

CHEMICAL, BIOLOGICAL AND RADIATION CASUALTIES: CRITICAL CARE CONSIDERATIONS

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Abstract

A chemical, biological, radiological or nuclear (CBRN) event would require a critical care response from point of exposure to definitive hospital management. Critical care staff should be aware of the potential agents and possible hazard they present to responders. The treatment of CBRN casualties should reflect the conventional incident and casualty management paradigms with additional safety and treatment considerations. Specific agents may require specific airway and respiratory considerations including surrogate ventilation strategies. All critical care staff training should include CBRN awareness and personal protective equipment training. Some staff may benefit from additional training including the recognition and investigation of CBRN casualties as well as their treatment and the equipment available. Critical care departments should also contingency plans for CBRN events including surge capacity.

Introduction

The use of chemical, biological and radiation devices remains a credible threat. However, over the last decade the threat has change from Cold War scenarios to the asymmetric and terrorist threat [1]. Examples of CBRN weapon use have been seen across the spectrum of military and civilian operations (Table 1). Although the term weapons of mass destruction (WMD) is sometimes used to describe CBRN weapons, this only really applies to a nuclear detonation and the phrase 'weapons of mass effect' is perhaps more appropriate. The mass effect seen by the deployment of a CBRN device is likely to have significant critical care issues throughout the casualty evacuation chain from Point of Exposure (PoE) through to repatriation to a Role 4 facility [2,3]. There are a number of scenarios that would require a CBRN critical care response, these include:

- Presentation of a single casualty with multi-organ failure (ricin or sepsis)
- Presentation of multiple casualties with respiratory support requirements (chlorine and nerve agents)
- Presentation of a single casualty (botulinum) or multiple casualties (chemical burns) with long term critical care or specialist needs
- Presentation of casualties with high bio-security needs including negative-pressure barrier nursing (contagious casualties)

In addition, CBRN weapons are a potential hazard for health care workers due to secondary *contamination* or patients who are *contagious*. This healthcare risk may be mitigated by implementing steps such as donning appropriate *personal protective equipment* (PPE), casualty *containment* and subsequent *decontamination* or *isolation* (hazard management). The choice of PPE may result in a reduction in clinical interaction with the patient ('medical dexterity') and this is balanced against the level of protection provided by the PPE.

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 regimes

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 biowarfare agent such as anthrax [4].

Threat

The acronym CBRN was adapted from the original NBC (Nuclear, Biological and Chemical). NBC reflected the likely Cold War use of these weapons on a strategic scale with the potential use of nuclear weapons against both military and civilian targets. Following the end of the Cold War and the reduced threat from nuclear weapons, the greater risk from a radiological (radioactive) dispersal device (RDD), known as a 'dirty bomb' was reflected by the addition of the 'R'. The threat from a CBRN device depends of the *intent* of the enemy and their *capability*. Non-state organizations are more likely to use improvised CBRN devices using available hazardous materials including toxic industrial hazards (TIH) or endemic disease. State organizations especially with an established weapons program are more likely to have more sophisticated weapons and dispersal methods. Where there is a credible threat, then the *likelihood* of an event depends on the *vulnerabilities* of the target. Threats to deployed forces depend on the nature of operations (see Table 1) and the level of Force Protection applied including physical protection and medical countermeasures. Although threat and risk are related they are not the same and the *consequences* may still present a significant risk. Where there are health consequences this equates to a significant *clinical risk* (Figure 1).



Figure 1. Threat and clinical risk (Bland, 2009).

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Type of operation/scenario	Location	Agent(s)
Armed conflict (war fighting)	Western front (1915-18)	Chlorine, phosgene, mustard, cyanide
	Manchuria (1938-45)	Plague, anthrax, typhoid, cholera
	Hiroshima, Nagasaki (1945)	Nuclear detonation
Insurgency	Iraq (2007)	Chlorine
Counter-insurgency	Moscow (2002)	Fentanyl analogue
Civil-ethnic conflict	Halabja (1988)	Mustard, nerve agent, ?cyanide
Terrorism	Tokyo (1995)	Sarin
	Eastern US (2001)	Anthrax
Assassination	London (1978)	Ricin
	London (2006)	Polonium-210
DNBI	Bagram (2002)	Norovirus
Humanitarian operations	Zaire (1994)	Cholera
Accidental	Sverdlovsk (1979)	Anthrax

Table 1. Examples of confirmed and potential CBRN events.

Routes Of Exposure

There are a number of ways that CBRN agents can penetrate the body. Some routes are quicker than others for systemic effects and may also result in localised effects [5].

- **Inhalation** - agent (gas, vapour, particles & smoke) breathed in (cyanide gas, carbon monoxide, chlorine, inhalational anthrax). This route is probably of greatest significance for critical care as it is not only an effective way of introducing an agent into a victim but may also have significant local effects on both the airway and lungs (mustard, nerve agent and pulmonary agents). The inhalational route is unlikely to lead to a significant saturation of the casualty to be an off-gassing hazard as this would exceed the likely lethal dose for the casualty.
- **Ingestion** – agent (liquid, solid) eaten or drank (cyanide salts, intestinal anthrax, salmonella). Ingestion may result in a sink of hazardous chemical that may be an off-gassing hazard (cyanide salts reacting with stomach acid to evolve hydrogen cyanide gas).
- **Inoculation** – any penetration of the skin to introduce the agent (liquid, solid). An example of this was the ricin loaded pellet used to killed Georgi Markov, the weapon being disguised as an umbrella. Medics routinely inoculate patients when given intramuscular injections and malaria is transmitted by this route [6].
- **Wound** – any contamination after the skin has been broken, mainly by live biological agents (tetanus, MRSA). There is some overlap with inoculation depending upon the wound size and primary intent of the weapon.
- **Transcutaneous** – agent (liquid, solid) is absorbed through the skin without initially breaking it. This includes VX (causing no skin damage), mustard, acids and alkali (causing blistering wounds). In certain cases the transcutaneous routes (immersion in a lipid soluble chemical such as trichloroethylene) may allow sufficient saturation of the agent to act as a sink for significant secondary exposure by off-gassing or excretion [7].
- **Ocular** – agents (gas, vapour) may have a local effect on the eye, although there may not be any significant systemic absorption. A good example of this is low level nerve agent exposure causing pin-point pupils (miosis) only. Ocular injuries may also need specific management and follow-up (mustard casualties).

General Considerations

There are several classification systems for CBRN agents based

upon effect and physical properties. Some of the classification systems date back to World War I but remain in common use. They were based upon the initial presenting complaint as seen by the physicians of the time before the true pathophysiology was understood. Examples include ‘choking’ (chlorine) and ‘blood agents’ (cyanide), however these terms are misleading and should be avoided. Using an all-hazards approach, most CBRN agents can be categorised into one of three groups [5], although this classification was originally applied to chemical agents only:

- Lethal (cyanide, nerve agents, anthrax, plague, high dose radiation)
- Damaging (mustard gas, low dose radiation)
- Incapacitating (CS, LSD, salmonella, tularemia)

Physical properties. The physical properties of CBRN agents are very important because of the potential risk to responders of secondary exposure by contamination or contagion.

