prolonged exposure is expected. This protective effect may be especially important in communities with low-iodine diets.

Table 35-1: Stable Iodine Recommended Doses (usually as a single daily dose).

Population group and thyroid exposure risk	Daily iodine Equivalent	Potassium Iodine	Potassium iodate 85 mg tablets
Adults > 40 y of age (with thyroid exposure ≥ 5 Gy)	100mg	130 mg / day	2 tablets / day
Adults 18 – 40 y of age (with thyroid exposure ≥ 0.1 Gy)	100mg	130 mg / day	2 tablets / day
Pregnant or lactating women (with thyroid exposure ≥ 0.05 Gy)	100mg	130 mg / day	2 tablets / day
Children and adolescents 3 – 18 y of age (with thyroid exposure ≥ 0.05 Gy)	50mg	65 mg / day	1 tablet / day
Infants 1 month – 3 y of age (with thyroid exposure ≥ 0.05 Gy)	25mg	32 mg / day	½ tablet / day
Neonates from birth – 1 month of age (with thyroid exposure ≥ 0.05 Gy)	12.5mg	16 mg / day	¼ tablet / day

4. The use of stable iodine is relatively safe but its implementation may be applied to different populations at risk:

a. *Paediatric population*. Infants and children are most susceptible to the effects of radioiodine. Information on thyroid cancer among exposed children and on the risks of side-effects from stable iodine indicate that thyroid dose aversion may best be achieved by stable iodine prophylaxis. A significant risk of thyroid cancer following radioiodine exposure is considered up to 40 years of age.[4]

b. *Pregnant mothers*. Pregnancy causes an increase in thyroid gland activity and will result in an increased uptake of radioiodine. The foetal thyroid gland is extremely sensitive to radioiodine, but prolonged administration of stable iodine will result in blocking the foetal thyroid function during critical brain development. Maternal overdosage with iodine must be avoided, but adequate short-term administration of iodine is justified. Pregnant women and neonates should therefore be removed from the contaminated area as quickly as possible.

c. *Over 40 years of age*. For > 40 years of age population, there are less protective benefits and a greater risk from an adverse reaction, unless a high dose exposure (>5

[4] An increased incidence of childhood thyroid carcinoma was documented following the Chernobyl reactor accident.

Gy thyroid dose) is suspected. The internal dose is unlikely to be quantified early enough to make an informed decision – adult's absorbed radioiodine dose exceeds a level sufficient to cause significant injury (>5Gy). Any benefit may be offset by side-effects from pharmacological iodine administration especially with a history or undiagnosed thyroid disease. Where the risk is unknown, iodine prophylaxis is probably limited to a single dose until further assessment has been made. Protracted usage (i.e. 2-3 weeks) is not recommended due to potential drug-induced toxicities.

d. *Iodine allergy*. Caution is recommended before stable iodine administration to those with known severe adverse reactions to iodine. Other methods of dose avoidance or reduction should be used to prevent internalisation of radioiodine or enhanced medical screening if exposure is suspected in the absence of stable iodine use especially if < 40 years of age.

35.7.3. ENHANCED EXCRETION

1. The excretion of radionuclides can be increased either by:

a. *Redistribution (mobilising)*. Redistribtuion mobilises the radioisotope back into the systemic circulation that allows excretion to take place usually by the kidneys. This may be achieved by shifting the equilibrium with a stable isotope of the same nuclide or a nuclide with similar biochemical distribution. In cases of exposure to tritium, such as tritiated water, it is possible to increase its excretion by increasing oral fluids and possible use of a diuretic. These *mobilising agents* are more effective the sooner they are given after the exposure to the isotope. The mechanism is a dynamic process and differs to the saturation method used for blocking radioiodine with stable iodine.

b. *Chelation (chemical-binding).* Chelating agents may be used to bind metals in the systemic circulation. Once bound, they become biochemically inert and are usually excreted by the kidneys. Many of the chelating agents are also used for the treatment of metal poisoning.

2. A list of mobilising and chelating agents is found in Annex 35B with more details in Annex 35C.

35.7.4. PULMONARY LAVAGE

Physical removal methods can be used to remove some forms of contamination, especially low-solubility and/or chemically inert. Pulmonary lavage may be considered following inhalation of radioactive contaminants such as plutonium. This procedure is not an emergency and should only be undertaken after evacuation, complete assessment of the dose burden and other health risks and under controlled conditions. A formal risk assessment should be conducted and include:

a. *Benefit of pulmonary lavage*. Extensive studies with plutonium indicate that this procedure can remove over 50% of contaminants and can be performed with equal efficacy at any time during the first 14 days post inhalation. Risk of a radiogenic reactive pulmonary oedema or fibrosis are indications for pulmonary lavage.

b. *Risks*. The overall risk assessment includes both that of the procedure itself and that from the lifetime accumulated dose. Pulmonary lavage should be reserved for those patients with known significant body burden. The procedure itself comes with a

significant risk of complications including death and should only be performed after formal risk assessment and by an experienced practitioner.

35.8. MANAGEMENT OF ACUTE RADIATION SYNDROME

1. Deterministic radiation damage results from the inherent sensitivity of certain cell types to radiation, with the most undifferentiated and mitotically active cells being the most sensitive to acute effects. The inherent sensitivity of these cells results in a combination of clinical sub-syndromes that occur with radiation exposure above a threshold of 2Gy.

2. The clinical components of ARS include hematopoietic (H), gastrointestinal (G), and neurovascular (N) and cutaneous (C) sub-syndromes; the latter component being more complex (see Chapter 32). NHCG organ specific grading (from 1 to 4) is the basis of METREPOL Response Category accounting for radiation injury level. This assessment is more practical that dose estimate alone, that cannot be accurately known until after 3-4 days using cytogenetics.

3. The medical management of patients with potential acute moderate to severe radiation exposure (effective whole body dose > 2-3 Gy) should emphasise:

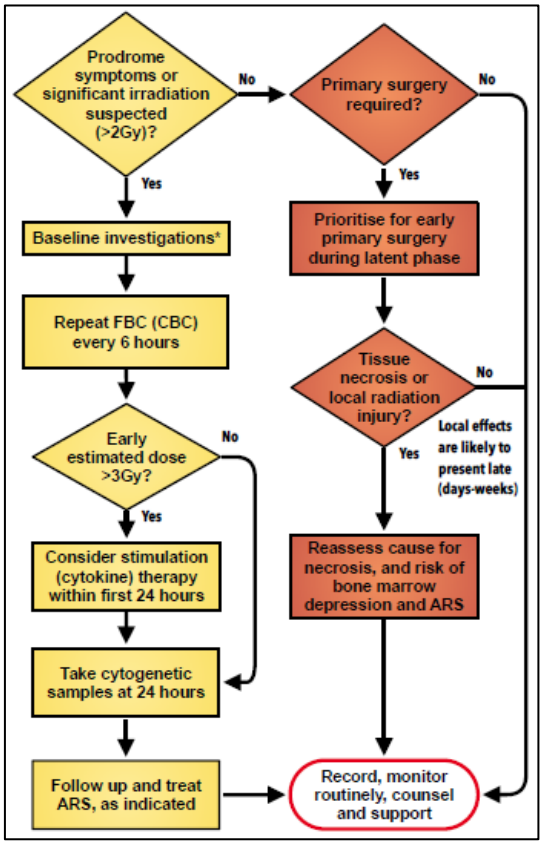

Figure 35-4: Management of Significant Irradiation and Primary Surgery.

a. Assessing absorbed dose (see Chapter 33) with an early dose estimation within 24 hours.

b. Early cytokine (CSF stimulation) therapy, as indicated, within 24 hours.

c. Formal dose assessment using cytogenetics and clinical prediction.

d. Supportive (and prophylactic) management of whole body irradiation.

e. Definitive management of whole body irradiation.

(1) Stem cell transplant.

(2) Bone marrow transplant.

f. Early primary surgery. In the case of co-existing trauma (combined injury) primary surgery should be performed within 36-48 hours due to the increasing risks from infection, coagulopathy and delayed healing (see below).

g. Manage local radiation injury (see below).

4. The merits of modern supportive care lie in its significant prolongation of survival. The $LD_{50/60}$ is approximately 3.5 Gy in persons managed without supportive care. The $LD_{50/60}$ may

be increased to 4-5 Gy when antibiotics and replacement therapy are provided. The lethal dose may also be somewhat higher with early initiation of colony stimulating factors (CSFs). Casualties whose radiation dose is most amenable to treatment will be those who receive between 3 and 6 Gy (H3-H4 score using the METREPOL system). The primary goal of medical therapy is to shift the survival curve to the right by 2 Gy or more.

