# **Basic Principles of Chemical Defense**

## **1. Introduction to the Chemical Weapons Convention**

The information presented in this chapter gives a short summary of the Chemical Weapons Convention (CWC). The CWC serves as an introduction to chemical defense, which encompass a set of protection strategies against chemical weapons attacks. Several aspects of the CWC, such as the prohibition, disarmament, export control of chemicals, as well as policies of non-proliferation and criminalization of offenses in the context of chemical defense strategies have reduced the attractiveness of chemical weapons use in wars waged amongst the Community of Nations, excluding rogue states and terrorist elements. When defensive measures are implemented, casualties in military conflicts due to chemical weapons are minimal.

## **1.1. The Chemical Weapons Convention is special**

Treaties that ban certain classes of weapons are of little value if strict adherence to the rules of the treaty is impossible due to ambiguities in the rules or definitions. Additional difficulties arise if the treaty is not combined with sanctions or determined responses to the use or threat of the weapon's use.

The CWC remains the first and only disarmament treaty to introduce a verifiable ban on an entire class of weapons of mass destruction. In 2009, more than 188 states became party (State Parties) to the CWC. Seven states remain outside the CWC and are referred to as non-State Parties. In addition to a strict verification regime, the CWC provides rules for export controls and proliferation prevention. In addition, national laws impose criminal penalties to the development of any such weapons.

Compliance with a treaty is enhanced by the administration of specific benefits for State Parties and limitations for non-State Parties. One limitation takes the form of export controls on scheduled chemicals. (A State Party will not export chemicals to a non-State Party if those chemicals could be used to produce chemical weapons). One benefit is that any State Party threatened with chemical weapons is entitled to assistance in the areas of chemical defense, in the form of protective equipment or software from the Organization for the Prohibition of Chemical Weapons (OPCW) or other State Parties. This support includes guidance for setting up a chemical defense program to protect from military or terrorist attacks. The objective of the present document is to provide basic information on the elements of such a protective program.

The CWC includes implementation of the above-mentioned measures, and many State Parties provide active support to the OPCW and member states.

## **1.2. History**

Several attempts have been made to ban poison from battlefields. In the early 1600s, international law condemned what would today be regarded as chemical or biological warfare.

Serious attempts to ban these weapons were made with the Brussels Declaration of 1874, even before the weapons were systematically used in war. The intention to ban poisons and poisonous weapons was articulated at conferences in The Hague in 1899 and 1907. These first attempts did not have much impact in view of their widespread use during World War I (WWI). In 1922, an attempt was made to simultaneously ban submarines and chemical weapons (both considered covert weapons), but the ban never came into force. After WWI, some chemical weapons officers went so far as to promote chemical weapons (CWs) as a more humane battlefield strategy. One out of every three high explosive casualties died, whereas less than one out of ten CW casualties proved lethal, and the number dropped to fewer than one in fifty for mustard gas exposure. In 1925, the Geneva Protocol established a "Prohibition of the Use in War of Asphyxiating, Poisonous, or other Gases and of Bacteriological Methods of Warfare". Although their intentions were different, many State Parties reserved the right to use CWs in retaliation against an attack that employed such weapons. This reservation made the treaty a non-first-use agreement, and a comprehensive prohibition of CWs still was considered a necessity.

The CWC was negotiated in Geneva over the course of 22 years and was opened for signing and ratification in 1993. By April 1997, 60 State Parties had ratified the treaty, which enabled the treaty to be enforced. By the year 2009, 188 Nations had ratified the CWC. Unfortunately, some of the countries of most concern have not yet ratified the CWC treaty, but the situation is improving. Where applicable, State Parties have declared their possession of chemical weapons and initiated the destruction of these weapons, although destruction in Russia, for example, has proceeded thus far at a slow pace. Although the pace of weapons destruction has been slower than the original agreed time frame, within the next decade, most if not all of the larger stockpiles in the world are expected to be destroyed. When this occurs, the mass of CWs that might be encountered is likely to be two to three orders of magnitude smaller than was in the previous century, which represents a significant threat reduction. What remains might be a few hundred tons of CWs in the hands of non-member States. *Without appropriate chemical defense measures, these quantities have a high casualty potential.* 

## **1.3.** Aspects of the CWC

The CWC contributes to world safety through the prohibition of State Party CW stockpiles and limiting the access of non-State Parties to chemical weapons. Restricting proliferation of chemical weapons is another aspect of the CWC. Most chemicals that could be used to develop chemical weapons are subject to verification inspections. Trade and export of these scheduled compounds to non-State Parties is prohibited. Verification inspections check the handling of scheduled compounds. Export controls increase the difficulty and costs associated with the development of massive amounts of chemical weapons by non-State Parties.

If a State Party is suspected of CWs research and development or may be stockpiling CWs, the CWC may order "challenge inspections" and even an investigation into alleged activities. Although many of the nations on the suspect list, given in open sources, (e.g., SIPRI, Association of American Scientists, etc.) are State Parties of the CWC, none has been challenged thus far. The most likely explanation for the absence of challenge inspections is a lack of confidence in the information on which the suspicion is based.

The CWC contains a general purpose criterion that states that every toxic chemical and its precursors is a CW if it is used to harm life processes, whether human, animal, or plant. If an attempt is made to circumvent CWC verification inspections by developing CWs based on

non-scheduled compounds, which are not subject to verification inspections, the general purpose criterion would allow for challenge inspections. One method for making non-scheduled compounds subject to verification inspections would be to modify the schedules each time such a compound is identified. However, this method would make public the details of potential uses of this compound, such as how it may be produced. Non-State Parties could gain access to this information and circumvent the CWC. Another approach would be to state that every compound with a toxicity of less than X mg/kg is regarded as a scheduled compound. A similar criterion is used for toxins listed in the rolling text of the Biological and Toxin Weapon Convention (BTWC).

State Parties of the CWC that feel threatened by an opponent that might use CWs against them may receive chemical defense support from the other State Parties. This support may take the form of information or hardware, such as detectors, masks, or protective clothing. The CWC, together with its broad band of protective measures, makes chemical warfare very unattractive to any possible opponent. The combination of prohibition, export controls on scheduled chemicals, verified destruction of current stockpiles, and a well-organized chemical defense are mutually reinforcing measures that significantly deter potential users. The measures in the CWC form a web of deterrence in which prohibition and protection are important contributors.

## **1.4.** Changes over time

In the 1990s, the US Defense Department began publishing assessments of CW proliferation. These assessments, together with other open source reports, for example, media reports and research, were used by several organizations (see Chapter 3) for threat assessments that were then published online. During the 1990s, the main contributors to both chemical and biological weapons proliferation were countries in the Middle East and North Africa, with several countries showing increased activity. Several countries in South and East Asia were assessed as retaining probably CW and BW programs. According to those assessments, some proliferant States had become largely self-sufficient in their CBW requirements, making the task of tracking and preventing CBW proliferation activities more difficult. Two nations mentioned in those assessments deserve special attention: Iraq and Libya.

<u>IRAQ</u>: Since the withdrawal of UNSCOM inspectors, assessments of the Iraqi CW and BW programs have varied widely. Some assessments reported that the whereabouts of many precursors are unknown and BW agents have been hidden in the desert. These reports concluded that Iraq may have retained stockpiles of CW and BW agents, including in weaponized forms. The technology and expertise for the production and use of CWs and BWs had not disappeared from Iraqi, and resurrection of the CW and BW programs was a definite possibility. Despite the damage sustained during the Gulf War and the industrial deterioration associated with eight years of sanctions, Iraq was assessed as being capable of quickly restarting production of CW and BW agents. World leaders used sections of these assessments to justify the invasion of Iraq in 2003. Interviews presented in Axis of Evil, the war on terror by Moorcraft, Winfield and Chisholm form an illustration of the uncertainties in CBW threat assessment and intelligence.

Several years after the invasion of Iraq, the search for CWs, programs, or precursors has not revealed any evidence to support these suspicions. Moreover, Iraq has become a State Party to the CWC.

<u>LIBYA</u>: Libya was suspected of supporting a CW production plant at Rabta, known as Pharma 150. This plant was renovated for the production of pharmaceuticals, but again the technology and production expertise for CWs had not disappeared during this restructuring program. The lifting of sanctions (Libya admitted involvement in the downing of a passenger airliner over Lockerbie in Scotland) was thought to have encouraged Libya to restart CWs production. In a Pentagon assessment, Libya was assumed to support an offensive BW program, possibly at the R&D stages. However, the biological knowledge base for such a program was assessed as limited.

Libya has recently abolished all activities pertaining to the development and maintenance of weapons of mass destruction, has opened its country to international inspections, and has become a State Party to the CWC.

## **1.5.** The contents of the CWC

The main text of the CWC comprises a preamble and 26 articles (45 pages). The document contains two Annexes: one annex covers scheduled chemicals that may be used for the production of chemical warfare agents; the other annex describes the implementation and verification policies of the convention. The second annex is referred to as the *verification annex*. The verification annex comprises 11 sections with 4 additional annexes (100 pages in total). The construction and implementation of such a complex treaty required an in-depth review process in which several points were open to debate. Books longer than 500 pages have attempted to elucidate the legalities and correctly interpret all articles and obligations contained in the annexes. A detailed treatment of the CWC is beyond the scope of this book, although some aspects will be highlighted.