- **Non-persistent.** Gases (hydrogen cyanide, carbon monoxide) and volatile liquids / vapours are unlikely to remain for any significant period of time, although dependant on ambient conditions, and are called non-persistent. Some vapours may be absorbed into clothing and it is prudent to remove the clothing of any patient that has had any significant exposure. Further decontamination should be assessed based on the agent and resources available but should not prevent or delay life saving interventions (LSIs). Hazard management priorities for these agents are rapid extraction from the ‘hot zone’ and ventilation of the area.
- **Persistent.** Some agents are less-volatile liquids and viscous (mustard / VX nerve agent) or particulate / dry material (asbestos/ anthrax spores / radioactive debris). These agents require formal decontamination and the actual decontamination method and agent used depends upon the physical and chemical properties of the agent. The urgency of decontamination depends upon the toxicity of the substance, risk of transcutaneous absorption (VX) and state of the patient. The decontamination process may consist of the physical removal of the contaminant (Fullers Earth, high flow water) and chemical deactivation (hydrolysis). Hazard management priorities for these agents are extraction from the hot zone and rapid decontamination with concurrent life saving interventions, as required.

Contamination / Contagious (Two C’s). All CBRN hazards have the potential to cause contamination. The extent of the contamination depends on the physical properties of the agent and its presentation (persistent / non-persistent). Contamination can be *external*, *internal* as well as *wound contamination*. Internal

contamination may be due to *inhalation, ingestion, inoculation* (breaks the skin), *transcutaneous* (through intact skin) and *wound* contamination. Some live biological agents, once an infection has been established, will self replicate and have the potential to be spread by person-to-person, this status is known as *contagious*.

The safety priorities for incidents where there may be a CBRN hazard are:

- *Contain* (hazard management)
- *Assess* the scene (sense) with
- *Assess* the casualties (sense)
- *Decontaminate* (hazard management), as required if contaminated
- *Isolate* casualty (hazard management), as required if contagious
- *Quarantine* contacts (hazard management), as required if contagious and consider wider *Restriction of Movement* (ROM) if limiting risk of epidemic/pandemic.

Effects of CBRN exposure (Four I's)

The effects from CBRN agents can be summarised using the four I's. When assessing a casualty or incident scene where there may be a CBRN threat, consider potential clinical signs that may suggest there has been a significant exposure.

- *Intoxication* (Chemical & Biological (toxins))
- *Infection* (Biological (live agents))
- *Irradiation* (Radiological / Nuclear)
- *Injuries* (Trauma)

The onset of symptoms is very dependent on the type of CBRN agent. In general, the more complex the agent the longer the latency period or, in the case of live biological agents, incubation period (Figure 2). This latency period may allow for post-exposure prophylaxis is what is termed the 'window of opportunity'. Medical countermeasures include antibiotics, vaccination or antidotes. However, this relies on recognition of an event before the manifest illness usually due to environmental detection of a CBRN release. The recognition or confirmation of a CBRN event may not be immediate and there are several steps in the cycle of CBRN recognition [8]. For critical care staff, this includes clinical recognition (diagnosis), investigations and potentially forensic. This is supported by environment/scientific information with the background of the intelligence assessment.

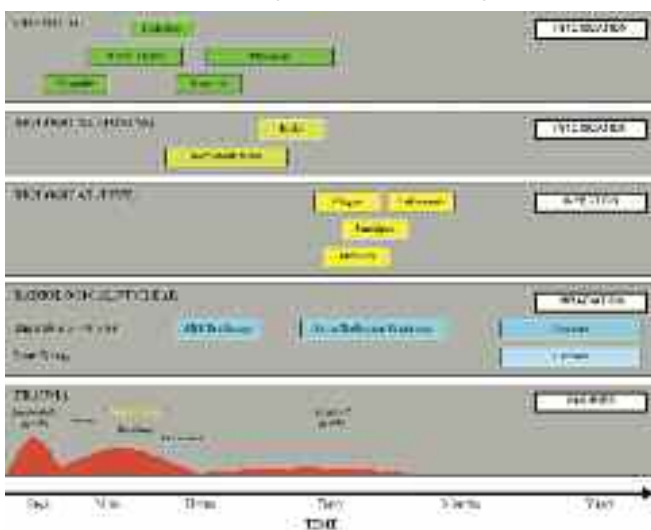


Figure 2. CBRN onset of action and latency periods.

This all adds to situational awareness (Figure 3). Critical care patients are likely to have received a greater dose of the 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 (pneumonic plague/smallpox/covert anthrax release) [9].

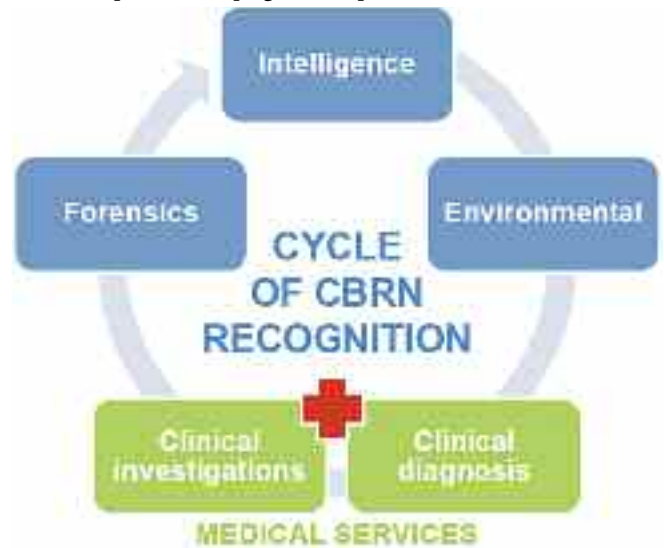


Figure 3. Cycle of CBRN recognition.

Chemical Agents

Chemical agents can also be classified into specific groups based upon their mechanism of action as well as using the general descriptions given in the previous section (Box 1). These include:

- *Nerve agents*, such as Sarin and VX.
- *Cyanides* (and hydrogen sulphide), formerly though incorrectly termed blood poisons.
- *Vesicants* (blistering agents), such as mustard and lewisite.
- *Acids and alkalis*, notably hydrofluoric acid.
- *Pulmonary agents*, such as chlorine and phosgene. Formerly referred to as choking agents.
- *Methaemoglobin* (MetHb) and *carboxyhaemoglobin* (COHb) *inducers*, some MetHb effects can be caused by antidotes and other pharmaceuticals.

Toxic industrial chemicals (TICs) are often treated as a separate group of chemicals, however many of the above chemical agents fall into this group (chlorine, phosgene) [10]. The main difference is the operational considerations as the clinical effects will be the same. In general most of the lethal chemical agents affect oxygen delivery from air to the mitochondrial enzymes and therefore aerobic respiration [5].