5. Following a nuclear or 'dirty bomb' scenario, many casualties with doses exceeding 6 to 8 Gy will also have significant blast and/or thermal injuries that will preclude survival when combined with their radiation insult.

35.8.1. INITIAL ASSESSMENT OF IRRADIATED CASUALTIES

1. The initial assessment of an irradiated casualty includes:

   a. History and location to event.

   b. Physical dosimetry, if available.

   c. Timing and severity of prodromal symptoms.

   d. Serial full (complete) blood counts with differential, repeated every 6-12 hours.

   e. Serum biomarkers (see below).

   f. Samples for future HLA tissue typing taken 12-24 hours (see stem cell therapy).

   g. Cytogenetics at 24 hours.

2. When the irradiated patient is first evaluated, the following labs are important to acquire as time permits:

   a. Initial clinical investigations:

      (1) Full (complete) blood count with differential (baseline) and repeat full (complete) blood count every 6-12 hours – to evaluate lymphocyte kinetics and to calculate the neutrophil/lymphocyte ratio.

      (2) Serum amylase and C-reactive protein (baseline) and every 24 hours as there is a dose-dependent increase in amylase after 24 hours. Both are widely available in most deployed field hospitals with diagnostic laboratory capabilities.

   b. Important clinical investigations (where available):

      (1) Blood FLT-3 ligand levels – Marker for hematopoietic damage.

      (2) Blood citrulline – Decreasing citrulline indicates GI damage.

      (3) Interleukin-6 (IL-6) – Blood marker increased at higher radiation dose.

      (4) Quantitative G-CSF – Blood marker increased at higher radiation dose.

      (5) Cytogenetic studies at 24 hours to establish accurate whole body dose using lymphocyte dicentric analysis and over dispersion index to evaluate for partial-body exposure.

35.8.2. ESTIMATION OF DOSE BASED ON INITIAL ASSESSMENT

1. The severity of the ARS is directly proportional to the severity of the radiological component of the incident. In order to gauge the severity of an incident, the radiation dose to a casualty can be estimated early post-event using rapid-sort, automated biodosimetry (where available) and clinical parameters such as the onset of various clinical symptom complexes, the time to emesis (TE), lymphocyte depletion kinetics, and through combinations of various biochemical markers. No single technique is satisfactorily sensitive, but multiparametric techniques have been shown to have good predictive value.

2. The estimation must be made in the first 24 hours to inform the decision to start early cytokine therapy, prioritise for surgery, start supportive and prophylactic regimes in anticipation of immunosuppression, and also early STRAT MEDEVAC.

3. Guidance for early dose estimation include:

a. *Low risk (<1 Gy) casualties*. For external dose D<1 Gy, generally the patient is asymptomatic and blood parameters will be within the normal range. Upon admission to emergency care post-incident, it is always appropriate to obtain a full (complete) blood count with differential (FCBC), either as a baseline level or as a beginning step for lymphocyte kinetic analysis.

b. *Risk based on time to emesis*. TE, measured from the irradiating event, decreases generally with increasing dose. For 1 h < TE < 2 h, the effective whole-body dose is likely at least 3-4 Gy. If TE < 1 h, the whole body dose likely exceeds 4-6 Gy. In a mass-casualty tactical event, patients who experience emesis less than four hours post-accident should be triaged to professional medical care while those with emesis greater than four hours can be instructed to receive delayed medical attention. Casualties who experience radiation-induced emesis within one hour after a radiation incident will require extensive and prolonged medical intervention, and a poor outcome will occur in many instances.

c. *Dynamic leucocyte profile*. An additional parameter from serial haematology assessments is the neutrophil / lymphocyte (N/L) ratio. This provides a comparison using the relatively rapid fall in lymphocyte compared to neutrophils. This has been incorporated into some of the biodosimetry toolkits.

4. The patient history, physical exam and early estimate of the severity of the radiation incident may be rapidly analysed using multiple clinical and dosimetry parameters into a clinically meaningful estimate of radiation exposure using tools and resources such as:

a. The Armed Forces Radiobiology Research Institute (AFRRI) Biodosimetry Assessment Tool (BAT) and mobile First Responder Assessment Tool (mFRAT) software packages (www.afrri.usuhs.mil/).

b. The REMM website (http://www.remm.nlm.gov/) developed by the Department of Health and Human Services (DHHS), National Cancer Institute (NCI) and the National Library of Medicine (NLM) is a very important resource in patient management.

c. The Centers for Disease Control have a useful compendium of radiation medicine information and protocols (http://www.bt.cdc.gov/radiation/).

d. Medical Management of Radiation Accidents: Management of the Acute Radiation Syndrome. The British Institute of Radiology. (https://www.clintox.org/documents/radSIG/RAD-SIG-Euro-med-mngt.pdf).

35.8.3. EARLY STIMULATION (CYTOKINE) THERAPY

1. Following total body irradiation, there is a window of opportunity to stimulate surviving granulocyte progenitor cells before significant bone marrow depression. Recent studies[5] show efficacy to mitigate lethality in the hematopoietic sub-syndrome (ARS-H) is dependent on the interval between irradiation and administration of the MedCM.[6,7] It is recommended that granulocyte-colony stimulating factor (G-CSF) is given within 24 hours of the initial radiation exposure.

2. *Cytokine (CSF) MedCM*. Currently, the hematopoietic cytokines available in some NATO countries include those used to treat oncology and haematology patients as well as licensing for radiation casualties. The recombinant forms and dosing include:

a. Filgrastim (G–CSF) 2.5-5 µg/kg/d subcutaneously once daily.

b. Sargramostim (GM–CSF) 5-10 µg/kg/d subcutaneously once daily.

c. Pegfilgrastim (pegylated G-CSF) 6 mg once subcutaneously.

3. *Indications for CSF use*. The indications for cytokine early therapy includes:

a. Estimated acute radiation dose of ≥ 3Gy (or 2Gy in children, or combined injury).

b. Within 24 hours.

4. *Continuation of therapy*. Treatment should be continued until there is a sustained absolute neutrophil count > 1000 x 10^6 cells/L.

5. *Complications of cytokine therapy*. CSFs have been associated with rare splenic rupture and, more commonly, bone pain.

6. Further details on stimulation therapy and current NATO research can be found in Annex 35C.

35.8.4. SUPPORTIVE MANAGEMENT

1. The supportive management of the irradiated patient includes the following considerations:

a. *Antibacterial, antiviral and antifungal prophylaxis, and treatment of febrile neutropenia.* In non-neutropenic patients, antibiotics should be directed toward the foci of infection and the most likely pathogens. For those who experience significant neutropenia (absolute neutrophil count, ANC <500 cells/mm^3), broad-spectrum

[5] Filgrastim (10 µg/kg/d) administered beginning 1 day after TBI (7.5 Gy; LD50/60) and continued daily until absolute neutrophil count was > 1000/µL for three consecutive days significantly increased survival by 38% over controls. By contrast, filgrastim initiated 48 hours after irradiation, did not improve survival.
[6] Farese AM, Cohen MV et al. Filgrastim improves survival in lethally irradiated non-human primates. Radiat Res. 2013;179:89-100.
[7] Farese AM, Brown CR et al. The ability of filagrastim to mitigate mortality following LD50/60 total body irradiation in administration time-dependent. Health Phys. 2014;106:39-47.

prophylactic antimicrobials should be given during the potentially long duration of neutropenia. Prophylaxis should include a fluroquinolone, an antiviral agent (if indicated, as discussed below), and an antifungal agent. The justification for fluroquinolone (FQ) prophylaxis includes pre-clinical and clinical studies demonstrating decreased infectious episodes in irradiated animals and neutropenic oncology patients, respectively. Streptococcal coverage with the addition of penicillin or amoxicillin should also be considered, if not inherently covered by the FQ, given the increased treatment failure observed due to this pathogen and the benefit demonstrated with expanded anti-streptococcal coverage in neutropenic animals. Current antimicrobials should be continued until there is an indication to change due to sepsis or neutrophil recovery (ANC >500 cells/mm^3). Early microbiological consultation is advised and local regimens may vary.