The preamble refers to the Geneva Protocol of 1925, the wish of the UN to achieve disarmament, and a statement in the Biological Weapon Convention that articulates the need for a general prohibition of the use, production, and stockpiling of chemical weapons (CW).

Article I describes 5 general obligations:

- 1. Completely prohibits CWs, including: development, production, acquisition, stockpiling, retention, transferral, or usage of CWs, also including preparations for the use of, or assistance, encouragement, or induction of others to engage in any activity prohibited by the convention;
- 2. Each State Party will destroy all CWs in its possession;
- 3. Each State Party will destroy all abandoned CWs;
- 4. All CWs production facilities will be destroyed;
- 5. Riot control agents shall not be used as a method of warfare.

Article II provides a series of definitions of chemical weapons, toxic compounds, etc., including the important General Purpose Criterion: Every toxic chemical that is used to harm life processes is a chemical weapon and thus prohibited under the CWC.

Article III declares a set of policies regarding the treatment of CWs, old and abandoned CWs, CW production facilities, and related facilities.

Articles IV and V describe the obligations of a State Party in possession of CWs or CW production facilities.

Article VI describes activities not prohibited by the convention. One such activity is the right to perform research that may improve protections against CWs.

Article VII describes national implementation measures, including the establishment of a National Authority.

Article VIII establishes the Organization for the Prohibition of Chemical Weapons (OPCW), the Conference of State Parties, the Executive Council, and the Technical Secretariat.

Article IX treats aspects of consultations, co-operation, and fact finding. This section defines the procedures for challenge inspections.

#### **1.6.** Assistance and Protection, Article X

Article X addresses assistance and protection policies against chemical weapons, which is the subject of this book. Each State Party has the right to request and receive assistance and protection against the use or the threat of use of CWs if

- a) CWs have been used against it;
- b) Riot control agents have been used against it as a method of warfare;
- c) It is threatened by actions or activities of any State that are prohibited for States Parties by Article I.

It should be noted that only under item (c) is a State mentioned. Activities of non-state actors are included in items (a) and (b). Article X is quite specific in articulating the assistance that should be provided and the time frame for that assistance once a State Party has asked for help. Assistance is granted not only in the case of an eminent threat, but also for advance preparations, particularly in setting up a passive chemical defense program. The actual assistance provided may be related to a challenge inspection, pursuant to article IX, or an investigation into an alleged use, pursuant to article X, described in detail in Part X and XI of the Verification Annex.

Specifically for this book, Article X paragraph 5 is relevant and is, therefore, presented in full:

"The Technical Secretariat shall establish, not later than 180 days after entry into force of this Convention, and maintain, for the use of any requesting State Party, a data bank containing freely available information concerning various means of protection against chemical weapons as well as such information as may be provided by State Parties.

The Technical Secretariat shall also, within the resources available to it and at the request of a State Party, provide expert advice and assistance to a State Party in identifying implementation strategies for its programs that develop and improve its protective capacity against chemical weapons."

The remaining articles treat the economic and technological development, relation to other agreements, settlement of disputes, and management aspects of the convention.

## 1.7. The CWC; Does It Make a Difference?

### 1.7.1. Introduction

Examining the history of the CWC over a 25 year period, from 1987–2012, enables us to assess the efficacy of the CWC toward its intended goals. There are good reasons for choosing to examine this period in particular. In 1987, the former Soviet Union publicly displayed its chemical weapon arsenal for the first time in history, clearly demonstrating its intention to decommission this class of weapons. The Soviet show was a response to an opening invitation presented by the US President Ronald Reagan, who had announced a change in the US policy on disarmament: "Trust but Verify". At this time, the working text of the CWC was near completion. Everyone involved knew what was at stake. The year 2012 is the date at which, according to the original text of the CWC, all chemical weapons, production facilities, etc., in the "possessor States" were to have been destroyed.

At the start of the 25-year period, two opposing blocs were prominent: the old NATO and the former Warsaw Pact. Both blocs felt strongly that the other was threatening, amongst other threats, with the use of chemical weapons. Has anything changed? Does the CWC make a difference in global chemical weapon security? Many feel that there is still a serious threat of chemical warfare, for instance, from non-member States. Many fear the use of toxic chemical compounds by terrorists. It is often pointed out that a significant fraction of the former Soviet Union stock, now controlled by Russia, will still be around in 2012. The complete destruction of weapons stocks in other possessor States was scheduled to be destroyed by 2012, but this assessment must be made. The real question is: do we believe that the CWC and OPCW have contributed to a safer world; was it worth the investment? The following paragraphs attempt to answer that question from a technical viewpoint in relation to chemical defense.

## 1.7.2. Quantifying the hazards of chemical weapons.

If a small quantity of CWs is nearly as hazardous as a large quantity, then destruction of 90% or 99% of the present stock would mean little in terms of threat or hazard reduction. To evaluate if the CWC makes a difference, it is valuable to quantify the hazards posed by the present quantities of chemical weapons.

After WW I, a discussion took place regarding the effectiveness of some weapon systems and whether or not the weapons were humane. The issue of chemical weapons incited fierce debate, in which those in favor of use stated that they were more humane than high explosives, and those opposed to use emphasized the cruel nature of the weapons. Those opposed to CWs partly won the debate, which resulted in the non-first use declaration of the Geneva Protocol in 1925.

The arguments used by those in favor of CWs were based on WWI statistics that were correct but somewhat manipulated. The statistics are discussed in detail in Chapter 3. At the moment, it is sufficient to state that the statistics must be viewed as summarizing the cumulative effects, and large margins of error may apply. Later, computational models of the CW attacks showed that for unprotected personnel in a defensive scenario, the number of effective dosages that would produce a casualty was on the order of 1 to 10 million. Another rule of thumb was that the use of 1 billion effective dosages against a military target would produce 30% casualties, sufficient to neutralize the target. For nerve agents, this dosage would be delivered by about 1 kg of the agent to produce one casualty, or 1 ton of agent to attack a military target of limited size (infantry).

The message from WWI was: relatively small amounts of CW may be effective in producing casualties amongst unprotected personnel (on average 1 ton was estimated to produce 10 - 100 casualties).

The second important lesson from WWI was that equipping troops with rudimentary forms of protection increased the amount of chemical agent required to produce a casualty. This increase was proportionally to the gear protection factor. In 1916, all British troops were equipped with some form of mask. These masks prevented the recurrence of serious Wehrmacht attacks using CWs until 1917, when the first mustard gas attacks were aimed at troops with respiratory protection. The results were devastating. Within three weeks, the British faced more CW casualties than they had in all of 1916.

The conclusion again was CWs worked when protection was circumvented, but they became almost completely ineffective when some form of protection was deployed. The WWI statistics found that if the respiratory protection factor of the troops was 10, the amount of asphyxiating gases required to cause one casualty would have exceeded 1000 kg, disregarding the explosives and containers required to transport the agents.

The short-term goal of the CWC was to reduce the quantities of chemical agents present in the world. Each ton of modern agent may still produce 100 to 1000 casualties amongst military personnel in defensive positions. The few hundred tons that are available in rogue states may be able to cause several hundred thousands of victims. If the quantity of CWs was reduced to 10,000 tons in the timeframe 1987 to 2012, a full chemical defense system would still be required. However, the picture becomes very different if protection is deployed. When the skin and respiratory system are continuously protected by a factor of 1000 (present day masks and clothing aim at these or higher numbers), the few hundred tons of CWs in rogue states would only be sufficient to attack a single target with minimal effect. Donning the full protection, in particular protective clothing takes time; it would therefore be wise to wear the protective clothing continuously during operations in a high threat environment.

#### 1.7.3. Incomplete destruction by 2012

It is unlikely that possessor States will have destroyed all CW stocks by 2012, and significant quantities are expected to remain. However, technological developments beyond the agent itself are required to start a chemical war. At the beginning of WWII, the US estimated that within the first two months of a chemical war 25,000 tons of (mainly) mustard agent would be required. This estimate is still considered accurate, and large stocks are assumed to be required to start a chemical war.

If the agent is available, the first requirement for weaponization is the establishment of a filling station that would fill shells and bombs. Next, weapon systems must be available to deliver the weapons, and trained personnel are required to carry out the missions. For instance, artillery-firing tables are required to calculate ammunition expenditure for various climatic conditions. The military doctrine for using chemical weapons, troop training, standard operating procedures, and the artillery firing tables have all disappeared from the military scene over the last 20 years. Above all, the political will to carry out a chemical war has been abandoned by the member states.

The conclusion is that even if the destruction is not complete by 2012, what remains of the original 100,000 tons does not pose a real hazard. If a hazard is present, it may be posed by the few hundred tons that are possibly available in rogue states.

The few hundred tons estimate comes from US open sources published on the Internet. Recently, a larger quantity (3000–5000 tons) was mentioned in a South Korean intelligence estimate. However, records of the intelligence community regarding this estimate are not very accurate. In 1987, the same sources estimated the quantities in the USSR to be 400,000 tons, more than 10 times the present day value. The last estimate of the Iraqi capabilities, 700 tons, appeared to be false, as was the estimate for Libya's CW capabilities. For the time being, it is reasonable to assume that a few hundred tons per rogue state is the best available estimate.