Biological Agents

Biological agents can be classified as:

- *Live agents*, including bacterial, viral, fungal, chlamydial and rickettsial.
- *Toxin*. A toxin can be considered to be a chemically active agent of biological origin. It therefore does not self-replicate and so is non transmissible once decontamination, if indicated, has taken place. Biological toxins may be derived from microbes (botulinum), animal (snake and scorpion venom) or plant origin (ricin and digitalis).

Toxins will generally have a shorter latency period (hours – days) compared to the longer incubation period of a live agent which may be up to weeks and in some cases months (viral hepatitis, HIV). Some live agents will also produce toxins as part of the infection (tetanus, anthrax, botulinum). However some toxins can be used and spread without needing a live agent (botulinum, ricin, staphylococcal toxin). Biological agents can be injected (inoculated), ingested or inhaled. However, internal contamination or infection through intact skin (percutaneous) is more difficult than for chemicals. Agents may be delivered by

NERVE AGENTS		NATO DESIG	G & V agents
Mechanism: Inhibition of the enzyme acetylcholinesterase that breaks down the nerve transmitter acetylcholine. This results in over stimulation of the parasympathetic system, motor neurons (leading to paralysis) and CNS.		QUICK LOOK	
Mild:	Miosis, eye pain, red eyes	Resp Rate:	↑↑
Moderate:	Secretions, wheezing, nausea, vomiting, diarrhoea, difficulty in breathing	Pupils:	Pinpoint
Severe:	Muscle weakness, respiratory fatigue, respiratory arrest, seizures, death	Skin:	Sweaty
Antidotes:	Anticholinergics (atropine), oximes (pralidoxime/obidoxime), diazepam	Secretions:	+++
		Other factors: For skin exposure – local fasciculation	
CYANIDES		NATO DESIG	AC, CG
Mechanism: Cyanide inhibits the mitochondrial enzymes in cells. This stops cells using oxygen and metabolising glucose completely (aerobic respiration). This leads rapidly to a metabolic (lactic) acidosis (chemical asphyxiant).		QUICK LOOK	
Mild:	Nausea, dizziness, agitation	Resp Rate:	↑↑ / Apnoea
Moderate:	Hyperventilation, confusion	Pupils:	N/dilated
Severe:	Loss of consciousness, seizures, coma, respiratory arrest, death	Skin:	Pink or cyan
Antidotes:	Oxygen, dicolbalt edetate, sodium / amyl nitrite & sodium thiosulphate	Secretions:	Normal
		Other factors: VERY RAPID ONSET (Secs)	
MUSTARD		NATO DESIG	H
Mechanism: Damage to DNA resulting in cell death of exposure tissue including skin and airway mucosa.		QUICK LOOK	
Mild:	Erythema (red skin), eye pain	Resp Rate:	↑
Moderate:	Skin blistering (small area), airway irritation	Pupils:	Normal
Severe:	Airway burns / obstruction, large area blisters	Skin:	Red / blisters
Delayed:	Immunosuppression, acute respiratory distress syndrome	Secretions:	Normal / ↑
Antidotes:	None	Other factors: Note mustard has delayed onset – 2-4 hrs	
PULMONARY AGENTS (chlorine, phosgene)		NATO DESIG	
Mechanism: Direct irritation of airways. Damage to cell membranes of the respiratory tract and lungs either directly or by the formation of free radicals.		QUICK LOOK	
Mild:	Eye pain	Resp Rate:	↑↑
Moderate:	Airway irritation	Pupils:	N/dilated
Severe:	Pulmonary oedema, death	Skin:	Normal or cyanosed
Antidotes:	None, possible role for inhaled steroids	Secretions:	Normal / frothy
		Other factors: Phosgene effects may be delayed or worsen with exercise.	
METHAEMOGLOBIN FORMERS (nitrites, other TICs)		NATO DESIG	NONE
Mechanism: Turns Fe ²⁺ Hb into Fe ³⁺ (Met) Hb. This prevents the red blood cells carrying oxygen from the lungs to the tissues.		QUICK LOOK	
Mild:	No obvious effect	Resp Rate:	↑/↑↑
Moderate:	Cyanosis and shortness of breath	Pupils:	Normal
Severe:	Severe cyanosis and shortness of breath, confusion, death	Skin:	Blue
Antidotes:	Methylene blue	Secretions:	Normal
		Other factors: Cyanosis not improved with O ₂ . Chocolate coloured blood.	

Box 1 Classification of Chemical Agents

overt or covert means. Some infections may be secondary to trauma either due to open wounds or contamination by gut flora. There is also the potential for bloodborne virus spread between perpetrator, co-victims and responders.

Biological agents can be categorized as *lethal* or *non-lethal*, *transmissible* or *non-transmissible*. *Pathogenicity* is used to describe live agents that have the ability to cause disease (pathogen).

Transmissibility reflects the ability of a live agent to spread from

person to person. *Infectivity* differs from transmissibility as it reflects the ease an agent can establish an infection. For example, tularemia is extremely infectious requiring only 10-50 organisms, while anthrax spores require 10⁴-10⁵ spores to establish an infection. However both are virtually non-transmissible directly from an infected patient. Some patients may therefore be infected but not infectious / contagious. Malaria is another important disease that is not transmissible from human to human and

requires an animal *vector*, in this case the mosquito [11].

Syndromic approach to biological agents – The effects of an exposure to a biological agent are best considered using a *syndromic approach* (Figure 4). Most live agents will initially present with flu-like symptoms including pyrexia, myalgia and lethargy. Toxin exposure may not be associated with prodromal flu-like syndromes. One organism may cause a number of syndromes usually depending on the route of exposure (inhalational, cutaneous and intestinal anthrax), while another organism may present with a combination of syndromes (pneumonic plague with coagulopathy) [12]. Specific biological agents can be categorised using the Centre for Disease Prevention

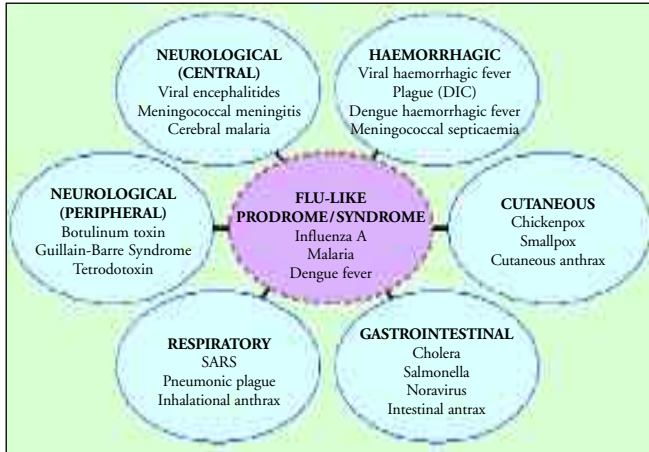


Figure 4. Syndromic approach to biological agents.