b. *Management of neutropenic sepsis and opportunistic infections*. In many cases of neutropenic sepsis, a patient will not exhibit the normal physiological derangement seen in sepsis. This is also with a background of organ dysfunction and potentially combined injury. Clinicians should have a very low-threshold for treating this life-threatening emergency and support the decision making with periodical septic screening for bacteraemia and supporting laboratory diagnostics. For patients who experience first fever, traditionally the FQ is stopped and therapy directed at gram-negative bacteria (in particular, *Pseudomonas aeruginosa*) as infections of this type may be rapidly lethal. Anti-pseudomonal coverage serves as the foundation antibiotic and additional coverage is then added to address other foci of infection such as mucosal or integument injury. Empiric therapy of patients with neutropenic sepsis with or without a focus of infection, or other unusual signs suggesting an opportunistic infection, should be guided by the current recommendations of the responsible microbiologist and local guidelines based on prevalence and resistance patterns. Any focus of infection that develops during the neutropenic period will require a full course of therapy.

c. *Fluid and electrolytes replacement*. Radiological casualties are at risk of fluid loss and electrolyte imbalance for a number of reasons including vomiting, diarrhoea, burns and concurrent trauma. Careful fluid balance should be documented.

d. *Early oral feeding*. Physiological interventions include maintenance of gastric acidity; avoidance of antacids and H_2 blockers; use of sucralfate for stress ulcer prophylaxis when indicated to reduce gastric colonization and pneumonia. Early oral enteral feeding is highly desirable when feasible.

e. *Transfusion support*. Transfusion of cellular components such as packed red blood cells and platelets are required for patients with severe bone marrow damage and are an important component of the clinical management. Fortunately, this complication does not typically occur for 2-4 weeks after the exposure unless losses from concurrent trauma are present. All cellular products must be leucocyte depleted and irradiated to prevent transfusion-associated graft-versus-host disease in the immunosuppressed patients.

f. *Reverse barrier nursing*. ARS patients with whole-body dose > 2-3 Gy should be in isolation rooms. Medical personnel should also be aware of the need for rigorous environmental control, including possibly laminar flow isolation, strict hand washing, surgical scrubs and masks for staff.

g. *Minimising invasive procedures*. These should be minimise so as to reduce the risk of introducing infection especially hospital associated. Lines should be taken out as soon as possible. Any signs of febrile illness should be managed aggressively and all lines should be considered as possible foci for infection.

h. *Other considerations*. Povidone-iodine or chlorhexidine for skin disinfection and shampoo, as well as meticulous oral hygiene. Additional supportive medications may include anti-emetics & anti-diarrheals.

35.9. STEM CELL AND BONE MARROW TRANSPLANT

1. At doses >6Gy without trauma, stem cell transplantation therapy can be considered. This is especially for where there is no sparing of bone marrow due to uniform distribution of whole body irradiation. However, most of the documented accidental irradiation have been characterised as non-uniform in dose distribution, and spontaneous hematopoietic recovery was often observed following bone marrow transplantation.

2. To support this HLA typing assays should be taken within the first 24 hours.

3. Allogenic stem cell transplantation may have limited usage due to severe concurrent non-hematopoietic syndromes sustained at bone marrow-lethal doses of radiation.

4. Stem cell and bone marrow transplant are considered as advanced home-based delivered care and outside the remit of this publication.

35.10. SURGICAL MANAGEMENT OF COMBINED CASUALTIES

1. The management of trauma patients can be divided into:

a. Damage control surgery, requiring urgent surgical interventions to stop bleeding and to save life and limb. This should be delivered within 2 hours from time of wounding or as soon as possible.

b. Primary surgery should be delivered within 4 hours and covers a broad number of procedures with the aim to preserve function and prevent infection.

c. Minor procedures, such as small wound wash out and dressing.

d. Reconstructive surgery including skin grafts.

2. The priorities for surgery will initially be based on surgical triage but will later take into account estimated dose either to prioritise for primary surgery or to defer. In extreme circumstances, combined ARS and significant trauma or extensive burns (>30%) might justify the use of the expectant triage category (see Figure 35-2).

35.10.1 DAMAGE CONTROL SURGERY

For damage control surgery, it is unlikely that accurate dose estimation will be available in the first 2 hours and therefore the priority for surgery is based on surgical need and resources. The main radiological concerns will be external and wound contamination, and the presence of any high dose radiological shrapnel. Damage control surgery must not be significantly delayed due to the presence of low dose contamination. However surgery should be in a controlled area allowing for monitoring of personnel and equipment, as well as surgical waste.

35.10.2. RADIOLOGICAL RISK TO SURGICAL TEAM

1. There is a theoretical risk that contaminated wounds (shrapnel or impregnated clothing) may pose a threat to a surgeon team. Any radiological hazard is quantifiable with monitoring equipment.

2. For light contamination, the risk is minimal and surgical gowns and face protection will provide adequate protection. All dressings may be contaminated and should be removed safely. Standard surgical procedures including the use of aseptic technique and surgical instruments, such as forceps, will minimise any contact risk. Conventional wound debridement (standard military surgery practice) is recommended for all contaminated wounds. Irrigation where it is used should be controlled with effluent being collected by suction and contained. Amputation should not be considered purely on the basis of reducing contamination burden.

35.10.3. RADIOLOGICAL SHRAPNEL

1. For radiological shrapnel, the risk including a dose rate at one metre surgical distance can be quantified. This provides a time and distance calculation to allow the surgeon to perform a procedure within regulatory limits for occupational exposures.

2. For a high dose rate shrapnel fragment that poses a significant risk to the surgeon, it is highly likely that the fragment during the pre-surgical period will have caused significant local radiation injury. In these extreme circumstances, amputation might be considered to reduce the time of the procedure and to remove a non-viable limb. Any radiological shrapnel should be removed as soon as possible and safety disposed of in a shielded container. For embedded high dose rate fragments within the torso, such injuries are likely to be lethal due to deep tissue damage and vascular necrosis.

35.10.4. LOW DOSE (<1 Gy) WHOLE BODY IRRADIATION SURGICAL CASES

Casualties requiring surgery with an estimated dose of less than 1 Gy should be treated using normal surgical priorities. An accurate dose estimation using cytogenetics, where available i.e. by reach back, would provide an accurate estimation in order to plan primary.

35.10.5. HIGH DOSE (> 2 Gy) WHOLE BODY IRRADIATION SURGICAL CASES

1. Combined casualties likely to have ARS should be triaged for early surgery before the syndrome manifests itself as coagulopathy and immunosuppression with concurrent bacteraemia. This may also determine the decision of type of surgery balancing the use of internal fixation, skin traction and external fixation for limb fracture management as well as early skin closure.

2. In some cases, surgical procedures such as reconstruction surgery may be deferred until the casualty has survived the manifest syndromes of ARS and there is restoration of bone marrow function with or without CSF therapy.

35.10.6. BURNS AND RADIATION

1. Studies demonstrate that the mortality of thermal burns markedly increases with irradiation. Burns with 50% mortality may be increased >90% by concurrent radiation doses as small as 1.5Gy. Triage of combined injuries with burns may use radiation dose and body surface area of burn (see Figure 35-2).

2. Aggressive damage control resuscitation may diminish this effect. The primary treatment of significant radiation injury includes reduction or elimination of the foci of infection. Medical personnel must consider early wound and surgical management as required. Full-thickness burns are ideal bacterial culture media and excision of these burns may be indicated to reduce the risk of infection. Indications for escharotomy, a life and limb threatening condition, remain the same.

3. Limited information regarding the use of modern skin graft techniques in irradiation injury victims is available. Use of topical antimicrobials should be considered whose side-effects include leukopenia may be a complicating factor in radiation-immunocompromised patients. No data are available regarding the response to clostridial infection and strong consideration should be made as to the use of passive tetanus immunisation even in previously immunised patients. Patients whose burns are contaminated by radioactive material should be gently decontaminated to minimise absorption through the burned skin. Most radiation contaminants will remain in the burn eschar when it sloughs.

Table 35-2: Priority of Combined Injuries (Burns).[8]

		Burn (% body surface area)		
		I < 10%	II 10-30%	III > 30%
Radiation dose (Gy)	I < 2	Minor	Moderate*	Severe
	II 2 - 4	Moderate*	Moderate*	Severe
	III > 4	Severe	Severe	Severe

Triage considerations

Unless beta burns, minor cases should be managed as conventional burns, with appropriate radiation monitoring and follow up for < 2 Gy.