#### 1.7.4. The consequences of a few hundred tons

To neutralize the few hundred tons of CWs that are outside of the control of the OPCW, chemical defense is mandatory. Without this defensive posture, the effects of an agent might be devastating.

The available quantity of agent in a conflict will be reduced by pre-emptive strikes. In addition, superior air power will not permit a full attack to develop. The total amount of agent in a conflict will have been reduced by at least a factor of one hundred if not one thousand relative to what it was 25 years ago. This means that a chemical attack will become a rare incident and will incur few consequences, provided the troops are protected and trained. The paradox is that if the troops lack chemical defense techniques and equipment, the consequences of a CW attack might be very serious. Chemical defense is usually formulated in several steps:

The first step is to identify the agents of interest and assess the likely quantity that soldiers may encounter in the battlefield? Combined with this is the effective dosage of an agent or how much protection is required to prevent casualties in an attack. Actually, this is the ratio between the challenge dosage and the "just" no-effect dosage. The next steps are the technical chemical defense issues of

Detection / Physical Protection / Medical Countermeasures / Decontamination / Training.

With a fair chemical defense system, comprising mainly detection, protection, and training, the effects of a few hundred tons will be largely neutralized to the degree that they are militarily non-significant. However there will be encumbrances due to the physical protection and possibly psychological effects due to the use of CW. Present day detectors are capable of detecting most classical agents of interest (scheduled compounds). Problem areas sometimes mentioned are toxic industrial chemicals (TICS) and non-scheduled highly toxic compounds (Novichoks?) or synthesized toxins. Detection of TICS is not a problem because one can smell their presence. Detectors are discussed in Chapter 5

Non-scheduled nerve agents act as cholinesterase enzyme inhibitors, and enzymatic detectors successfully detect them. Methods are under development to rapidly detect pathogenic aerosols and toxin aerosols.

Two parameters, the efficiency (reduction in the challenge dosage) and the capacity (total dosage that can be stopped by the protective system) characterize the physical protection

provided to the troops. The total amount of agent, concentrations, and dosages in a future individual attack are lower than they were in the previous century. Potential opponents will not have the capabilities to carry out major attacks. The casualty acceptance is also reduced. As a consequence, the efficiency, defined as the ratio between the challenge dosage and the permissible exposure dosage, remains constant. Physical protections are discussed in detail in Chapter 6. In this picture the most common form of possible CW attack is used, a low intensity but possibly a high frequency. Some opponents might try to generate a high intensity, cold war type of attack but that automatically reduces the frequency to extremely low values due to the limited amount of agent and the limited number of weapon systems that can reach a high value (rear area) target. If such a high intensity attack would be carried out against an infantry target it might be wise to withdraw, clean up and continue operations.

The capacity required to protect against the lower quantities is considerably reduced. Multiple attacks to a single unit are no longer of interest. Secondly, the "fight dirty" concept has been abandoned, and troops will withdraw to a contamination-free area shortly after contamination. The required protection times and, therefore, the capacity are reduced. Consequently, the number of spare canisters (filters) can be reduced, the number of protective suits, gloves, and boots can be reduced, protective clothing can be made lighter, and the decontamination of suits after exposure to chemical agents becomes superfluous.

TICS should not pose a real threat. The locations of large TICS storage sites in areas of operation are known. Operations in the vicinity of storage sites should be prevented. The canisters used currently to store the compounds release small amounts of TICS, as reflected in the detectable chemical levels encountered some distance from a storage site. Special canisters are required if operations will approach the storage sites at closer range.

Because chemical warfare will be reduced to rare incidents with very limited numbers of casualties, it is unrealistic to pay enormous attention to medical countermeasures for chemical casualties within the military. In contrast, medical countermeasures are one of the few aspects of chemical defense that ameliorate civilian casualties. Large sums have been spent over the past 90 years in a search for treatment of, as an example, mustard gas poisoning. Success has been elusive, and breakthroughs are not expected in the near future. Medical countermeasures are discussed in detail in Chapter 7. The extensive research toward treatment therapy and prophylaxis for CW agents would better be spent in the area of countermeasures for biological and toxin weapons.

Decontamination of CW agents will seldom, if at all, be used in future conflicts. Nuclear and biological decontamination may be of interest, but nothing more complex than water rinses will be sufficient to reduce exposure at sites to acceptable levels during operations. Chemically contaminated equipment will be abandoned, and clean-up might become of interest only after the conflict ends. No military wants to transport equipment that has been contaminated, because a toxic-free guarantee is impossible outside of a laboratory. Decontamination is described in detail in Chapter 8.

In view of the updated CW status, it is mandatory to establish new doctrines and new forms of training with respect to chemical defense. Without training, chemical defense systems will not work adequately, and the few hundred tons of CW may become very hazardous again.

#### 1.7.5. Does the CWC make a difference; or, where is the CWC dividend?

Technically, the main success of the CWC is the significant reduction in quantities of CW agents intended for use in conflicts. Chemical warfare will be restricted to rare incidents. This has consequences for the defensive posture of armed forces. A lower emphasis on medical countermeasures and decontamination may be applied. Detection remains crucial, but less emphasis is required on the capacity of physical protection. These conditions reduce investments in canisters and in the number of suits required per person. Protective clothing can be made lighter, which will reduce the physiological burden of a mask and clothing. Physical protection, decontamination, and medical countermeasures are the main areas in which a return on investment in the CWC can be found. Training of the adjusted chemical defense posture is essential. An important aspect of the training is anticipating and preparing for the psychological effects of a chemical or biological attack. If this is not adequately covered, a minor attack could quickly paralyze large groups. Although there are undoubtedly more efficient means of killing large numbers of people, none are as terrifying to unprepared troops and civilian populations, and, therefore, no other attack carries as great a psychological impact as chemical or biological agent exposure.

When the military is properly trained, few casualties will be incurred and the terrifying effects are reduced. In this case, the CWC and chemical defense will work together toward a common future goal, to make chemical warfare an issue of the past. The motto of this book is, therefore:



## "Trust, Verify, but Protect".

During inspections, complex pieces of equipment are often encountered, from ammunition to rockets or missiles. Detection of leakages and decontamination can become complex. However, inspectors are well-trained and well-protected.

## 2. Protection against chemical and biological agent attacks

## 2.1. Chemical and biological agents

In principle, the Chemical Weapons Convention (CWC) (1) is concerned with chemical agents, usually manmade compounds, although some toxins are included. In the future, the distinction from the Biological and Toxin Weapon Convention (BTWC) (2), concerned with agents of biological origin, might become even more blurred due to the increasing possibilities of synthesizing toxins and even viruses (In 2001, the polio virus was synthesized from mail order house chemicals.)

Chemical (C) or biological (B) incidents have very little specific signature. Unless an attack was announced as C or B or advanced detection equipment was available, it is most likely that the incident of release of a biological agent would go unnoticed. Chemical incidents, which are usually associated with a distinct odor and rapid casualty development, will be the incidents to which emergency services will respond. Biological incidents would require a medical response. When more is known about the type of agent released, more specific countermeasures can be taken.

From the defense and protection point of view, many countermeasures may be applied toward both chemical and biological agents; individual protection, collective protection, and decontamination apply to both C and B agents. The areas of detection, identification, and medical countermeasures include distinct differences in a response to C or B agents.

Because this book was initiated by Article X of the CWC, it should cover only C agents. However, as argued above, because the distinctions between the response to C agents and the response to B agents are often blurred and sometimes nonexistent, both types of agent will be covered here, with an emphasis on chemical agents.

## 2.2 Methods for defending against CB attacks

During and after WW 2, CB attacks were discussed mainly in the framework of military operations. Few countries (Israel, Sweden, and Switzerland) provided protection for the civilian population against these attacks. Although attacks against a civilian population are now considered (see 2.4), most of the protection concepts originate from consideration for classic military scenarios. The military defense strategy with respect to chemical and biological weapons typically relies on four pillars:

- Passive defense measures, all forms of protection;
- Active defense, including determined responses;
- Verifiable arms control regimes;
- Non-proliferation by control of relevant materials, for example, hazardous materials or their precursors, (bio) reactors, etc.

Although each individual pillar will reduce the threat of chemical and biological weapons, none of the pillars is absolute. Therefore, a combination of pillars forms the best available web of deterrence.

In this respect, the opinion of the relative value of the pillars by an Alliance, such as NATO, is relevant. "The Alliance's defense posture against CB weapons and their delivery means must continue to be improved. This will include work on missile defense with the aim of deterring and defending against the use of CB weapons. The alliance's strategy does not include a CB capability. The Allies support universal adherence to the relevant disarmament regimes. But, even if further progress with respect to banning chemical and biological weapons can be achieved, defensive precautions will remain essential." (Statement from the Washington Summit; 50 years of NATO, 1998).

Arms control regimes and non-proliferation pillars are less effective in the case of terrorist involvement in CB attacks. Active defense through which the capabilities of terrorist groups are reduced to the degree that they no longer form a hazard is extremely difficult if not impossible. Often it is not known who the opponent is until an incident has taken place, and the identity of the terrorist may not be known for some time (US 2001 Anthrax letters).