A	Botulism
	Plague
	Smallpox
	Tularemia
	Viral hemorrhagic fevers
	Brucellosis
	Epsilon toxin of Clostridium perfringens
	Food safety threats
	Glanders
	Melioidosis
B	Psittacosis
	Q fever
	Ricin toxin
	Staphylococcal enterotoxin B
	Typhus fever
	Viral encephalitis
	Water safety threats e.g. cholera
	Nipah virus
	Hantaviruses
	C
Tick-borne encephalitis viruses	
Yellow fever	
Multidrug-resistant tuberculosis	

Table 2. CDC classification of biological agents.

and Control (CDC) classification based upon risk (Table 2) [13].

Radiation And Nuclear Hazards

A radiological dispersion device (RDD) also known as a “dirty bomb” is currently more likely than a true nuclear detonation. An RDD is an improvised explosive device (IED) with a potent radiation source. Radioactive materials are used widely in

medicine, agriculture, industry, and research and relatively easy to obtain (‘orphaned sources’). The degree of contamination depends on a number of factors including the size of the explosive, the amount and type of radioactive material and weather conditions. The detonation of an RDD releases radioactive contamination although the conventional blast is more likely to cause death than exposure to the radioactive material. For this reason, traumatic injuries take priority although decontamination should be concurrent. A covert radioactive source or an RDD before activation may cause significant irradiation without necessarily causing contamination. Radiation is very detectable and the greater the hazard (doserate) the more detectable it is. A radiological attack would however cause significant psychological impact [14-18].

There are four main types of radiation, although neutrons are usually only present during the nuclear process:

- *Alpha particles* are charged helium nuclei. They only travel short distances (3cm in air) and cannot penetrate the outer layers of skin. Damage is caused if ingested, inhaled or incorporated.
- *Beta particles* are electrons. They can penetrate skin and will cause damage if taken internally. They will travel about 3m in air or about 5mm in tissue.
- *Neutrons* are non-charged particles and usually only occur during the fission (nuclear) process. They are highly penetrating and, when they interact with biological tissue, are very damaging.
- *Gamma / X-ray radiation* is at the high energy end of the electromagnetic spectrum. With no mass, it has a very long range and is difficult to shield against.

Ionising radiation causes its effect by its interaction with DNA. At low levels, there is damage to the DNA increasing the chance of mutation and cancer. The greater the dose, the greater the chance of cancer and this is called the stochastic effects. At very high levels greater than 2 Sieverts (~1000 times the annual background dose in the UK or ~100,000 chest radiographs), radiation causes cell death. This is called *deterministic* as once passed this threshold level the effects are very predictable and are due to the relative sensitivities of various tissues to radiation. The most sensitive tissues are those that have the highest turnover (bone marrow and gastrointestinal mucosa). Death is generally due to infection or coagulopathy. The failure of different systems (haematological, gastrointestinal and cerebrovascular) due to radiation is called Acute Radiation Syndrome (ARS). This occurs after an initial prodromal stage (nausea, vomiting ± diarrhoea) that lasts a few hours and a latency period of a few days. Safety measures to reduce radiation exposure are based upon *time, distance* and *shielding* [19,20].

The impact on health from a radiation incident is significant and a nuclear detonation would be catastrophic. The demand for critical care following a radiological incident is primarily due to the management of trauma with early surgery and any consequences of bone marrow failure and secondary sepsis. The lead specialties for the management of significant radiological casualties are haematology and oncology due to their experience with radiation and bone marrow dysfunction with early microbiology and critical care consultation [21].

Combined Injuries

The presentation of CBRN casualties with concurrent trauma (combined injuries) may be complicated by a complex clinical picture and worst outcome due to the synergistic effects of both insults. The presence of chemical agent effects may complicate the trauma primary survey as well as further compromise and already traumatised patient, some of these effects are listed in Table 3 [22]. Both trauma and lethal chemical agent affect oxygen delivery and

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) will affect head injury assessment. NA effects on the autonomic nervous system (including bradycardia) may cause cardiovascular compromise in a compensating hypovolaemic casualty.
Nerve agent (late)	Intermediate effects of organophosphates 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 secondary infection.
Pulmonary agents	Agents, such as chlorine and phosgene, 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 prolonged latency period and potential for delayed diagnosis or complication of blast lung.
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 (methaemoglobin formation). Supportive management with high flow oxygen is recommended.
ComboPen (atropine)	Inadvertent use of atropine may lead to anticholinergic (muscarinic) 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.

Table 3. Specific chemical agent effects on the traumatised casualty.

for this reasons the priorities of trauma and chemical casualty supportive management are the same (<C>ABC) [23].

In many ways, the release of live biological agents is more likely to be covert and not associated with trauma. Toxin may complicate wound contamination as would some spores. Biological agents are not new to trauma management and the third mode of trauma death originally described was due to sepsis, although seen less now. Most trauma patients will receive broad spectrum antibiotics as part of trauma resuscitation as well as tetanus prophylaxis prior to any surgery. These antibiotics will also cover many of the potential bacterial agents especially in the early stages post exposure.

Radiological combined injuries with significant irradiation would compromise trauma care with bone marrow suppression leading to immunosuppression and thrombocytopenic coagulopathy. Aggressive antibiotic prophylaxis and early triage for surgery during the ARS may improve outcome. Otherwise surgery is deferred until bone marrow function is restored by either stimulation therapy using cytokines or replacement therapy (bone marrow/stem cell transplantation). Following a catastrophic event such as a nuclear detonation, the presence of significant trauma including burns and a high radiation dose (>2Sv) may be a criteria for palliative care only (T4 – expectant triage category) [24,25].

Diagnosis Of Death

In the context of trauma, cardiovascular collapse is likely to be the cause of death with simultaneous respiratory arrest. However, many of the chemical agents (cyanide, nerve agents, fentanyl analogues) cause a respiratory arrest preceding cardiac arrest by several minutes. For this reason, where resources allow, resuscitation should continue in those patients that are in respiratory arrest. Formal diagnosis of death should be supported by evidence of asystole, thereby excluding profound nerve agent induced bradycardias. For mass casualty incidents, respiratory arrest should be treated as expectant (T4) with

casualties laid in the recovery position and given a ComboPen(s), if nerve agent is suspected.