All burns should be monitored for complications including local irradiation injury and deep tissue necrosis, delayed healing and keloid scarring.

During a MASCAL, limited resources may be more appropriate to be diverted to moderate cases with greater survivability or the B(III) criteria is increased to 50%. Cases with > 50% BSA would either be treated after the other cases or as expectantly (T4).

* Any B (II) or R(II) with injuries requiring thoracotomy, laparotomy, craniotomy or major amputation would also be considered severe. Minor cases would be prioritised in accordance with the conventional priority for the surgery.

34.10.7. RADIATION AND WOUND MANAGEMENT

1. Where injuries threaten life, trauma management takes precedence over radiological management. Dose reduction by decontamination, even if limited to the removal of clothing, and removal of amenable shrapnel should be attempted. Damage control resuscitation and surgery is possible even in the presence of contamination but casualties should be screened for any high dose rate fragments and managed by early removal or shielding until safe to remove surgically.

2. Wounds that are left open and allowed to heal by secondary intention will serve as potentially fatal foci of infection in the irradiated patient. If possible, wounds should be closed primarily as early as possible. In irradiated patients, all surgeries should be completed within 36 hours of radiation injury. Extensive debridement of wounds may not allow this to be initially

[8] Adapted from Kumar P, Jagetia GC. A review of triage and management of burns victims following a nuclear disaster. Burns. 1994; 20:397-402.

possible. Conventional combat wounds are not closed primarily due to the high levels of contamination and devitalised tissue.

35.11. MANAGEMENT OF THE COMPLICATIONS OF ACUTE RADIATION SYNDROME

Immune reconstitution inflammatory syndrome (IRIS, also known as immune recovery syndrome) is a condition seen in some cases of acquired immune deficiency syndrome (AIDS) or immunosuppression, in which the immune system begins to recover, but then responds to a previously acquired opportunistic infection with an overwhelming inflammatory response that paradoxically makes the symptoms of infection worse. There are two common IRIS scenarios. The first is the “unmasking” of an occult opportunistic infection. The second is the paradoxical symptomatic relapse of a prior infection despite microbiologic treatment success. Often in paradoxical IRIS, microbiologic cultures are sterile. In either scenario, there is hypothesised reconstitution of antigen-specific T cell-mediated immunity with activation of the immune system. Infections most commonly associated with IRIS include cytomegalovirus, herpes zoster, Mycobacterium avium complex, Pneumocystis pneumonia, and Mycobacterium tuberculosis.

35.12. POST OPERATIONAL FOLLOW UP

Role 4 management of ARS is largely supportive. Further guidance may be found in the TMT Handbook developed by the EU (www.tmthandbook.org).

In addition to the management of above, cancer screening and ophthalmologic evaluation for cataracts may be important.

Any casualty exposed to ionising radiation should also be offered counselling and be informed of the health risks associated with a significant exposure. All exposures and dosimetry should be recorded in the health or occupational record.

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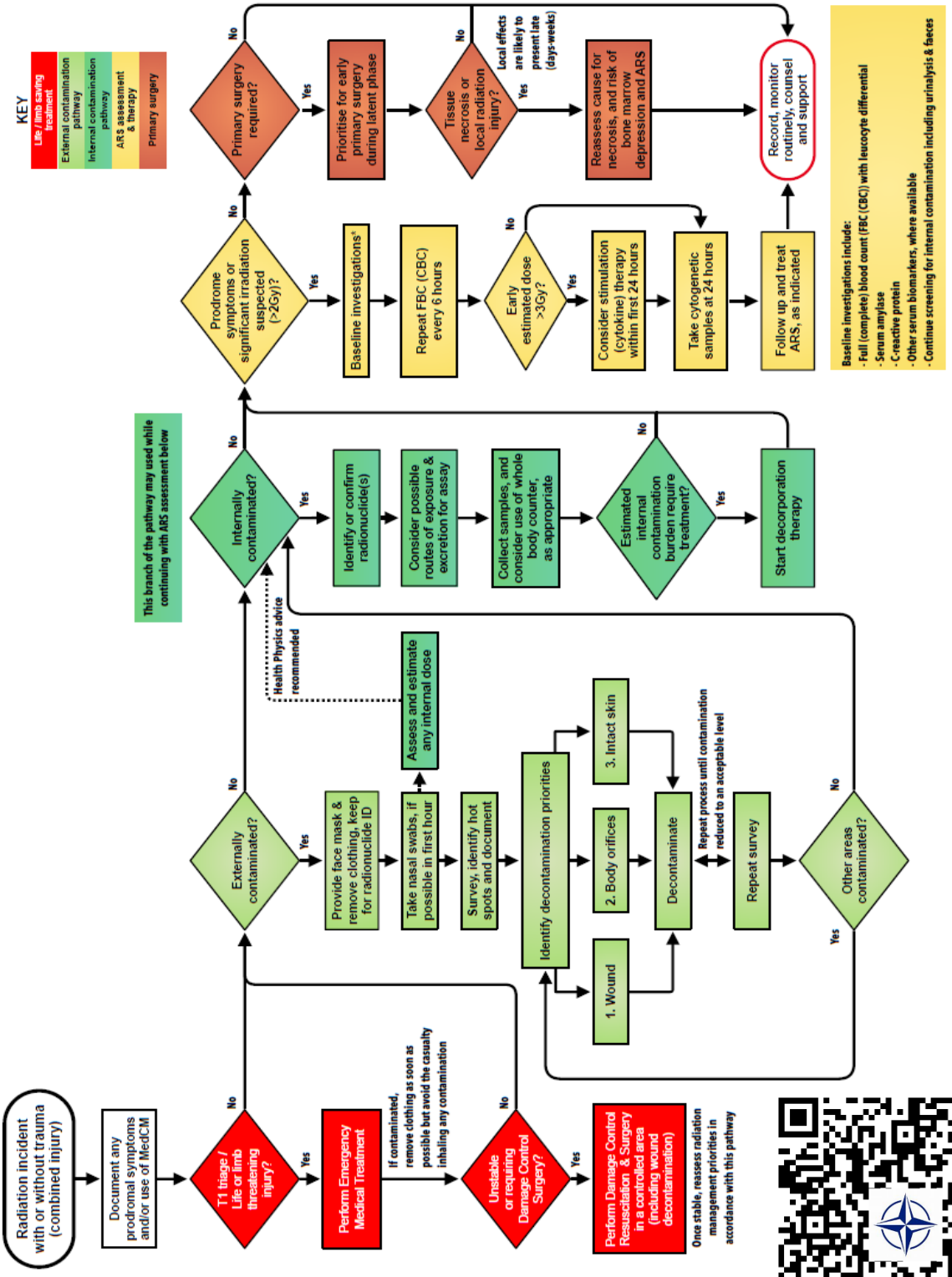
ANNEX 35A – RADIATION CASUALTY PATHWAY



Adapted from REAC/TS (www.orise.orau.gov/reacts)

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ANNEX 35B – SUMMARY OF DECORPORATING AGENTS

Recommendation for decorporation therapy for specific radionuclides and drug information for treatment. Table below is based on NCRP 161 (2009), which is widely considered an important reference USA document for decorporation therapy in patients with internal deposition of radionuclides. A summary and treatment guidelines are provided in Annex 35E for the more common indications and preferred treatment.

Note. Before starting decorporation therapy seek specialist radiation medicine advice.