In any case, arms control through CWC and BTWC should go hand in hand with passive defense to reduce the probability of a CB incident and to reduce the casualty numbers as much as possible in the event of an attack.

## 2.3. Chapters

To cope with any type of CB incident, the military has traditionally relied upon the countermeasures of:

- Detection, including warning of a potential hazard, detection of the type of hazard, monitoring the course of the hazard over time, and identifying the exact nature of the hazard to support medical countermeasures.
- Physical protection, including respiratory protection, masks, skin protection in the form of protective suits, gloves and boots, and collective protection inside an enclosed space where ingress of agent is reduced.
- Contamination control and decontamination; Contamination control prevents the spread of contamination and decontamination involves cleanup.
- If these protective measures have failed to fully protect the potential victims, medical countermeasures are needed to reduce fatalities. In general, two medical countermeasures are used. One is prophylaxis, which is a part of protection because it is administered prior to exposure to enhance the resistance of an individual. The best known examples of prophylaxis are vaccinations against common diseases. The other medical countermeasure is therapy that attempts to cure a victim. In contrast with the physical protection, which is non-specific, medical countermeasures are usually highly specific.

Warning the civilian population of a CB incident is very difficult. Physical protection of the civilian population in most countries relies on duct taped sealed rooms. Although chemical agents act quickly, decontamination of victims can reduce casualty numbers and help protect the first responders. Consequently, protective measures applied to the civilian population emphasize therapeutic measures. (Prophylaxes for several types of potential CB agents are not available. Prophylaxis administered after contamination is regarded as a therapeutic measure.)

To develop countermeasures, it is of vital importance to identify the types of agents that might be encountered and the quantities or dosages. This is generally called threat analysis or hazard analysis.

Equally important is to know the concentration levels or dosages below which agents are no longer hazardous. Together, these two aspects guide the degree to which outside exposure must be reduced to safe levels, e.g., by physical protection or prophylaxis. The non-hazard threshold guides detector development and sets the required levels of decontamination. For this reason, this book is organized in the following way:

Chapter 3: Threat; Chapter 4: Human Toxicity Estimates; Chapter 5: Detection; Chapter 6: Physical Protection; Chapter 7: Medical Countermeasures; Chapter 8: Decontamination;

Annex A presents some background information about chemical warfare agents.

As with all emergency response measures, it is essential to train procedures thoroughly in realistic field exercises. This book is not designed as a training manual; instead, this book provides only the background information required for dealing with CB incidents.

#### 2.4 Response to Terrorist Incidents

The response to a non-military chemical or biological incident comprises essentially the same steps as the response to a military incident, but the task is more complex and requires the cooperation of many services, each with their own expertise and operating procedures. The services involved comprise emergency response teams, police, and medical services, including ambulances and medical personnel. Civil defense organizations are sometimes available or special military units come into play to mitigate the effects of an event. Incidents may occur at facilities, such as a metro station or a chemical plant, that require local expertise. In responding to a problem it is, therefore, important that all personnel involved are trained to work together and the strategy employed to address an incident should be common knowledge across all services involved, particularly among those implementing the command structure. Generally, first responders follow standard risk management procedures in a series of steps. It is important to note that although the generic steps are the same; the approaches to chemical versus biological incidents differ widely, and approaches to toxin incidents fall between these classes of incident. One difference is the time over which an incident develops. A comprehensive description of risk management has been provided by the group of experts that produced the report on chemical and biological weapons for the World Health Organization. A short summary is provided here to outline the process.

After an incident involving toxic chemicals, casualties appear within minutes and the effects may develop on the time scale of hours, up to 24 hours. To have a widespread effect, kilogram quantities of the more toxic chemical agents must be released. Among toxic industrial chemical agents, release must be on the order of tons, and both releases most likely will be noticed. If the release itself is not noticed, the location of the casualties will rapidly indicate the location of the hot zone. The most hazardous (high) concentrations introduced in a chemical incident typically last no more than a few minutes. If the hazard is spread over

longer times, the concentration involved is reduced by diffusion or decomposition. The most important response action that will save lives is to evacuate the site of release and to move persons to non-hazardous areas. Responders need some degree of protection, but it is extremely unlikely that they ever will see high concentrations of hazardous compounds during terrorist incidents.

Two examples of chemical incidents demonstrate the timeline of an adequate response. Consider, for example, a truck loaded with a volatile hazardous compound that is exploded in the vicinity of a staging area. The agent will evaporate quickly, a cloud of appreciable concentration will form within 15 minutes, and the cloud will drift over the target area. With a low wind speed of 1 m/s the front of the cloud moves at a rate of 3.6 km/h. For most hazardous toxic industrial chemicals, the concentration (dosage) will decrease to non-lethal levels after the cloud travels 1–2 km, on the order of half an hour. (For higher wind speeds, the velocity of the cloud increases and the concentration is inversely proportional to the wind speed. At 5 m/s wind speed, the hazard zone is 0.2–0.4 km, and this distance is covered within ten minutes.) The conclusion is that the timeframe for protective actions employed by responders is very short.

As a second example, consider the well-known Tokyo metro incident of 1995. Terrorists released a total of one kg Sarin inside several metro cars and stations in the Tokyo subway system. The incident developed within a few minutes. People panicked and left the subway stations as quickly as possible. One courageous metro employee seized one of the packages containing the agent and brought it into the open. In doing so he was exposed to such a degree that he died. Because the incident occurred in a relatively enclosed environment, the hazardous concentrations were present for a longer time, but after 15–30 minutes the concentrations had been diluted such that only simple respiratory protection was needed.



Tokyo March 1995; the second large-scale terrorist attack using a homemade nerve agent. Some months earlier, the same group had launched an attack in Matsumoto. 13 people died in the attack. One thousand victims suffered from effects of nerve agent poisoning. Over 4000 other subway travelers sought medical attention. During the commemoration of 2005, it appeared that a large fraction of the victims still showed some effects. including psychological effects. After 14 years, one more victim died. Exposure to Sarin was partly responsible for at least one premature death.

A large number of healthcare workers who responded to the incident became exposed due to off-gassing of the agent from the hair and clothing of the primary victims or casualties. Protection of healthcare workers against secondary hazards would have been appropriate.

Incidents involving pathogenic biological agent microorganisms develop on a much longer time scale. If the release is not observed (witnessing a release will be an exception because of the small quantities of agents involved) or has not been announced (terrorists may announce an attack or intent to conduct an attack to cause panic, even though such a warning might reduce the spread of disease) effects may not become apparent for usually more than one but up to several days. Response actions at this point are now mostly medical. It must be determined if the disease is contagious and whether affected persons should be quarantined. Hazardous concentrations of airborne biological agents in the area of release will dissipate rapidly, leaving only some surface contamination.

We shall consider two examples of biological incidents. The first example consists of the only known terrorist attack involving a biological agent, in which a sect in the US sought to influence local elections by contaminating the salad bar in a restaurant with Salmonella. During the night and the following day, several people came down with serious diarrhea. If present in sufficiently high dosages, salmonella will take effect quickly. As an example, at a street party in a suburb of Dusseldorf, Germany, a potato salad with homemade mayonnaise was served. Within two hours of ingestion, people began to leave not feeling very well. Within 5 hours, everyone who had eaten a serving of potato salad was sick and 1 hour later ambulances brought the most serious cases to the hospitals. No one died in this case, but it is not uncommon that Salmonella infections are fatal to the elderly.

The second example concerns infections with the Legionella bacteria. The disease was discovered in the US during a 1976 reunion of veterans. Tens of veterans died from mysterious causes before the bacterium causing the disease was found in the hot water tanks of the hotel in which the veterans had stayed. In 1999, during a flower exhibition in the Netherlands, fountains of water containing Legionella contaminated the air and tens of visitors fell ill in the days and weeks following the visit to the exhibition. It took several weeks before the cause of the small epidemic became clear and the "hot zone" was discovered. It is not clear how many people were exposed and how many fell ill. At least 32 victims died. Estimates led to the conclusion that the infection was lethal in 20 to 30% of cases.

Toxic effects in patients take between a few hours and a few days to develop symptoms. Only a few grams of the more highly toxic toxins are required to pose an effective threat, so a release event would probably not be noticed. The situation is similar to the release of microorganisms, and the response will be medically directed. An important difference is that the toxins never produce a contagious disease. Again, toxins are most damaging when exposure occurs through the lung tissue. To affect a large number of people, particles should be so small that they float in the air. Larger particles will settle in the vicinity of the release point and even if they became airborne they would not reach the lungs because they would be trapped in the upper respiratory tract.