Initial Management

Whether pre-hospital or in-hospital management, the priorities of CBRN management follow a modification of the conventional <C>AaBC treatment paradigm with the additional considerations of specific medical countermeasures (antidotes (a)) and hazard management (containment/decontamination/isolation). The critical care issues for the management of CBRN casualties start at the point of exposure with early antidote treatment. This is primarily the ComboPen (atropine 2mg, pralidoxime 500mg and diazepam 5mg equivalent) for nerve agent poisoning. When considering casualties within a non-permissive CBRN

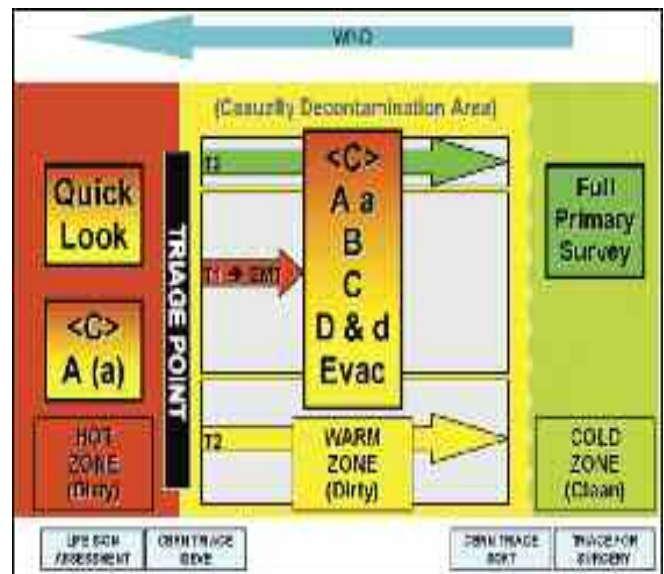


Figure 5. Zonal CBRN management.

environment (**hot / warm zone**), the same principles as Care Under Fire (CUF) and Tactical Field Care (TFC) apply and is summarised in Figure 5 [23,26].

- Safety
- Pattern (syndrome) recognition / CBRN 'Quick Look'
 - Blast
 - Penetrating
 - Blunt
 - Environmental (heat / cold / altitude / diving-related)
 - CBRN
 - Psychological
 - Other?
- Self / buddy administered antidotes, including
 - Nerve Agent ComboPens
- Life Saving Interventions (LSIs)
 - <C> Tourniquet application
 - A - Basic airway management
 - a - Intraosseous antidotes, where available
- Decontamination
 - Expose to Treat
 - Full decontamination, as permitted
- Evacuation to a more permissive environment
 - Hot Zone → Warm Zone → Cold or Clean Zone.

Further treatment in a permissive / clean environment allows:

- Full decontamination
- <C>ABC
- Supportive management as required, including:
 - Oxygen
 - Fluids
 - Analgesia
 - Broad spectrum antibiotics (benzylpenicillin, cefuroxime)
- Diagnosis
- Definitive management as indicated, including:
 - Antidotes
 - Disease specific antibiotics (such as ciprofloxacin for anthrax)
 - Surgery

Safety Considerations

During a CBRN attack military personnel may need to protect and treat patient as the medical treatment facility may be compromised. The same hazard also applies to the pre-hospital management of CBRN and conventional casualties although clinicians operating within this environment should receive specialist training. Safety considerations during any incident response are already incorporated into medical doctrine as part of the METHANE report and 'CSCATTT' assessment following the declaration of a major incident. CBRN is an additional hazard and should be risk assessed in the same way that secondary devices and environmental factors are [27,28].

The main hazard from CBRN for critical care staff depends upon whether the casualty is treated in the hot zone or in more permissive environments. For hospital staff, the main hazard is from external contamination of persistent CBRN agents and the penetration of non-persistent agents into clothing. During the 2005 Tokyo sarin attack, health care workers described symptoms following exposure to contaminated clothed casualties, although this was exacerbated by crowding and poor ventilation in ad hoc treatment areas. Off-gassing in exhaled air is rare but occasionally occurs due to ingested chemicals (cyanide salts, aluminium phosphide) or tissue saturation of volatile chemicals (immersion in trichloroethylene). In these cases, gas scavenging system may be needed [7]. Contagious patients may need to be isolated, ideally in negative pressure cubicles and increased levels of personal protective equipment should be used for high risk procedure for airborne spread such as intubation and suctioning. This was a lesson identified from the recent Severe Acquired Respiratory

Syndrome (SARS) outbreak in 2003. Wound contamination will be discussed later. Personal protective equipment can be classified into four levels (CDC level A-D) [29] as well as the usual standard precautions used for managing any patient.

- Level A – Self contained breathing apparatus and gas tight outer suit
- Level B – Self contained breathing apparatus and splash proof outer suit
- Level C – Particulate and chemical respiratory filter, includes IPE and NHS suit
- Level D – Standard precautions and high specification particulate filter mask (FFP3)

Full details are provided on the CDC website including the correct method of donning and removing PPE [30-32].

Clinical Investigations

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-Barré syndrome), sepsis (live agent, biotoxin and heavy metal poisoning) and radiation both, systemic and local manifestations (thallium/gastroenteritis and dermatitis/vesicant/ cutaneous anthrax & leishmaniasis).

Near patient 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 acetylcholinesterase levels and normal levels may be useful as a discharge criterion although lack of pin-point pupils is also a useful indicator.

Recommended investigations depending on the scenarios include but are not limited to:

Chemical incident [33]

- 10ml blood in plastic (PP) lithium heparin tube
- 5ml blood in glass* lithium heparin tube
- 10ml blood in plastic (PP) EDTA tube
- 30ml urine without preservative EDTA blood sample (*avoid vacuum bottles and rubber bungs)

Biological incident⁴

- 'Septic screen'
- Blood for cultures and polymerase chain reaction (PCR)
- Urine (culture and atypical pneumonia screen)
- Sputum
- Faeces, if gastrointestinal syndrome
- CSF, if neurological syndrome

(Field Hospitals deploy with a Specimen Collection Kit Infectious Disease(s) (SCKID) (NATO Stock Number 6545-99-235-4703)) [34].

Radiological / Nuclear incident [35]

- Nasal swabs, if contamination suspected
- Serial full blood counts (lymphocyte count)
- Blood for HLA serotyping within 12 hours, in case of transfusion

- Blood for cytogenetics (biodosimetry) at 24 hours
- Urine and faeces, if internal contamination suspected

For a CBRN/deliberate release and potential violation of the Chemical or Biological Warfare Conventions, any sample may also be used for forensics and a chain of custody should be supported with appropriate documentation or witness statements. Further information on the UK civil health response is available from the Health Protection Agency website (www.hpa.org.uk)

Airway Considerations

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

- Reduced level of consciousness (nerve agents, cyanide and some incapacitating agents)
- Direct mucosal injury (mustard)
- Increased secretions and vomiting (nerve agent)

The goals of initial airway management should therefore be:

- Removal of any airway secretions or vomitus
- Adequate oxygenation
- Adequate ventilation

Management of the airway depends on the environment (permissive or non-permissive), level of PPE 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 [36-38]. 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; this will be discussed later. If resources allow, casualties in respiratory arrest should be considered for the resuscitation and would tolerate intubation without drugs. The benefits of a secured airway are:

- Airway patency and protection
- Adequate suctioning
- High ventilation pressures and feedback on the adequacy of atropinisation for nerve agent bronchospasm
- Capnography

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 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 effecting 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 [39-44].