Radionuclide	Treatment (*preferred treatment)
Actinium (Ac)	Consider diethylenetriaminepenta acetic acid (DTPA)
Americium (Am)	DTPA
Antimony (Sb)	British Anti-Lewisite (BAL)*, penicillamine
Arsenic (As)	BAL*, Dimercaptosuccinic acid (DMSA)
Barium (Ba)	Ba, Ca therapy. See NCRP 161.
Berkelium (Bk)	DTPA
Bismuth (Bi)	BAL, penicillamine, DMSA*
Cadmium (Cd)	DMSA*, DTPA, Ethylenediaminetetraacetic acid (EDTA)
Californium (Cf)	DTPA
Calcium (Ca)	Ba, Ca therapy. See NCRP 161.
Carbon (C)	No treatment available
Cerium (Ce)	DTPA
Caesium (Cs)	Prussian blue
Chromium (Cr)	DTPA*, EDTA (Antacids are contraindicated.)
Cobalt (Co)	DMSA, DTPA*, EDTA, *N*-acetyl-*L*-cysteine (NAC)
Copper (Cu)	EDTA, penicillamine*, trientine
Curium (Cm)	DTPA
Einsteinium (Es)	DTPA
Europium (Eu)	DTPA
Fission products (mixed)	Management depends on predominant isotopes present at time. Early: iodine Late: strontium, caesium, and other
Fluorine (F)	Aluminium hydroxide
Gallium (Ga)	Consider penicillamine
Gold (Au)	BAL*, penicillamine
Indium (In)	DTPA
Iodine (I)	Potassium iodide / iodate (KI)*.propylthiouracil, methamizole. For patients with iodine sensitivity, consider potassium perchlorate.
Iridium (Ir)	Consider DTPA*, EDTA.
Iron (Fe)	Deferoxamine (desferrioxamine) (DFOA)*, DTPA, DFOA and DTPA together
Lanthanum (La)	DTPA
Lead (Pb)	DMSA*, EDTA, EDTA with BAL
Manganese (Mn)	DFOA, DTPA*, EDTA
Magnesium (Mg)	Consider strontium therapy
Mercury (Hg)	BAL*, EDTA, penicillamine, DMSA
Molybdenum (Mo)	Limited clinical experience
Neptunium (Np)	Consider DFOA and/or DTPA.
Nickel (Ni)	BAL*, EDTA

Niobium (Nb)	DTPA
Palladium (Pd)	Penicillamine*, DTPA
Phosphorus (P)	Phosphorus therapy
Plutonium (Pu)	DTPA*, DFOA, EDTA, DTPA and DFOA together
Polonium (Po)	BAL*, DMSA, penicillamine
Potassium (K)	Diuretics
Promethium (Pm)	DTPA
Radium (Ra)	Ra, Sr therapy
Rubidium (Rb)	Prussian blue
Ruthenium (Ru)	DTPA*,EDTA
Scandium (Sc)	DTPA
Silver (Ag)	No specific therapy. Consider gastric lavage and purgatives.
Sodium (Na)	Diuretic and isotopic dilution with 0.9 % NaCl
Strontium (Sr)	See radium and strontium therapy described above
Sulfur (S)	Consider sodium thiosulfate
Technetium (Tc)	Potassium perchlorate
Thallium (Tl)	Prussian blue
Thorium (Th)	Consider DTPA
Tritium (3H)	Force fluids; Water diuresis*
Uranium (U)	Bicarbonate* to alkalinise the urine. Consider dialysis
Yttrium (Y)	DTPA*, EDTA
Zinc (Zn)	DTPA*, EDTA, zinc sulfate as a diluting agent
Zirconium (Zr)	DTPA*,–ED–A

BAL - British Anti-Lewisite; DFOA - Deferoxamine; DMSA - Dimercaptosuccinic acid (succimer); DTPA – Diethylene triamine pentaacetic acid; EDTA – Ethylenediaminetetraaceteic acid; KI - Potassium iodide; NAC - *N*-acetylcysteine.

ANNEX 35C – SIGNIFICANT RADIOLOGICAL MEDICAL COUNTERMEASURES[1]

35C.1. Efficient radiological triage is the prerequisite to providing treatment to casualties that would develop ARS, and therefore need early specific medical support. The main challenge lies in the categorization of casualties:

a. Unexposed individuals or worried well;

b. Exposed individuals not requiring specific medical treatment; and

c. Exposed individuals requiring early (within 24 hours) administration of hematopoietic growth factors / cytokines.

35C.2. The third category, described above, currently relies on irradiation dose estimation and the early initiation of treatment. While the initial assessment of radiological victims includes the timing and severity of prodromal clinical signs and symptoms (in particular vomit onset) and serial complete blood counts with differential, repeated every 6-12 hours, 3-4 days are required to perform the gold standard cytogenetics-based biological dosimetry. There is still a need for improving diagnosis for early differentiation and categorization of ARS in order to begin appropriate medical management during the optimal therapeutic window (using new tools and biomarkers especially differentiating total- and partial-body exposure, and strategies based on bioeffects).

35C.2. In addition to therapies already discussed (antimicrobials, etc.) recent progress has been made through research and exercises performed by STO/HFM-222 RTG to improve both the treatment of the hematopoietic syndrome of the ARS (H-ARS) and the diagnosis of exposed individuals. In 2015, after many years of extensive preclinical research, the US Federal Drugs Administration (FDA) approved the use of granulocyte colony-stimulating factor (G-CSF and pegylated G-CSF, cytokines stimulating the production of neutrophil granulocytes) for mitigating radiation-induced neutropenia in nuclear/radiation accident victims. In addition, CBRN Med WG assessed the impact of medical treatment on the nuclear casualty estimate based on AMedP-7.5 SD3. This study suggests that all victims exposed to doses higher than 1.25 Gy may benefit from G-CSF administration. Since earlier G-CSF treatment can be more effective, (i.e. 24 hours after irradiation is recommended whatever the form, pegylated or not), the task remained to shorten time to dose determination and/or diagnosis. Recent work strongly suggests that reliable diagnosis of external irradiation using the METREPOL (Medical Treatment Protocols for Radiation Accident Victims) clinical severity score (including hematopoietic, gastrointestinal, neurovascular and cutaneous sub-syndromes evaluation) could be achieved within 3 days after irradiation or earlier.

35C.3. Table 35C-1 illustrates the relationship between biodosimetry versus bioeffects – based grouping of expected bone marrow status and general therapeutic interventions. Dose ranges are shown for photon-equivalent acute exposures and response category (RC) levels are based on the METREPOL radiation injury severity scoring system. Dose ranges for the expected bone marrow status and therapeutic interventions are influenced by several confounders including the presence of combined injury, partial-body exposures, and the dose rate of exposures. Use of an effects-based scoring system (i.e., METREPOL response category) can effectively overcome significant confounders (i.e., partial-body exposures, dose

[1] Incorporating the recommendations from STO/HFM-222 RTG.

rate and radiation quality effects) in the development of effective medical treatment strategies for radiation casualties.

Table 35C-1: Photon Equivalent Acute Dose and Response Category Ranges: General Guidelines for Expected Bone Marrow Status and Therapeutic Interventions.

Dose range[1]	Response category	Bone marrow status	General therapeutic interventions
7 - 10 Gy	RC 4	Autologous recovery most unlikely	Stem cell transplantation[2]
3 - < 7 Gy	RC 3	Autologous recovery possible	Stimulation (growth factor therapy); supportive care: Substitution (blood component therapy) as needed
0.5 – < 3 Gy	RC 2	Autologous recovery likely	Supportive care: Substitution (blood component therapy) as needed
< 0.5 Gy	RC 1	Autologous recovery certain	General support of recovery processes; usually no specific therapy

1. Dose ranges are based on photon equivalent acute exposures to the whole body for a mass casualty exposure of healthy persons with no other injuries.
2. Stimulation should be discussed by physicians to fill the gap with delayed hematopoietic transplantation (as signs of recovery in response to growth factor therapy would be expected in case of partial-body irradiation which would contraindicate stem cell transplantation).

35C.4. The current goal for on NATO operations for the management of ARS-H is the administration of cytokine stimulation therapy by 24 hour to casualties receiving 3Gy or more; or 2Gy or more, if a child or combined inury. During a MASCAL and following the authority to implementation the T4 Expectant triage category, an upper threshold may be set (i.e. 8Gy but resource dependent and may be reduced for combined injury).

ANNEX 35D – DEPLETED URANIUM MEDICAL GUIDANCE

35D.1. INTRODUCTION

This Annex is based on the WHO scientific monograph and medical guidance on depleted uranium (DU) by the World Health Organization and United Nations Environment Programme guidance on the possible health effects of exposure to chemical, physical and biological agents. NATO nations may also have their own national specific guidance include post-exposure risk assessment and follow-up.[1,2]

35D.2. NATURAL URANIUM

1. Uranium is a naturally occurring, ubiquitous, heavy metal found in various chemical forms in all soils, rocks, seas and oceans. It is also present in drinking water and food. On average, approximately 90 µg (micrograms) of uranium exist in the human body from normal intakes of water, food and air; approximately 66% is found in the skeleton, 16% in the liver, 8% in the kidneys and 10% in other tissues.

2. Natural uranium consists of a mixture of three radioactive isotopes which are identified by the mass numbers ^{238}U (99.27% by mass), ^{235}U (0.72%) and ^{234}U (0.0054%).