The conclusion is that the development of casualties over time is the most important indicator of the type of incident: chemical or biological/toxin. This indicator guides risk management in the follow on steps:

- i. Identify the hazard;
- ii. Evaluate the situation and determine the initial risk;
- iii. Apply risk reduction;
- iv. Determine residual risk. Is it acceptable or should it be further reduced?
- v. Monitor the program. Is everything working as expected?

| Chemical   | Biological  |
|--|---|
| (1) Identify the hazard<br>Detect hazard zone using rapid detectors Sampling<br>and Identification by specialists.   | Identify the agent. Define a sick person:<br>a difficult decision must be made<br>regarding who will benefit from<br>treatment. Determine the distribution of<br>casualties. Determine the hazard zone.<br>Where did the agent originate, what was<br>the nature of the attack? |
| (ii)Evaluate the situation and determine the initial risk<br>Assess the type of release that has occurred Assess   | Is the infection contagious?  |
| the quantities involved? Evaluate the impact<br>on response? Carry out downwind hazard area<br>prediction. (Use military procedures not models<br>based on Gaussian plume). Assess potential<br>casualty numbers.  | Spreading of disease. Assess casualty management required capacity.   |
| <ul><li>(iii) Apply risk reduction</li><li>Implement risk communication based on<br/>information and instructions on how to handle<br/>Protect responders to the extent required.</li><li>Prevent spread of contamination.</li><li>Decontaminate casualties to prevent exposure of<br/>medics or ambulance personnel. Triage casualties.</li><li>Provide medical care and evacuation of casualties</li></ul> | Risk communication and instructions to<br>the affected population. Protect<br>responders and healthcare workers.<br>Introduce prevention procedures.<br>Conduct triage.<br>Provide medical care to casualties.  |
| <ul><li>(iv) Determine residual risks</li><li>Assess if resources are adequate.</li><li>OPCW assistance required?</li></ul>  | Assess if resources are adequate.<br>International assistance required?   |
| <ul><li>(v) Monitor program</li><li>Monitor effectiveness of prevention and control and adjust if required.</li><li>Rehabilitation of hot zone if contamination present.</li></ul>   | Monitor hazard level on the site of<br>release. Assess potential long-term<br>effects on casualties.<br>Rehabilitation of the hot zone.   |

It is obvious from the above considerations that protection of the civilian population and responders to CB incidents will be different from protection of the military. Protection from biological incidents must be sought mainly through medical countermeasures. It is of little use to protect the first responders to the highest possible degree because they will not be exposed to a hot zone situation. Chemical incidents develop much faster in time, and protection of the civilian population must be improvised, e.g., by evacuating the hot zone or finding shelter inside houses with windows and doors closed. Again, it is highly unlikely that first responders will need protection from secondary contamination, e.g., low vapor concentrations from residual liquid or small not evaporated droplets that have settled on a surface.

#### 2.5. NBC/HAZMAT/CBRN incidents

The three abbreviations in the title describe different types of incident. The most common understanding is that NBC stands for nuclear, biological, or chemical incidents in war directed mainly against military, such as exposure to vapor, liquid drops, aerosols, or detonated nuclear devices. In almost all cases, it will be obvious that an attack has occurred through detection, a system for reporting, and the ability to predict the downwind hazard. The military can don the required protective equipment and mitigate the effects through decontamination and medical countermeasures.

HAZMAT indicates an incident with hazardous chemicals at an industrial site or during transport. It mostly involves quantities of more than 100 kg to several tons. It will be obvious if and when an incident is developing. Only those who approach the source might risk liquid splashes. The agents involved are rapidly identified. First responders have protective equipment that can provide protection against most if not all agent. The population in the downwind hazard area can be ordered to take shelter inside houses or other suitable structures. In extreme cases, populations can be evacuated.

CBRN incidents indicate the terrorist use of chemical, biological, radiological, or nuclear weapons against the civilian population. The indicator that an incident has taken place will, in most cases, be the occurrence of casualties: for chemical attacks, within minutes to hours after release; for biological attacks, after several hours to days; for radiological attacks, after weeks to months; and for nuclear attacks, within seconds. If the release of the agent is noted or casualties occur quickly after the release, first responders will be alerted and can appear on the scene. Equivalent to the response of fire brigades to local fires, the response time can be as long as 15 minutes. In those 15 minutes, the CBRN agents may become largely dispersed in the environment. Pockets of liquid chemical agent or radioactivity may remain at hot spots near the release. If they are not evaporated or dispersed they produce only low concentrations of hazardous compounds in the atmosphere. Detecting hot spots and identifying the agent involved is a laborious process that requires hours or even days. Several rescue services have taken the position that protection of responding personnel, as in the case of HAZMAT incidents, is mandatory. This requirement makes the response process even more timeconsuming in situations in which the fastest response possible is required to reduce casualties. However, because contamination as a result of liquid splashes is extremely unlikely, and vapor and aerosol concentrations will have reduced considerably by the time of arrival at the incident site, less elaborate protection should be more than sufficient. Time should, therefore, be used to save casualties instead of donning complex protection.

The UK Police correctly stated at the VIIth CBRN Symposium at Shrivenham in October 2004: "CBRN is not NBC, CBRN is not HAZMAT". CBRN might have some similarities with NBC and HAZMAT but it should be treated as a separate type of incident requiring its own type of dedicated countermeasures. This book discusses two of the situations: NBC for military incidents during war and CBRN for civilian incidents.

## 3. The Threat from Chemical and Biological Warfare Agents

## 3.1. General

### 3.1.1. Introduction

Over the past two decades, the geopolitical situation in the world has changed considerably. The two major power blocs of the past, NATO and the Warsaw Pact, are no longer opponents and, thanks to an effective Chemical Weapon Convention, the large stores of chemical weapons are undergoing destruction. It is expected that in the next decade these 60,000+ tons CWAs will be destroyed. Similarly, programs that develop biological weapons have been abandoned.

The reduction of threats through the Chemical Weapons Convention (CWC) (1) is significant. Negotiations with the goal of strengthening the Biological and Toxin Weapon Convention (BTWC) (2) are ongoing. The CWC imposes a strict verification regime and trade limitations with non-State Parties and has been in effect since 1997. Threat reductions through the CWC and BTWC (when it becomes effective) should only improve in the next decade.

Chemical and Biological (CB) terrorism has become a reality in the past decade. The most infamous example is the Sarin attack in the Tokyo metro in March 1995. Another example is the anthrax letters in the US in 2001

#### 3.1.2. Sources of information

Countries that have their own intelligence systems will usually make assessments regarding the CB capabilities of potential opponents. These reports are mostly classified and are not directly available. Excerpts from the US reports are made available on a yearly basis in a CB threat review from the US Defense Department. This report discusses the types of agent involved and the countries and terrorist organizations of concern to the US. Recent events with regards to Iraq and also the historical record of CB intelligence dating back to WW I have shown that the reliability of CB threat assessments is poor. Situations of both over- and underestimation of an opponent's capabilities have occurred frequently. However, several open sources provide information (3–8). The reliability of this information is uncertain, although it is expected that a group of WHO consultants (9) will be able to provide an unbiased review of threats. Similar information is generated by the Swedish Stockholm International Peace Research Institute (SIPRI) (10). Both internet information and the WHO and SIPRI reports contain references to original documents from which information can be derived.

The information provided in the various sources is not always consistent. In addition, extensive research performed over the years has shown that some of the specific threats reported are less likely to occur. A scientific and critical review of the information quoted by the various sources is impossible, although from time to time these sources may indicate less likely threat agents.

#### 3.1.2.1. Incorrect assessments

A few examples from earlier publications (3) illustrate that in the past 100 years,

- (1) The military assessment of the use of chemical and biological weapons,
- (2) The intelligence regarding an opponent's capabilities,
- (3) The scientific aspects of chemical and biological weapons, and
- (4) The reporting in the media

often have led to a completely incorrect assessment of a threat of biological and chemical weapons. In fact, misunderstandings and misinterpretations have occurred so frequently in history that extreme caution should be taken when presenting new intelligence data or using the data for justifying an action to counter the supposed threat. Both the scientific community and military analysts have often wrongly assessed the capabilities of C and B weapons, but the intelligence community and media have most often provided unreliable information. This is at least partly due to the mythic perceptions of chemical and biological warfare. Often, the casualty potential from an agent is estimated from the ratio of the total amount of agent to the lethal dosage per individual, disregarding all loss due to dissemination and dispersion. WWI statistics as well as modern computer simulations show that in a military scenario, an average of one million times an effective dosage must be released to produce a single casualty.

#### 3.1.2.2. The start of chemical warfare

Despite the possibilities discussed during the Brussels convention and the conferences in The Hague around 1900 on the control of chemical weapons, despite the use of many types of chemical weapon during the early stages of WW I, despite the information provided by several prisoners of war and defectors, the massive chlorine attack of April 1915 came as a complete surprise.

The effect of chemical weapons was new to the German military, and they did not exploit the sudden advantage that was achieved. At the end of WWI, experts agreed that CWs were effective against an unprotected opponent, but were ineffective against protected troops. In fact, it is useless to attack protected troops with chemicals.

#### 3.1.2.3. The mustard gas case

In the search for new weapons that could break the protection provided by masks, both sides in WWI screened many compounds. In 1916, the UK rediscovered a compound, mustard gas, synthesized for the first time nearly 100 years earlier. It caused serious blisters but the British military rejected the compound as ineffective because it did not kill. Angry young scientists wanted to prove the effectiveness of the compound and placed a drop on the chair of the Director of Porton Down. He had to eat his meals from the mantelpiece standing for a month. One year later, British troops were attacked with mustard agent and in three weeks faced more casualties due to chemicals than in the twelve preceding months. The German scientific community, not aware of the fact that the UK had produced these compounds, told the military that they did not have to fear retaliation on the grounds of the difficulty for the British to identify the exact structure of mustard gas. The UK had established the correct formula within a week. In those years, mustard gas was called the king of the war gases. Even today, it is one of the most effective CWA.