Respiratory Considerations

Exposure to CBRN agents has specific effects on the respiratory system. These effects can be categorised on time of onset:

- Acute
- Delayed
- Chronic

Another classification can be based upon mechanism of action:

- Direct
- Indirect
- Non-specific

Some of these effects are direct and due to inhalation of these agents. 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 particle size and water solubility. 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/botulinum exposure) or pulmonary fibrosis by paraquat ingestion. 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.

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. Specific respiratory management includes reducing the closing capacity and recruiting more alveoli either with continuous airway positive pressure (CPAP) or positive pressure ventilation with positive end-expiratory pressure (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 compare 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.

The role of inflammatory mediators following pulmonary agent exposure raises the question of anti-inflammatory prophylaxis such as inhaled steroids. There is some anecdotal evidence and animal studies that suggest there may be a benefit without any significant side effects. 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 mustard. The role and benefits of acetylcysteine is less clear and further research is required.

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.

- 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.

- 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 mustard and some of the toxins (ricin), as well as sepsis. The strategic uses low tidal volumes at the expense of retaining off carbon dioxide in order to reduce barotrauma and alveolar damage.

- Positive end-expiratory pressures - For cases of type I respiratory failure, increasing PEEP while reducing inspired oxygen may

Drug	Nerve agents
Succinylcholine	Inhibition of the acetylcholinesterases, including pseudo-acetylcholinesterase, would prolong the effect of the drug. There may also be an increase in parasympathetic side effects such as bradycardia. The period of relaxation was, however, prolonged. A significant disadvantage for this drug during a rapid sequence induction (RSI). A non-depolarising agent such as rocuronium is suggested although an increased dose is required.
Non-depolarising muscle relaxants	Non-depolarising muscle relaxants would expect to have competitive pharmacodynamics with NAs due to excess ACh competing for the ACh receptor sites. Animal models support this theory seeing adequate relaxation at higher doses. The period of paralysis was prolonged, which was unexpected.
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 does cause cardiovascular depression and this may restrict its use in trauma. Animal models suggest that NAs exaggerate the cardiovascular depression. Thiopentone may also precipitate bronchospasm, especially in the presence of cholinergics. This was not commented on in the experimental data, but should be considered.
Ketamine	Ketamine is widely used in pre-hospital scenarios due to its dissociative anaesthesia effects. It also has positive cardiovascular effects and acts as a bronchodilator. It would theoretically be the drug of choice in NA poisoning. Unfortunately, animal models demonstrate depressant effects and possible CNS side effects. There may also be increased airway secretions. These features suggest ketamine is unsuitable in NA poisoning.
Propofol	In NA poisoned animal models, propofol appears to have protective properties 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. Further research is recommended.
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. Some agents, such as halothane, may also have a role due to its bronchodilatory effects. Newer volatile agents may need further investigation.
Drug	Mustard (HD)
Succinylcholine	Succinylcholine appears to cause prolonged apnoea in mildly toxic animal models. Mustard has weak cholinergic properties. Its use in HD poisoning is not recommended and a non-depolarising agent such as rocuronium is advised.
Ketamine	Ketamine appears to have several adverse effects in mild systemic toxicity. Effects include prolonged apnoea (in contrast to its normal effect) and seizure-like muscular activity, resistant to benzodiazepines. It should be avoided.
Drug	Pyridostigmine (PYR)
Succinylcholine	PYR will inhibit plasma acetylcholinesterase, this will reduce degradation of the drug and prolong its effects. This was seen in animal models. An increase in ACh will enhance the effect of succinylcholine and so a reduced dose may be required. In the first 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 may be an alternative.
Non-depolarising muscle relaxants	Non-depolarising muscle relaxants, such as rocuronium and vecuronium, may be antagonised by PYR (PYR acting similar to neostigmine) by increasing Ach levels. This was 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 first Gulf War.
Ketamine	There is no experimental evidence to suggest any specific interaction. However, both PYR and ketamine increase airway secretions and this may precipitate respiratory distress.
Antimuscarinics	PYR will increase upper airway secretions. Antimuscarinics, such as atropine, may be required in higher doses to antagonise this effect.
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.	

Table 4. Chemical agent and drug interactions.

optimise gaseous exchange while reducing further lung damage from oxygen toxicity. The role of CPAP is 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.

Some toxins, such as botulinum, saxitoxin and neurotoxic snake venoms, cause an uncomplicated type II respiratory failure 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 [45].

Circulatory Considerations

Most CBRN agents do not cause a direct or immediate effect on the cardiovascular system. Nerve agent will cause a bradycardia that is responsive to atropinisation. 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/hr) is therefore a useful guide to circulatory status.

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. Some of the data comes from animal models. Table 4 summarises the potential interactions with chemical agents and nerve agent pre-treatment and pharmaceuticals [47-49].

Biological Agents And Surviving Sepsis

Biological agents used maliciously are likely to 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 ITU population. The treatment bundles recommended for the treatment of sepsis are listed in Table 5 [4,50,51].

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. Mustard is absorbed by tissue rapidly and would only be a hazard if a large wad of

mustard saturated material remained in the wound. The blister fluid from 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

Initial resuscitation
Diagnosis
Antibiotic therapy
Source identification and control
Fluid therapy
Vasopressors
Inotropic therapy
Steroids
Recombinant human activated protein C
Blood product administration
Mechanical ventilation strategies
Sedation, analgesia and neuromuscular blockade
Glucose control
Renal replacement
Bicarbonate therapy
Deep vein thrombosis prophylaxis
Stress ulcer prophylaxis

Table 5. Surviving Sepsis treatment bundles.

precautions will also reduced the risk. Radiological shrapnel may be present but the dose rate from the wound is quantifiable and by reducing time and increasing distance and shielding, such as using of a lead apron draped over the wound, the risk to staff can be reduced to negligible [23,52].

Operational Considerations

While on deployment, there are finite resources and to optimise a medical response to a CBRN event all staff should be aware of their roles and responsibilities during a major incident involving CBRN. Medical commanders should review their plans based upon the threat, likelihood and consequences and reflect this on the Med Group risk register. In addition to the standard major incident response, the casualty sanitisation process should include assessment of casualties for contamination/contagious and decontamination initiated as required with life saving interventions. Decontamination may be at the point of exposure or at the Emergency Department. The type of decontamination depends on the location of medical facility, availability of water and containment of the water run-off; dry decon compared to wet decon. Additional operational considerations include the management of any surge and a mass casualty (MASCAL) scenario where demand outweighs resources. The majority of the work on surge capacity has been focused on conventional terrorism [53-55].