35D.3. DEPLETED URANIUM

1. Uranium is used primarily in nuclear power plants. Most reactors require uranium in which the fissile ^{235}U content is enriched from 0.72% to about 3%. The uranium remaining after the removal of the enriched fraction is referred to as *depleted uranium*. Depleted uranium typically contains about 99.8% ^{238}U, 0.2% ^{235}U and 0.0006% ^{234}U by mass. For the same mass, depleted uranium has about 60% of the radioactivity of uranium.

2. Depleted uranium may also result from the reprocessing of spent nuclear reactor fuel. Under these conditions another uranium isotope, ^{236}U may be present together with very small amounts of the transuranic elements plutonium, americium and neptunium and the fission product technetium-99. The increase in the radiation dose from the trace amounts of these additional elements is less than 1%. This is insignificant with respect to both chemical and radiological toxicity.

3. Depleted uranium has a number of peaceful applications:

   d. Counterweights or ballast in aircraft

   e. Radiation shields in medical equipment used for radiation therapy

   f. Containers for the transport of radioactive materials.

4. Due to its high density, which is about twice that of lead, and other physical properties, depleted uranium is used in munitions designed to penetrate armour plate. It also reinforces military vehicles, such as tanks.

[1] WHO. Monograph: Depleted uranium: sources, exposure and health effects. May 2001.
[2] UNEP. 2001. Depleted uranium in Kosova, Post-conflict environmental assessment. March 2001.

35D.4. ROUTES OF EXPOSURE TO DEPLETED URANIUM

1. Individuals can be exposed to depleted uranium in the same way they are routinely exposed to natural uranium i.e. by inhalation, ingestion, skin contact and wounds including embedded fragments.

a. *Inhalation*. Inhalation is the most likely route of intake during or following the use of depleted uranium munitions in conflict or when depleted uranium in the environment is re-suspended in the atmosphere by wind or other forms of disturbance. Accidental inhalation may also occur as a consequence of a fire in a depleted uranium storage facility, an aircraft crash or the decontamination of vehicles from within or near conflict areas.

b. *Ingestion*. Ingestion could occur in large sections of the population if their drinking water or food became contaminated with depleted uranium. In addition, the ingestion of soil by children is also considered a potentially important pathway.

c. *Skin contact*. Contact with intact skin is considered a relatively unimportant type of exposure since little of the depleted uranium will pass across the skin into the blood.

d. *Wound contact*. Depleted uranium could enter the systemic circulation through open wounds or from embedded depleted uranium fragments.

2. Inhalation and wound exposure are the two most important routes of operational exposure to depleted uranium of military personnel. Both routes are likely to be due to munition use, impact and combustion leading to high and relatively short exposures to both insoluble fragments and particles, and soluble combustion products. Conversely the risk to the local population is more likely to be lower levels and longer, due to the local introduction of DU, especially soluble forms, into the water and food chain. Direct contact exposure and subsequent ingestion may be a particular problem for children playing in areas of heavy DU contamination.

35D.5. HEALTH EFFECTS

1. Most (>95%) uranium entering the body is not absorbed, but is eliminated via the faeces. Of the uranium that is absorbed into the blood, approximately 67% will be filtered by the kidney and excreted in the urine in 24 hours. Typically between 0.2 and 2% of the uranium in food and water is absorbed by the gastrointestinal tract. Soluble uranium compounds are more readily absorbed than those which are insoluble.

2. Potentially depleted uranium has both chemical and radiological toxicity with the two important target organs being the kidneys and the lungs. Health consequences are determined by the physical and chemical nature of the depleted uranium to which an individual is exposed, and to the level and duration of exposure:

a. *Renal function*. Long-term studies of workers exposed to uranium have reported some impairment of kidney function depending on the level of exposure. However, there is also some evidence that this impairment may be transient and that kidney function returns to normal once the source of excessive uranium exposure has been removed.

b. *Respiratory effects*. Insoluble inhaled uranium particles (1-10 μm in size) tend to be retained in the lung and may lead to irradiation damage of the lung and even lung cancer if a high enough radiation dose results over a prolonged period.[3]

c. *Skin contact*. Direct contact of depleted uranium metal with the skin, even for several weeks, is unlikely to produce radiation-induced erythema (superficial inflammation of the skin) or other short term effects.

d. *Wound fragments*. Follow-up studies of veterans with embedded fragments in the tissue have shown detectable levels of depleted uranium in the urine, but without apparent health consequences.

e. *Ionising radiation effects*. The radiation dose to military personnel within an armoured vehicle is very unlikely to exceed the average annual external dose from natural background radiation from all sources.

35D.6. WHO GUIDANCE ON HEALTH EFFECTS

1. The WHO monograph gives for the different types of exposure the tolerable intake, an estimate of the intake of a substance that can occur over a lifetime without appreciable health risk. These tolerable intakes are applicable to long term exposure. Single and short term exposures, which is more likely to be seen in an operational environment, to higher levels may be tolerated without adverse effects but quantitative information is not available to assess how much the long term tolerable intake values may be temporarily exceeded without risk.

2. The WHO guidance for safe levels include:

a. *DU ingestion by general public*. The general public's ingestion of soluble uranium compounds should not exceed the tolerable intake of 0.5 μg per kg of body weight per day. Insoluble uranium compounds are markedly less toxic to the kidneys, and a tolerable intake of 5 μg per kg of body weight per day is applicable.

b. *DU inhalation by general public*. Inhalation of soluble or insoluble depleted uranium compounds by the public should not exceed 1 $\mu g/m^3$ in the respirable fraction. This limit is derived from renal toxicity for soluble uranium compounds, and from radiation exposure for insoluble uranium compounds.

c. *Occupational levels for DU ingestion.* With the exception of the close proximity to a DU impact site, occupational exposures are unlikely to be an issue as long as good water safety practice is maintained and local water avoided in preference to bottled water or reverse osmosis.

d. *Occupational levels for DU inhalation.* When deployed, especially where work and accommodation areas are in close proximity, 8-hour limitations are not applicable or corrected to assume a 24-hour period for a total of 6-12 months or other duration. Exposure in local areas of DU concentration i.e. within metres, should be mitigated by avoidance or use of protective equipment.

[3] The scientific evidence for this is tenuous because studies of uranium mill workers have not shown any excess of lung cancer. In studies of underground miners, the excess of lung cancer seems to be related to radon gas; radon being a radioactive decay product of natural uranium.

35D.7. ENVIRONMENTAL CONSIDERATIONS

1. Only military use of depleted uranium is likely to have any significant impact on environmental levels. Measurements of depleted uranium at sites where depleted uranium munitions were used indicate only localized (within a few tens of metres of the impact site) contamination at the ground surface. However, in some instances the levels of contamination in food and ground water could rise after some years and should be monitored and appropriate measures taken where there is a reasonable possibility of significant quantities of depleted uranium entering the food chain. The WHO guidelines for drinking-water quality, 2 µg of uranium per litre, would apply to depleted uranium.

2. Where possible, clean-up operations in conflict impact zones should be undertaken if depleted uranium contamination levels are deemed unacceptable. Areas with very high concentrations of depleted uranium may need to be cordoned off until they are cleaned up.

3. Since depleted uranium is a mildly radioactive metal, restrictions are needed on the disposal of depleted uranium. There is the possibility that depleted uranium scrap metal could be added to other scrap metals for use in refabricated products. Disposal should conform to appropriate recommendations for use of radioactive materials.

35D.8. PUBLIC HEALTH CONSIDERATIONS

Limitation on human intake of soluble depleted uranium compounds should be based on a tolerable intake value of 0.5 µg per kg of body weight per day, and that the intake of insoluble depleted uranium compounds should be based on both chemical effects and the radiation dose limits. Exposure to depleted uranium should be controlled to the levels recommended for protection against radiological and chemical toxicity outlined in the monograph for both soluble and insoluble depleted uranium compounds. Additional public health guidance includes:

a. General screening or monitoring for possible depleted uranium-related health effects in populations living in conflict areas where depleted uranium has been used is not necessary.

b. Individuals who believe they have been exposed to excessive amounts of depleted uranium should consult their medical practitioner for examination, appropriate treatment of any symptoms and follow-up.

c. Young children could receive greater depleted uranium exposure when playing within a conflict zone because of hand-to-mouth activity that could result in high depleted uranium ingestion from contaminated soil. This type of exposure needs to be monitored and necessary preventative measures taken.