#### 3.1.2.4. The dusty mustard agent case

The 1938 Italian forces in Libya introduced mustard agent that had been adsorbed onto a fine clay powder. The purpose was to increase the persistence of the agent in the hot desert and to facilitate dispersion by spraying. The clay concept was shared with the Nazi German allies of Italy during WWII, and investigations were carried out regarding the effectiveness of the dusty agent in the Kaiser Wilhelm Institute in Berlin. A handwritten report from 1944 (historical archives of the Wehrmacht in Freiburg, Germany) mentioned that those who carried out the experiments had protected their hands and arms with an impermeable material, but still they developed blisters in the wrist area. This finding induced the claim that dusty mustard was much more effective than ordinary mustard agent, ignoring the fact that the deposition of dusty mustard in the opening between the hand and arm protection on very wet skin may have contributed to the severity of the effects. Thus far, no studies have been published demonstrating that dusty mustard is more aggressive for humans than ordinary mustard agent or would cause more severe effects at a lower dosage. Dusty mustard agent was never introduced into the German army. Nevertheless, the dusty agent problem pops up from time to time, most recently in the attacks on the Kurds in the late 1980s.

#### 3.1.2.5. The HCN Cases

The use of HCN stretches over many years, even as far back as the Napoleonic Wars. In a later case, a German pharmacist suggested dipping the bayonets of the Prussian soldiers in cyanide to be more effective against Napoleon's troops. During the screening of potential CWAs during WWI, one of the first agents to be examined was HCN. Initially, there appeared to be significant difficulties in weaponizing the agent. (During testing, the HCN exploded or burst into flames.) Eventually, the French army succeeded in developing a usable form. To produce a sufficiently high concentration, they used a rapid firing 75 mm gun. Porton Down advised strongly against the use of HCN because in their opinion it was extremely difficult to create a sufficiently high concentration of HCN in the field. In a very illustrative and bold experiment, a researcher (a professor) and a dog were sealed in a gas chamber. When the gas chamber was filled with HCN, the professor continued to quietly read a book and the dog died in a matter of minutes. The professor knew that dogs were much more sensitive (at least ten times) toward HCN poisoning than humans. The French form of HCN was not very effective, and, as the record shows, once the German troops smelled the HCN they did not bother to don masks because they liked the smell (the smell is almond-like).

Despite this experience, HCN resurfaced in WWII. The German counter intelligence discovered that the Soviet Union (SU) used aircraft to spray HCN. Due to the boiling point and density of HCN gas relative to air, this seemed physically impossible. Germany carried out an experiment in Munsterlager, which failed. After capture of an SU spray aircraft, the experiment was repeated, this time with great success. The trick was that very large drops were sprayed. A fraction of the HCN evaporated during the free fall and cooled the agent to such a degree that it froze or water/ice from the atmosphere condensed onto the drops. The agent was described as HCN snow. Additional experiments were carried out using dogs, and the effectiveness of the agent was determined by the number of dogs that were killed. In some experiments, actual concentrations and dosages were measured. Seldom was a concentration or a dosage found that would kill a human. Nevertheless, HCN remained on the threat list, and it appeared in a 1969 study book from the former German Democratic Republic, East Germany: NATO was accused of possessing HCN snow weapons.

#### 3.1.2.6. The nerve agent case

The first Nerve agent was synthesized by Schraeder and co-workers in 1936. Industrial production of Tabun and Sarin began sometime between 1941 and 1942. Several thousand tons were weaponized but fortunately never used. The British counter intelligence received some reports about the German developments. After careful analysis, including scientific screening, the conclusion in 1944 was that such highly toxic agents did not exist and that the reports must be viewed as Nazi propaganda. One year later, British troops were involved in the demolition of the storage bunkers in Munsterlager, and they dumped CWAs into the Baltic Sea. The next group of even more toxic nerve agents, the V-agents, was developed in the UK some ten years later.

#### 3.1.2.7. The cobweb case

In 1939, guards on the Southern coast of the UK reported a phenomenon with potential implications for biological warfare. Cobwebs were floating through the air. The UK scientists quickly ascribed this to natural phenomena: each fall, spiders migrated by floating in the air. This phenomenon sometimes was observed in unusual intensity. In the 1890s, the sky around Chicago was blackened from cobwebs. In another example, during his trip to the Galapagos Islands, Darwin discovered migrating spider webs on board a ship 100 km outside the Rio Plata. Cobwebs appeared once more as potential agents during the Serbia–Croatia conflict in the 1990s, and some cobwebs were collected. After analysis, a manmade compound (poly ethylene glycol) was found, however neither the sample-taking nor the analysis complied with the strict rules of the OPCW. Cobwebs as biological warfare agents must be rejected as fairy tales.

#### 3.1.2.8. The yellow rain case

In the late 1970s and early 1980s, the use of yellow rain as a biological warfare agent was discussed. A much disputed analysis of a sample from Southeast Asia indicated the presence of trichotecene mycotoxins. The Secretary of State of the US, Alexander Haig, accused several countries of being involved in biological warfare. The book <u>Yellow Rain</u>, by Seagrave, described several incidents but independent investigative teams did not agree on the facts and explanations. The debate continued for some time until an alternative explanation for the yellow rain was presented, namely, bee droppings. Bees' droppings contain large amounts of yellow pollen. If these droppings are released during a rainstorm the surface tension causes the pollen to concentrate on the outer shell of a raindrop, turning the drop yellow. The likelihood of this explanation was increased when the Russian expert on mycotoxins, Joffe, who was living in Israel, declared that the effects cited by the victims could not be due to mycotoxins. Some years prior, Joffe had obtained from the Soviet Union (via mail) the most virulent strains of the fungi for experimental production of the toxin. He worked with those compounds in a minimally equipped laboratory close to the Israeli parliament without any special protective measures and never observed skin effects.

The "Yellow Rain" case was further discredited in a study by Tucker in 2001 (J.B. Tucker, "Yellow Rain" controversy: lessons from arms control compliance. The Nonproliferation Review, 29 January 2001, 8:25–42).

However, in 2004 another yellow rain incident was reported, this time in India 60 km north of New Delhi. At the time, considerable tensions between India and Pakistan led to the

circulation of rumors of biological warfare. In a matter of weeks, scientists in India declared that the phenomenon was due to yellow pollen from bee droppings. However, some investigators still believe that the Kurds were attacked with mixtures of mycotoxins and mustard agent. The evidence presented was not very convincing.

#### 3.1.2.9. The Angola case

In the late 1980s, accusations were made that the Angola government, supported by Cuban and Russian troops, used an agent against the rebel forces (UNITA), which were supported by South Africa. Medical personnel from South Africa indeed found a number of victims all with paralyzed extremities. Despite thorough investigations, no reasonable explanation was found. A year after the reported incidents, a toxicologist from Belgium conducted further research on some of the victims. During a televised session, the toxicologist showed that one year after exposure, the Chemical Agent Monitor gave a positive response to nerve agents, which seemed physically impossible. No one could duplicate this finding, but many believe the findings to be correct. As of today, no absolute proof has been presented of exposure to any agents. A more likely explanation is that the rebel forces prepared their food in gun oil or oils from tanks, armored vehicles, or downed aircraft. These oils contain tri-ortho-cresyl phosphate, a compound known to cause paralysis of the extremities. Similar incidents occurred accidentally with the Swiss army in 1939 and in Spain and Morocco when criminal merchants mixed olive oil with machine oil. References to the cases described above are given in the original publication.

## 3.1.3. Definitions

It is difficult to make a clear distinction between chemical and biological weapons. One class of biological weapons are microorganisms and other self-replicating entities, including viruses, infectious nucleic acids, and prions, the intended target effects of which are due to infectivity (9). The pathogenicity of some biological agents arises from the toxic substances that they themselves generate. These toxins are sometimes isolated and sometimes synthesized. As the name indicates, these toxins work through their toxicity, not infectivity, and therefore fall under the definition of chemical weapons. Originally, all toxins were of biological origin and were regarded as biological weapons (9).

Chemical agents work through their toxicity. The CWC defines a chemical action against life processes, as causing death, permanent harm, or temporary incapacitation. Chemicals used as propellants, explosives, incendiaries, or obscurants may also have toxic effects. Only in cases in which those toxic effects are exploited as a weapon system are they regarded as chemical weapons. Military use of any toxic chemical used for peaceful purposes must be regarded as a chemical weapon (1, 9). The verification schedules in the CWC contain, for special reasons, two toxins: saxitoxin and ricin.

## 3.1.4. History

The use of chemicals, in forms of evil-smelling smoke or poisonous substances, to disable an enemy dates back to antiquity. It was not until the growth of the chemical industry during the second half of the nineteenth century, however, that the technology was developed to produce chemical warfare agents on a large scale and the liquefaction of gases became feasible.