Training

The delivery of a CBRN medical response requires not only planning, doctrine and equipment but trained personnel. Training includes the clinical management of casualties, advising command and decontamination techniques. Training delivery is likely to be at both the individual and collective level. Personnel who are likely to be involved in the critical care of CBRN casualties (Role 1 /ED/ITU and medical wards) should have training based upon their operational and/or future roles. Specialist medical training for military consultants should also include this requirement. This response is not limited to operational deployments but also contributions to the UK civil response. Many staff are now seconded or come from UK National Health Service medical facilities, especially reservists. These staff provide additional support and in many cases expertise to the UK Health Resilience capability. This dissemination of knowledge and experience has already been acknowledged for

trauma [56]. Command and non-clinical staff should also be aware of the implications of CBRN casualties and their contribution to the CBRN medical response. The UK DMS currently delivers pre-hospital and hospital clinical training (CBRN Clinical course) and non-clinical training (CBRN Advisors course). A UK civilian CBRN training framework for UK Emergency Departments that can be applied to other specialties has been proposed and is currently being piloted [57].

Summary

All Defence Medical personnel due to deploy should consider the what-if scenarios as the CBRN threat is present and the implications for the medical assets are significant. The threat is not unique to operational deployments and personnel seconded to the NHS should also be acquainted with the UK and local NHS response to a CBRN event. These events have happened and are likely to happen again. Areas of development include training, the continuing development of medical countermeasures including antidotes. The critical care skills required depends on the provider and the permissibility of the scene including the level of PPE required. The provision of early basic airway management is as important to patient outcome as later intensive care in the casualty evacuation chain. Developing the CBRN medical response needs to look at future requirements also – horizon scanning. The role of CPAP following pulmonary agent exposure is an area of research as well as the decontamination techniques and deployable negative pressure cubicles. The CBRN response however should not be seen to be a highly specialised area as incident management and casualty treatment follows conventional practices using a modified CABC paradigm. All deployed medical personnel should have CBRN awareness training including the recognition of a CBRN incident. The early recognition of a CBRN will save lives while the early exclusion of CBRN in context of trauma may save even more.

References

1. MOD. The Strategic Defence Review: A New Chapter (Cm 5566 Vol I). London, 2002.
2. Baker DJ. Critical care requirements after mass toxic agent release. *Crit Care Med* 2005; **33**: S66-74
3. White SM. Chemical and biological weapons. Implications for anaesthesia and intensive care. *Br J Anaesth* 2002; **89**: 306-24
4. Dellinger RP, Levy MM, Carlet JM, et al. Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock. *Intensive Care Medicine* (2008) **34**: 17-60 and *Crit Care Med* 2008; **36**(1) 296-327
5. AMedP6(C) – Volume III Handbook for the management of NBC Casualties (Chemical). NATO, 2003.
6. Crompton R, Gall D. Georgi Markov – death in a pellet. *Med Leg J* 1980; **48**: 51-62
7. Murray V. Chemical incidents and their management. *Chemical Incident Report Jul* 2002; 2-4
8. Joint Defence Publication (JDP) 4-03.1: Medical Force Protection. (Draft)
9. Gursky E, Inglesby TV, O'Toole T. Anthrax 2001: Observations on the medical and public health response. *Biosecurity and bioterrorism: Biodefence strategy, practice and science* 2003. **1**: 97-110
10. MOD. Joint Warfare Publication (JWP) 3.61.1 Joint NBC Defence. 2003.
11. AMedP6(C) – Volume II Handbook for the management of NBC casualties (Biological). NATO, 2003.
12. Author. Joint Medical Command (Defence CBRN Centre). CBRN clinical course material (2007).
13. Centers for Disease Control and Prevention: Bioterrorism agents and diseases. <http://www.bt.cdc.gov/agent/agentlist-category.asp>. [Accessed 27 April 2009]
14. National Council on Radiation Protection and Measurements. NCRP report No. 138: Management of terrorist events involving radioactive material. Bethesda: NCRP; 2001
15. AMedP6(C) – Volume I Handbook for the management of NBC casualties (Nuclear). NATO, 2003
16. Armed Forces Radiobiology Research Institution. Medical Management of Radiological Casualties. 2nd ed. Bethesda: AFRRI; 2003
17. Department of Homeland Security Working Group on Radiological Dispersal Device (RDD) Preparedness. Report from the Medical preparedness and response sub-group [monograph on the Internet]. Jan 2003 [accessed 6 Sep 2004]
18. International Atomic Energy Agency. The radiological accident in Goiânia. Vienna: IAEA; 1988
19. International Atomic Energy Agency. Safety reports series, No.2: Diagnosis and treatment of radiation injuries. Vienna: IAEA; 1998
20. International Atomic Energy Agency. Safety reports series, No.4: Planning the medical response to radiological accidents. Vienna: IAEA; 1998
21. Flidner TM, Friesecke I, Beyrer K, editors. Medical management of radiation accidents. 1st ed. Oxford: The British Institute of Radiology; 2001
22. Abraham RB, Rudick V, Weinbroum AA. Practical guidelines for acute care of victims of bioterrorism: conventional injuries and concomitant nerve agent intoxication. *Anesthesiology* 2002; **97**: 989-1004
23. JSP 570. Battlefield Advanced Trauma Life Support Course Manual
24. Kumar P, Jagetia GC. A review of triage and management of burns victims following a nuclear disaster. *Burns* 1994; **20**: 397-402
25. Bland SA. Mass casualty management of radiation and nuclear incidents. *Journal of the Royal Army Medical Corp.* 2004; **150** (3 Suppl 1): 27-34
26. Baker DJ. Advanced life support for toxic injury (TOXALS). *Eur J Emerg Med* 1996; **3**: 256-262
27. Henning JDR, Lockey DJ. The protection of critically ill patients in a military chemical warfare environment. *Resuscitation* 2005; **64**: 237-9
28. Advanced Life Support Group. Major Incident Medical Management and Support course.
29. Siegel JD, Rhinehart E, Jackson M, Chiarello L, and the Healthcare Infection Control Practices Advisory Committee, 2007 Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings. June 2007 <http://www.cdc.gov/ncidod/dhqp/pdf/isolation2007.pdf>
30. Nozaki H, Hori S, Shinozawa Y, Fujishima S, Takumura K, Ohki T, Suzuki M, Aikawa N. Secondary exposure of medical staff to sarin vapor in the emergency room. *Intensive Care Med.* 1995; **21**: 1032-1035
31. Christian MD, Loutfy M, McDonald LC, et al. Possible SARS coronavirus transmission during cardiopulmonary resuscitation. *Emerg Infect Dis* 2004; **10**(2): 287-93
32. Cooper A, Joglekar A, Adhikari N. A practical approach to airway management in patients with SARS. *CMAJ* 2003; **169**: 785
33. Health Protection Agency. Human Biomonitoring Developments at the HPA Part 1: Chemical Exposure Assessment Kit (ChEAK). CHaPD Report; 2007:30-1. http://www.hpa.org.uk/webw/HPAweb&HPAwebStandard/HPAweb_C/1204012993762?p=1158945066435 [Accessed 27 April 2009]
34. MOD. Standing Operating Instruction (SOI) for Medical Officers: Collection of specimen(s) from patient with suspected infectious disease(s) (Version 8). London: Mar 2003.
35. Bland SA. Management of the irradiated casualty. *Journal of the Royal Army Medical Corp.* 2004; (3 Suppl 1): 5-9
36. Okumura T, Suzuki K, Fukuda A, et al. The Tokyo subway sarin attack disaster management, Part 1: Community emergency response. *Acad Emerg Med.* 1998; 5613-7
37. Okumura T, Takasu N et al. Report on 640 victims of the Tokyo subway sarin attack. *Ann Emerg Med* 1996; **28**: 129-135
38. Wax PM, Becker CE, Curry SC. Unexpected "gas" casualties in Moscow: A medical toxicology perspective. *Ann Emerg Med* 2003; **41**: 700-705
39. Goldik Z, Burstein Y, Eden A et al. Airway management by physicians wearing anti-chemical warfare gear: comparison between laryngeal mask airway and endotracheal intubation. *Eur J Anaesthesiol.* 2002; **19**: 166-169
40. Hender I, Nahtomi O, Segal E, Perel A, Wiener M, Meyerovitch J. The effect of full protective gear on intubation performance by hospital medical personnel. *Military Medicine.* 2000; **165**: 272-274
41. Flaishon R, Sotman A, Ben-Abraham R, Rudick V, Varssano D, Weinbroum AA. Antichemical protective gear prolongs time to successful airway: A randomised crossover study in humans. *Anesthesiology.* 2004; **100**: 260-266
42. Coates MJ, Jundi AS, James MR. Chemical protective clothing: a study into the ability of staff to perform lifesaving procedures. *J Accid Emerg Med.* 2000; **17**: 115-118
43. MacDonald RD, LeBlanc V, McArthur B, Dubrowski A. Performance of resuscitation skill by paramedic personnel in chemical protective suits. *Prehospital Emergency Care.* 2006; **10**: 254-259
44. Brinker A, Gray SA, Schumacher J. Influence of air-purifying respirators on the simulated first response emergency treatment of CBRN victims. *Resuscitation* 2007; **74**: 310-316
45. Maynard RL. Phosgene. In: Marrs TC, Maynard RL, Sidell FR, Eds. Chemical warfare agents: Toxicology and treatment. Chichester: Wiley, 2007: 477-494
46. The Acute Respiratory Distress Syndrome Network: Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 2000; **342**: 1301-1308
47. Keeler JR. Interactions between nerve agent pre-treatment and drugs commonly used in combat anaesthesia. *Mil Med* 1990; **155**(11): 527-533
48. Conley JD, Lundy P. The interaction of chemical agents and therapeutic compounds with drugs used during anaesthesia. Medicine Hat, Canada.