35D.9. MEDICAL CONSIDERATIONS

1. *Deployed medical personnel*. Medical personnel are able to deploy to DU conflict areas. However, they should avoid areas with significant DU debris and dust that should have been cordoned off and sign-posted accordingly.

2. *Pre-deployment medicals.* Staff undertaking a medical examination prior to taking up duties in areas where DU munitions were used should be healthy. If there is kidney impairment, judgement should be made on the basis of their fitness to perform the tasks required and not

on any possible exposure to DU. Patients undertaking a routine medical examination should be told that:

a. Normal levels of DU in the environment are extremely low.

b. The possible consequence of significant DU exposure is some transient impairment of the kidneys, but that this may not be clinically detectable.

3. *Population screening*. There is no health-based reason to perform medical screening for DU toxicity on populations living in regions where DU was used in conflicts, or for civilians staying in these places during their duty period. Measurements taken at selected sites during a UNEP mission to Kosovo found that levels of DU contamination were very low and localised only to impact areas.

4. *Exposed persons.* The general approach to dealing with patients who claim or suspect that they have been exposed to DU is as follows:

a. Any individual who feels that they have been exposed to high levels of DU and/or displays some symptoms that may be related to this exposure should be clinically evaluated. This includes:

(1) *Assessment of DU exposure*. Assess the relative amounts of dust or debris that could have been taken in by detailed questioning of the circumstances surrounding exposure - circumstances, date and time of the exposure, amount of dust in the air at the time, and could the water supply be near a high DU impact area?

(2) *If contracted near an explosion*. Is there evidence of a wound or embedded fragments?

(3) *If the patient is a child*. Was there play near damaged tanks or were fragments of munitions picked up, or is there a possibly of ingested DU dusts through play or hand-to-mouth activities?

(4) Has the patient kept any DU metal pieces as souvenirs or wears a necklace with a DU penetrator or similar object so that prolonged skin contact is possible.

b. Perform a routine examination

(1) Medical examination.

(2) Determine blood urea or creatinine.

(3) Conduct a routine urine analysis, checking for protein, albumin and glucose.

(4) Full blood count.

(5) Chest X-ray, if likely inhalation of significant amounts of material into lungs.

c. For patients whose history suggests proximity to a source of DU dust or injury with DU fragments, or who show abnormalities in the above routine examination, should be tested for uranium exposure.

5. *Renal assessment*. In the short term, the kidneys are the most susceptible organ to uranium, once absorbed into the bloodstream. Signs of tubulopathy should be investigated. If

the tubules are damaged, there are many low molecular weight proteins that appear in the urine, among which β2-microglobulin is the most common.

a. *24 hour urine collection*. This diagnostic procedure should be used to determine the level of β2-microglobulin in a 24-hours urine collection. Many hospitals or laboratories are able to perform this analysis. The patients must be informed how to collect the 24-hour urine specimen correctly.

b. *Urine alpha spectroscopy.* If the results indicate some pathology, the most appropriate test to show whether significant uranium has been incorporated is a urine uranium test. The most common laboratory method for measuring total uranium in a urine specimen is alpha spectroscopy (spectrometry). However, as only a few laboratories are equipped for the determination of uranium in the urine, it is necessary to contact the laboratory before collecting the urine and to follow their instructions. A spot urine analysis is of less value, and should at least be coupled with a creatinine determination.

c. *Uranium isotope studies*. If urine uranium is elevated, the amount of DU excreted in urine in 24 hours should be determined using isotope-specific methods to identify the isotopic ratios of natural, enriched or depleted uranium. Few laboratories are able to assess the amounts of the different isotopes of uranium.

6. *Specialist referral*. Referral to a nephrologist for further testing, diagnosis and treatment may be needed. Kidney disease is common, and many cases of proteinuria, even in the setting of proven DU excretion, may be found to be due to other causes.

7. *Treatment*. In case of acute exposure, it should be handled as any heavy metal incorporation. Treatment should be based on risk assessment. Increased urinary excretion of uranium is only temporary and these methods are only helpful when applied early after exposure before uranium is fixed in the skeleton or kidney. In case of renal tubulopathy being diagnosed, treatment should consist of:

a. Urine alkalisation with sodium bicarbonate infusion, to bind the uranium present in the blood stream, facilitate its renal excretion and prevent its reabsorption in the renal tubules.

b. Heavy metal chelation therapy may be useful, but it is doubtful whether this would be necessary for removal of DU material

c. Monitoring of the renal function if necessary; kidney dialysis may be indicated in cases of severe kidney damage.

d. Monitoring of the liver function.

8. *Prognosis*. In most cases no permanent effects will remain. In case of an acute DU exposure there is the possibility of renal tubular acidosis. If DU dust inhalation resulted in the incorporation of significant amounts of insoluble uranium compounds, long-term patient follow up should include checks for lung tumours. However, patients should be told that the likelihood that any health effects will develop is low.

RELATED DOCUMENTS

CBRN MEDICAL ALLIED PUBLICATIONS

AJMedP-7	2461	Allied Doctrine for Medical Support for CBRN Defensive Operations
AMedP-7.2	2358	CBRN First Aid Manual
AMedP-7.3	2954	Medical Management of CBRN Casualties
AMedP-7.4	2551	Regulations for Establishment and Employment of Medical Radiological Incident Investigation Team (MRIIT)
AMedP-7.5	2553	NATO Planning Guide for the Estimation of CBRN Casualties
AMedP-7.6	2873	Commanders Guide to Medical Support to CBRN Defensive Operations
AMedP-7.7	2529	Rapidly Deployable Outbreak Investigation Team (RDOIT) for Suspected Use of Biological Warfare Agents
AMedP-7.8	2474	Recording of Operational Ionizing Radiation Exposure for Medical Purposes and Management of Dosimeters

OTHER MEDICAL ALLIED PUBLICATIONS

AJP-4.10	2228	Allied Joint Doctrine for Medical Support
AAMedP-1.1	3204	Aeromedical Evacuation
AJMedP-1	2542	Allied Joint Medical Planning Doctrine
AMedP-1.6	2560	Medical Evaluation Manual
AMedP-1.7	2560	Capability Matrix
AMedP-1.8	2560	Skills Matrix
AMedP-1.10	2879	Medical Aspects in the Management of a Major Incident / Mass Casualty Situation
AJMedP-2	2546	Allied Joint Medical Doctrine for Medical Evacuation
AJMedP-3	2547	Allied Joint Doctrine for Medical Intelligence
AJMedP-4	2561	Allied Joint Medical Force Health Protection Doctrine
AMedP-4.1	2535	Deployed Health Surveillance
AMedP-8.6	2564	Forward Mental Health
AMedP-24	2549	Emergency Medical Care in the Operational Environment
-	2122	Requirement for Training in First-Aid, Emergency Care in Combat Situations and Basic Hygiene for all Military Personnel

CBRN ALLIED PUBLICATIONS

AJP-3.8	2451	Allied Joint Doctrine for CBRN Defence
AJP-3.8.1 Vol 1	2521	CBRN Defence on Operations
AJP-3.8.1 Vol 2	2522	Specialist CBRN Defence Capabilities
AJP-3.8.1 Vol 3	2520	CBRN Defence Standards for Exercises, Training and Evaluation
AEP-54	4634	Collective Protection in a CBRN Environment
AEP-66	4632	NATO Handbook for Sampling and Identification of Biological, Chemical and Radiological Agents
ATP-65	2499	The Effect of Wearing CBRN Individual Protective Equipment (IPE) on Individual and Unit Performance during Military Operations

OTHER ALLIED PUBLICATIONS

ATP-92	2070	Emergency Burial Procedures

RELATED STO REPORTS

TR-HFM-041	Prophylaxis and Therapy Against Chemical Agents	
TR-HFM-099	Radiation Bioeffects and Countermeasures	Not fully releasble
TR-HFM-186	State-of-the-Art in Research on Medical Countermeasures Against Biological Agents	
TR-HFM-222	Ionizing Radiation Bioeffects and Countermeasures	Pre-release