Toxic compounds and pathogenic microorganisms are natural health hazards. They form a threat that is insidious, damaging, or deadly (9). Throughout history, codes of military

conduct have forbidden the use of poisons and the deliberate spread of disease as a method of warfare. The last century saw, on the one hand, the massive use of chemical weapons, but also efforts to abandon them in the form of the Geneva Protocol of 1925 (11), the BTWC of 1972 (2), and the CWC in 1993 (1). During the First World War, chemical warfare began in its early stages. Only one month into the war, experiments with incapacitating agents commenced. Although not well documented, the first attacks with chlorine gas took place at the eastern front in January 1915. There are, however, few records of these attacks and the damage that they inflicted. Generally, the massive attack with chlorine gas near Ypres, Belgium, on 22 April 1915, was claimed as the beginning of modern chemical warfare. The German army, under the guidance of the well-known chemist, Haber, released 150 tons of chlorine gas from 6000 cylinders along a front line of 6 km. The results upon the unsuspecting and unprepared Allied troops were devastating. According to French reports: five to six thousand fatalities were recorded and an additional fifteen thousand men were poisoned to a lesser degree. In this total surprise attack, with no protection whatsoever or standard operating procedures to mitigate the effects (climb a tree, or breath through cloth watered by urine), nearly 100 kg was required to produce ten casualties.



Famous picture of a gas attack during World War 1, somewhere along the western front.

The employment of chemical warfare agents rapidly became a "normal" part of front-line life for both sides. In the years to come several additional chemicals that acted through the respiratory system and were more deadly than chlorine, were used. The most hazardous of these was phosgene. HCN is often mentioned as a very deadly poison, but it appeared not to have been very effective because high concentrations in short periods of time were required to generate the apparent effects. When soldiers were exposed to low concentrations of HCN over a long period of time, no casualties resulted. Air filters, masks, and respirators were quickly developed to counteract the effects of the poison. The first filters developed were based on the chemical destruction of the poison and could, therefore, easily be circumvented. When active carbon was used as a general filter media, almost any compound could be stopped. In turn, this induced the development of agents capable of circumventing the filter. The most successful development was the compound best known as mustard agent. As well as being an inhalation hazard, mustard agent also generated percutaneous effects. Protections against this type of compound were developed after WWI. Initially, these protections were based on chemistry that was easy to circumvent or on air-impermeable clothing for specialized troops. These suits were cumbersome to wear and offered little protection. This protective equipment existed until the Second World War, which saw the development of activated carbon-based filter layers. Today, most technologically advanced nations have their forces protected by this type of clothing. Together with a proper mask, these suits form a deterrent to the use CWA against troops because once protected, the effects of the poison are minimal.

Vulnerabilities remained for countries not in a position to invest in an elaborate protection of troops or the civilian population. Examples are mainly in Africa and Asia. The deliberate use of CW against totally unprotected populations is regarded as an act of barbarism.

The influence protection can have become clear during WWI. The first troops equipped with a mask were the British, by the end of 1915. As a result, the number of chemical casualties among British troops in 1916 was relatively small, the main reason being that German forces regarded chemical attacks on the British forces as useless. The statistic that 250 kg of explosives had to be targeted on the enemy to create one casualty (with 1 in 3 killed), and only 100 kg of asphyxiating gas was required to cause one chemical casualty (with 1 in 10 killed) demonstrated the effectiveness of chemical weapons. However, as soon as a respiratory protection factor of ten was provided, ten times the quantity of chemicals had to be used. Limited protection clearly was a serious disadvantage for the use of chemicals, causing explosive munitions to once again be favored.

At the end of the First World War, 110,000–125,000 tons of chemical warfare agents had been used, of which 90% were choking agents and the remaining 10% was mustard gas. Chemical warfare agents were responsible for 1.3 million casualties. The percentage of deaths due to chemical weapons was small in relation to that inflicted by other weapons, i.e., 7% (91,000 deaths) and 30%, respectively. Although the total number of casualties was high, the employment of chemical warfare agents was of minor efficacy: on average, one ton of chemical warfare agents put ten men out of action (note, the weight of the containers used in the dispersal of the gas was not included in the calculations). This low efficacy was partly due to protective measures, which, although primitive, were rapidly developed. It should be noted that the less than 10,000 tons of mustard agent used in the last year of the war was responsible for several hundred thousand casualties.

Only 30 kg of mustard gas was required to cause ten casualties, of which less than 2% were lethal. This was sometimes used to support the argument that chemical warfare was more humane than warfare in which metal and explosives were employed. The fact that CW casualties suffered for many years after the war ended was not considered when this conclusion was drawn. Chemical warfare casualties from the Iran–Iraq war in the 1980s have suffered for more than 20 years and possibly many more to come.

These WWI statistics are grand total averages. Later analysis by Haber's son (12) showed that the effectiveness of some attacks was much lower, and in some cases it was much higher. The total amount of agent used was around 125,000 tons, of which about 8000 tons was mustard agent. The total number of chemical casualties was estimated to be 1,250,000, of which 250,000 were due to mustard, mainly used by Germany. Assuming that 117,000 tons of non-mustard agents produced 1 million casualties, it appears that on the average just under 100 kg was required to cause one casualty. Because the toxicity of the agents with respect to producing casualties is, on average, 100 mg/man, one million times an effective dosage was required in WWI to have this effect. For mustard the effective casualty-causing dosage is a few milligrams (based on whole body exposure), requiring, once again, about one million

times the toxic dosage for casualties, using the 8,000 ton figure cited previously. (The calculations regarding mustard agents are somewhat uncertain because at the end of the war, starting in the second half of 1917, the quality of the German chemical munitions became unreliable and more than half of all chemical munitions did not detonate. In addition, there are no good records of the quantities of chemical ammunitions destroyed or dropped in the sea directly after the war.)



One of the first gas masks. It does not look too comfortable, but it worked well in preventing casualties.

Despite the low efficacy of the chemical weapons used during the First World War, research on CW agents continued after 1918. For example, the blistering agents Lewisite and nitrogen mustard gas were developed. The use of chemical weapons continued virtually wherever warfare was in progress during the inter-war period. Use by the Spanish and French in their North African colonies, by the Italians in Abyssinia (nowadays Ethiopia) in 1936–1937, and by the Japanese in China during the 1930s are well known events.

The first representative of a class of much more toxic chemical warfare agents, the nerve agents, was invented in 1936. In the course of his research on new insecticides, Schraeder prepared Tabun in the laboratories of IG-Farben, Germany, and several analogs were discovered a short time thereafter. In 1938, the Ministry of Defense of Nazi Germany decided to build a factory to produce nerve agents. The first lot of Tabun was manufactured in 1943, and small quantities of Sarin in 1944. A third nerve agent, Soman, was in the laboratory testing stages at the end of the WWII (18a).

Chemical warfare agents were not used during the Second World War, even though only the Germans possessed nerve agents. It is still a mystery why the Germans did not resort to the use of chemical weapons, particularly during the later stages of the war. In all likelihood, the main reason could have been fear of reprisal. In the beginning of the war, none of the belligerents had sufficient stocks to conduct chemical warfare, but each was convinced that

the others were fully prepared. Later in the war, Germany came into the possession of Tabun (1943), but it was dangerous to transport it to the front zone. Additionally, at that time they were convinced that the Allied troops also had supplies of nerve agents. Chemical warfare would have been less efficient because the troops were equipped with masks and later with forms of skin protection, although over the course of the war and in the absence of the use of chemical warfare, the haversacks for carrying a mask were often used for carrying liquor and other contraband. Amazingly, the Allied counterintelligence regarded the information that Germany was in the possession of nerve agents as war propaganda.

Still more toxic nerve agents, the V agents, were developed by researchers in the UK in the 1950s. Also in this period, military research institutes became interested in nonlethal incapacitating chemical warfare agents.

The end of the war in 1945 and beyond saw no prohibition of chemical warfare. During the Yemen War (1963–1967), mustard gas was used, and herbicides (defoliants) and tear gases were deployed on a large scale in the Vietnam War (1961–1970). Highly efficient chemical warfare protection devices of Soviet origin were captured by Israel during the Yom Kippur War in 1973, which resulted in an increased emphasis on chemical protection within NATO. By the end of 1970, the US accused communist troops of using unspecified chemical warfare agents, called "yellow rain", in Laos, Kampuchea, and Afghanistan. The agents used were denoted as trichothecenes, a group of mycotoxins. A team of experts that investigated the alleged use for the United Nations could neither state that the allegations had been proven nor could they disregard the circumstantial evidence presented of possible use. Later, some scientists argued that the positive analytical identification of the trichothecenes was an artifact, and that the yellow rain was most likely due to bees dropping their feces in a rain storm. In 2004, another "attack" with yellow rain took place in India 60 km north of New Delhi. This time scientists confirmed that the "attack" was indeed due to bee droppings.



Colombian army confronted with chlorine attacks from FARC.