- DRES and Kinchyle Enterprises Inc; 2001
49. Conley J, Hunter K, Lundy P, Hamilton M, Sawyer T. Domestic swine model for the assessment of chemical warfare agent-anaesthetic interactions: some effects of sulfur mustard. *Mil Med* 2000; **156**(8): 573-578
 50. Rivers E, Nguyen B, Havstad S, *et al*. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001; **345**: 1368-1377
 51. Marshall JC, Cook DJ, Christou NV, Bernard GR, Sprung CL, Sibbald WJ. Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome. *Crit Care Med* 1995; **23**: 1638-52
 52. Cooper GJ, Ryan JM, Galbraith KA. The surgical management in war of penetrating wounds contaminated with chemical warfare agents. *J Roy Army Med Corps* 1994; **140**(3): 113-8
 53. Frykberg ER. Medical management of disasters and mass casualties from terrorist bombing: How can we cope? *J Trauma* 2002; **53**: 201-212
 54. David DP, Poste JC *et al*. Hospital bed surge capacity in the event of mass-casualty incident. *Prehosp Disast Med* 2005; **20**: 169-176
 55. Shamir MY, Weiss YG *et al*. Multiple casualty terror events: The anaesthesiologist's perspective. *Anesth Analg* 2004; **98**: 1746-52
 56. National Confidential Enquiry into Patient Outcome and Death (NCEPOD). Trauma who cares? London, 2007
 57. Bland SA. Hazmat Training For Emergency Departments. *Chemical Hazards and Poisons Report (HPA)*. 2006; **7**: 45-8

CRITICAL CARE AIR SUPPORT TEAMS AND DEPLOYED INTENSIVE CARE

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Abstract

The evacuation of injured patients by air has been going on in one form or another for nearly 100 years. This paper presents some of the history behind Aeromedical Evacuation (AE), the current situation and looks to the future of this vital component in the chain of care from point of wounding to rehabilitation.

History

The first reliably documented evacuation occurred in 1915 when an unmodified French plane moved Balkan patients. The first recorded British aeromed flight occurred in 1917. This move reduced the patient transfer time from three days to 45 minutes when a Camel Corps soldier with an ankle injury was flown in a De Havilland (DH) 4 biplane to hospital in Turkey.

The first time the Royal Air Force (RAF) undertook a significant aeromedical evacuation (AE) was in Somaliland in 1919. The air ambulance was a DH9 modified to carry a stretcher and attendant, and though an experiment, quickly proved its worth. The red cross was draped over the stretcher-bearing section of the aircraft when a patient was being transferred. The fuselage opened coffin-style to allow the patient complete coverage with the attendant standing fore of the patient with his back to the pilot (Figure 1).



Figure 1. Modified DH9.

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The German Luftwaffe were the first to undertake AE missions which more resemble those of today. From 1936-41 the Luftwaffe flew missions of up to ten hours duration at heights of 18,000 feet in Junkers (JU) 52 aircraft during the Spanish Civil War.

The United States Military formed the first dedicated AE unit, the 38th Medical Air Ambulance Squadron in 1942. Using spacious transport aircraft (Douglas Skytrain and Skytrain) more than a million patients were returned to the US towards the end of WWII.

As technology improved driven by wartime necessity rotary assets became available. The first recorded AE mission using a rotary wing aircraft occurred in Burma in April 1944 using an R-4B Sikorsky helicopter. While slower than fixed wing aircraft, helicopters were irreplaceable during conflicts in jungles. The war in Korea saw the first major uses of helicopters for AE and the Vietnam war saw its development with the addition of continuing care to wounded during rotary wing flight.

Modern Critical Care Air Support Teams

The Medical Emergency Response Team (MERT) evacuates patients from the point of wounding to the Role 2 or 3 facilities in theatre. Once advanced resuscitation and/or damage control surgery have been undertaken within this setting, critically ill patients are returned to the UK using the Critical Care Air Support Teams (CCASTs).

At any time two CCASTs are immediately available for tasking. The in-theatre (tactical) team is used to move patients around theatre and the strategic team (based at RAF Lyneham, Wiltshire, UK) is tasked to return patients to the UK or other host nations. A third team is available at 6 hours notice to move (NTM) should another mission be raised while the strategic team is tasked.

Team Composition

The RAF provides the CCAST, which comprises of the following specialist personnel:

1. Two Flight nurses trained in intensive care, one of which is the Team Leader.
2. A medical devices technician to maintain the equipment.