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LEXICON

☑	Symbol for *best practice* or *lesson identified*.
●	Symbol for additional content including *augmented reality*
✍	Symbol for recent science (i.e. STO) recommendation
2-PAM	Pralidoxime Chloride
4-DMAP	4-Dimethylaminophenol Hydrochloride
5-HT	Serotonin
AAMedP	Allied Aeromedical Publication
AC	Hydrogen Cyanide
ACh	Acetylcholine
AChE	Acetylcholinesterase
AFRRI	(US) Armed Forces Radiobiology Research Institute
AJMedP	Allied Joint Medical Publication
AJP	Allied Joint Publication
ALI	Acute Lung Injury
AMC	Advanced Medical Care
AMedP	Allied Medical Publication
ANS	Autonomic Nervous System
AP	Allied Publication
AR	Augmented Reality
ARDS	Acute Respiratory Distress Syndrome
ARS	Acute Radiation Syndrome
ATI	Air Transportable Isolator
BA	Biological Agent
BAL	British Anti-Lewisite (Dimercaprol)
BAT	Biodosimetry Assessment Tool
BChE	Butyrylcholinesterase
BP	Blood Pressure
BSA	Body Surface Area
BSL	Bio-safety Level
BW	Biological Weapon
BWA	Biological (Warfare) Agent
BZ	3-Quinuclidinyl benzilate
<C>ABC	Catastrophic haemorrhage, Airway, Breathing and Circulation
CASEVAC	Casualty Evacuation
Cat Haem	Catastrophic Haemorrhage
CBC	Complete (Peripheral) Blood Count
CBRN	Chemical Biological Radiological and Nuclear
(Fwd) CCP	(Forward) Casualty Collection Point
CDA	Casualty Decontamination Area

CDC[1]	Casualty Decontamination Centre
CDC[2]	(United States) Centres for Disease Prevention and Control
CDL	Clean Dirty Line
CDU	Casualty Decontamination Unit
CG	Phosgene
CIMIC	Civil Military Co-operation
CK	Cyanogen Chlorine
CL	Chlorine
CN	Chloracetophenone (Mace Spray)
CNS	Central Nervous System
CO	Carbon Monoxide
COLPRO	Collective Protection
CPAP	Continuous Positive Airway Pressure
CPE	Casualty Protective Equipment
CRESS	Consciousness, Respirations, Eyes, Secretions and Skin (Assessment)
CRP	C-Reactive Protein
CS	Orthochlorobenzylidene Malononitrile (Tear Gas)
CSF[1]	Cerebral Spinal Fluid
CSF[2]	Colony Stimulating Factor
Ct	Concentration Time
CT[1]	Computed Tomography
CT[2]	Counter Terrorism
CW	Chemical Weapon
CWC	Chemical Weapons Convention
CX	Phosgene Oxime
CXR	Chest x-ray (radiograph)
DA	Diphenylchlorarsine
DAT	Defence Against Terrorism
DC	Diphenylcyanarsine
DFOA	Deferoxamine (Desferrioxamine)
DIC	Disseminated Intravascular Coagulopathy
DM	Diphenylaminochlorarsine (Adamsite)
DMSA	Dimercaptosuccinic Acid (Succimer)
DNA	Deoxyribose Nucleic Acid
DP	Diphosgene
DTPA	Diethylene Triamine Pentaacetic Acid
DU	Depleted Uranium
ECG	Electrocardiogram
ED_{50}	Effective Dose 50
EDTA	Ethylenediaminetetraacetic Acid
EEG	Electro-encephalogram

EIH	Environmental and Industrial Hazards
ELISA	Enzyme Linked Immunosorbent Assay
EMP	Electromagnetic Pulse
EMT	Emergency Medical Treatment
EMTm	Emergency Medical Team
EPD	Electronic Personal Dosimeter
EPR	Electron Paramagnetic Resonance
ERG	Emergency Response Guidebook
ERW	Enhanced Radiation Weapon
FBC	Full (Peripheral) Blood Count
FE	Fuller's Earth
FM	Titanium Tetraoxide
FP	Fission Products
FRC	Functional Residual Capacity
FS	Sulphur Trioxide-chlorosulphuric Acid
GA	Tabun
GB	Sarin
GD	Soman
GF	Cyclosarin
GI	Gastrointestinal
H	Sulphur Mustard
HAZMAT	Hazardous Materials
HC	Zinc Oxide Mixtures
HF	Hydrofluoric Acid
HME	Homemade Explosive
HN	Nitrogen Mustard
IAEA	International Atomic Energy Agency
IATA	International Air Transport Association
ICRP	International Committee for Radiation Protection
ICt_{50}	Incapacitating Concentration Time 50
ID^{1}	Identification Number
ID^{2}	Infectious Dose
$ID_{50}{}^{1}$	Incapacitating Dose 50
$ID_{50}{}^{2}$	Infectious Dose 50
IDAU	Infectious Disease Assessment Unit
IED	Improvised Explosive Device
IHR	International Health Regulations
IND	Improvised Nuclear Device
INES	International Nuclear Event Scale
INR	International Normalised Ratio
IPE	Individual Protective Equipment

IRT	Immediate Response Team
JAT	Joint Assessment Team
JOA	Joint Operational Area
KI	Potassium Iodide or Potassium Iodate
L	Lewisite
LCt_{50}	Lethal Concentration Time 50
LD_{50}	Lethal Dose 50
LEGAD	Legal Advisor
LET	Linear Energy Transfer
LI / LL	Lessons Identified / Learnt
LMA	Laryngeal Mask Airway
LSD	D-lysergic acid diethylamide
LSI	Life-Saving Intervention
MAGO	Medical Advisory Group on Operations
MASCAL	Mass Casualty
MEDAD	Medical Advisor
MedCM	Medical Countermeasure
MEDEVAC	Medical Evacuation
MO	Medical Officer
MOF	Multiple Organ Failure
MRIIT	Medical Radiological Incident Investigation Team
MTF	Medical Treatment Facility
NA	Nerve Agent
NA5CRO	Non Article 5 Crisis Response Operation
NAC	*N*-Acetylcysteine
N/L	Neutrophil / Lymphocyte (Ratio)
NO	Nursing Officer
NOx	Oxides of Nitrogen
NW	Nuclear Weapon or Warfare
OC	Oleoresin Capsicum (Pepper Spray)
OP	Organophosphate (-orous)
OPCW	Organisation for the Prohibition of Chemical Weapons
OPIDN	Organophosphorus Induced Delayed Neuropathy
P2S	Pralidoxime Mesilate
PAR	Population at Risk
PBA	Pharmaceutical Based Agent
PCC	Premature Chromosome Condensation
PCP	Phencyclidine
PCR	Polymerase Chain Reaction
PEEP	Positive End Expiratory Pressure
PF	Protection Factor

PFIB	Perfluoroisobutylene
PHEIC	Public Health Emergency of International Concern
PoE	Point of Exposure
PoW	Point of Wounding
PPE	Personal Protective Equipment
PS	Chloropicrin
PTSD	Post-traumatic Stress Disorder
QR	Quick Response
R_0	Reproduction Number (Ratio)
RBC-AChE	Red Blood Cell Acetylcholinesterase
RBE	Relative Biological Effectiveness
RC	Response Category
RCA	Riot Control Agent
RDD	Radiological Dispersal Device
RDOIT	Rapid Deployable Outbreak Investigation Team
REAC/TS	Radiation Emergency Assistance Centre / Training Site
RED	Radiological Exposure Device
RM / RoM	Restriction of Movement
RNA	Ribose Nucleic Acid
RP	Red Phosphorous
RSDL	Reactive Skin Decontamination Lotion
RSI	Rapid Sequence Induction
RTS	Revised Trauma Score
SARS	Severe Acquired Respiratory Syndrome
SCBA	Self-contained Breathing Apparatus
SCIAD	Scientific Advisor
SEB	Staphylococcal Enterotoxin B
SIBCRA	Sampling Identification of Biological Chemical and Radiological Agents
SIRS	Severe Inflammatory Response Syndrome
SRD	Standardisation Related Document
TE	Time to Emesis (Vomiting)
TIB	Toxic Industrial Biological
TIC	Toxic Industrial Chemical
TIM	Toxic Industrial Material
TIR	Toxic Industrial Radiological
TLD	Thermoluminescent Dosimeter
TNT	Trinitrotoluene
UV	Ultraviolet
VEE	Venezuelan Equine Encephalitis
VHF	Viral Haemorrhagic Fever
WCC	White Cell Count

WHO	World Health Organization
WOO	Window of Opportunity
WP	White Phosphorous
W_R	Radiation Weighting Factor
W_T	Tissue Weighting Factor
WW	World War

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