The recent past has seen several incidents of chemical warfare usage. Definite evidence was presented of the employment by Iraq of both nerve agents and mustard gas during the Iran–Iraq War (1980–1988). Furthermore, Iraq attacked the Kurds living in Iraq. Recently, Iraq

explicitly threatened Coalition troops with the use of chemical weapons during the Gulf Conflict (1991). South America was, until recently, a continent free of CWs. However, there are reports that the FARC used CWs against the Colombian military. Finally, CWs have become available to an increasing number of countries during the last decade. Fortunately, many of these countries have acceded to the CWC and are in the process of destroying their stocks.

#### *3.1.5. Threat reduction through arms control*

Arms control is covered by the CWC for chemical weapons and by the BTWC for biological weapons and toxins. It is generally understood that a treaty to ban a certain class of weapons is of little value if strict verification of the rules in the treaty is not possible or if they are not combined with sanctions or determined responses. Adherence to a treaty is enhanced if there are benefits for the State Parties and limitations for non-State Parties. Finally, national laws should criminalize research, development, or manufacture of any such weapons. The CWC has implemented most of these measures in its statutes.

Limiting proliferation of chemical weapons is another objective of the CWC. Most chemicals that could be used in the development of chemical weapons are subject to verification inspections. More importantly is that trade and export of these so-called scheduled compounds to non-State Parties is prohibited. Verification inspections check the correct handling of these scheduled compounds. Export controls make it more difficult and costly for non-State Parties to develop massive amounts of chemical weapons. Limiting proliferation could be less effective if the CWs are based on non-scheduled compounds that are not subject to verification inspections. The General Purpose Criterion however, would allow other forms of inspections, e.g., a challenge inspection.

Biological weapons have been totally prohibited by the BTWC (2), which was opened for signature in 1972 and entered into force in 1975. The BTWC banned, for the first time, a complete class of weapons but the treaty has no verification regime or benefits for State Parties. Therefore, the response to the threat of biological weapons has been somewhat different. Considering the threat posed by biological and toxin weapons thus far, that there are a number of measures that together are mutually reinforcing and form a web of deterrence.

With recent advances in biotechnology, it is tempting for certain States to develop biological warfare capabilities, particularly because it is possible to easily break-out of the convention and build a biological weapon in a matter of weeks, rather than several years. Therefore, it is important to maintain passive defense measures because these measures will considerably increase the required amount of warfare agent, as illustrated by the following example. A contamination of approximately 10 g pure Anthrax, containing  $10^{13}$  spores per gram is required per km<sup>2</sup> to produce significant casualties to make its use worthwhile, if the biological warfare agent is disseminated with high efficiency. Such a low level can only be achieved under ideal conditions (according to the former Soviet Union doctrine, 5,000 g/km<sup>2</sup> would be disseminated (13)). The level must be increased to one ton of Anthrax for every km<sup>2</sup> to achieve the same effect if the troops are warned and equipped with masks that provide a protection factor of  $10^5$ . (A factor of  $10^5$  is required for most military masks for protection against vapor, and the protection factor against 0.3 µm aerosol particles is around  $10^4$ ). Protection factors for larger 1–5 µm aerosol particles are usually 1–2 orders of magnitude higher.

Preventing proliferation, especially of the knowledge for producing biological warfare agents, is difficult. Students from States that might be interested in developing a BW capability may study at Western Universities. In addition to the required technologies, the means for production of CWs and BWs are proliferating over the world. Worldwide illicit drug trafficking and production form a good example that demonstrates how difficult it is to block the transport of illegal goods and the means to produce them. Shipments often pass through many countries before they arrive at countries of concern. An additional difficulty, at least with respect to BW proliferation, is that the research and means nearly always are dual use. Research into pharmaceuticals and cancer therapies are some of the many examples. The technical means for preparing pharmaceuticals and some BW agents are very much the same. Selectively blocking materials intended for BW development is not feasible. Stopping all research in this area, as suggested by Mirzayanov (CBRNe World summer 2009 issue), is not feasible either.

Occasionally, it is remarked that: "The CWC (and also the BTWC) may have had more of an effect on changing the character of proliferant activity than in stopping it."

Although not every State fully believes in non-proliferation and, therefore, threat reduction offered by the CWC, it is obvious that the CWC is a major contributor to these causes. At the end of the previous century, there were 80,000–100,000 tons of chemical warfare agents in the world. In the coming ten years, Member States will have destroyed their stocks, and a much smaller amount here and there may remain in States that do not adhere to the CWC. The total mass of CWAs will be reduced by a factor 100 if not 1000. If chemical warfare should occur, it would be on a smaller scale and less frequent, but unfortunately also less predictable.

#### 3.1.6. Dissemination of CB agents

The most common target for chemical and biological agents is the respiratory tract. Agents must be dispersed as vapors or aerosols that are absorbed by the lungs. This is particularly difficult to achieve for biological agents, because the size range of interest is around 5  $\mu$ m. Larger particles are stopped by the upper respiratory tract and very small particles are not absorbed well by the lungs; they are carried away in exhaled air. Advanced technologies are required to produce 1–10  $\mu$ m particles efficiently, and even then the efficiency will not exceed 25%. Some chemical agents also act through the skin, either in the form of vapor or as small droplets. The absorption of the liquid agents by a fine dust powder has been proposed as a means for deploying an agent. Essential to these threats are the capabilities of an opponent to deliver warfare agents to a target. Means of delivery for Weapons of Mass Destruction (WMD) include, for instance:

Ballistic Missiles Offensive Aircraft Unmanned Aerial Vehicles (UAVs), Cruise Missiles

Non-State actors may use any form of dissemination, from release of dust or aerosol spray cans to evaporating liquid depositions. UAVs have been considered by the Japanese Aum Shinrikyo sect for dispersal of biological agents.



Aircraft vortices, droplet size, etc. render the aircraft spray technique an unpredictable method for disseminating CWs.

## **3.2.** Threat agents

#### 3.2.1. Scope of threat agents

Chemical and biological warfare agents of interest form a spectrum from

- Toxic industrial materials (TIM's);
- Classical chemical warfare agents, including emerging chemical agents;
- Mid-spectrum agents, i.e., toxins and bioregulators, either from natural origin or synthesized;
- Self-multiplying organisms, possibly genetically modified.

The criteria for assessing the hazard of certain types of agent have often been defined from an offensive point of view. According to pre-WWII Soviet doctrine, the military value of a toxic or pathogenic agent is determined by:

- Its toxicity (inhalation or dermal), stability, and physical state;
- The capacity to produce the agent from indigenous raw materials;
- The simplicity and economy of its production; and
- The ability of the agent or its precursors to be used in peacetime. (14)

This type of assessment is subjective because all aspects involved must be assessed and rated on a scale. The scale, however, may be very different in different parts of the world.

#### 3.2.2. Toxic industrial materials (TIM's) or chemicals (TICs)

Toxic industrial materials (TIM) include toxic industrial chemicals (TICs). A TIC is defined as an industrial chemical that has an LCt<sub>50</sub> value of less than 100,000 mg $\cdot$ min/m<sup>3</sup> in any

mammalian species and is produced in quantities exceeding 30 tons per year at one production facility. TICs that pose an acute inhalation hazard are of greatest concern. TICs, such as chlorine, phosgene, hydrogen cyanide, or cyanogen chloride have been used as chemical warfare agents during WWI. (These agents are still classified as TICs rather than chemical warfare agents.) The most toxic of these compounds, phosgene, is at least 100 times less toxic than the nerve agent Sarin, requiring large quantities to be released before an effect is achieved. (The trench warfare of WWI required 100 kg for one casualty!) HCN and ClCN are only hazardous at high concentrations delivered within a short period of time. Detoxification of these agents in the human body proceeds very quickly. Other examples of TIMs include ammonia, acids, solvents, pesticides, herbicides, fertilizers, fuels, petrochemicals, and intermediates used in the manufacture of plastics. They are legitimate articles of commerce that are traded in very large volumes and are not subject to the same regulations or export controls as chemical warfare agents. TICs are still attractive as improvised chemical weapon fills and have potential for inclusion in clandestine weapons programs or contingency plans. These chemicals are stored in large quantities and are transported daily. Therefore, the deliberate or inadvertent (collateral damage, industrial accident, fire, or environmental disaster) release of TICs cannot be excluded. In the case of terrorist incidents, the maximum amount of TICs involved would likely be a truckload or a railroad car. Local storage sites could be attacked as well. The simplest form of protection is to reduce the quantity of locally stored chemicals as much as possible and to divide it over several separated storage tanks. TICs are particularly attractive agents because of their ready availability, but their efficiency is limited against troops that employ current measures of protection. However, TICs might be a serious threat for populations living in the vicinity of large storage sites. These people should be trained and provided protection inside their homes (see Chapters 5 and 6).

A group of experts in Canada, the UK, and the US (15) evaluated the inhalation hazards of TICs. An initial screening identified 1164 chemicals that met the toxicity criteria. This list of chemicals was reduced by including only those that were gases, liquids, or solids with an appreciable vapor pressure at 20°C, or those that were listed in the US Department of Transportation Emergency Response Guide. Available production data were used to reduce this list to 156 chemicals. Actual production figures were difficult to obtain. The Chemical Manufacturer Association provided a list of chemicals produced in excess of 30 tons per year in at least one production site. This information reduced the list to 98 chemicals.

A hazard index was then developed. This index considered the distribution of producers in the world as well as the number of continents in which the chemicals were produced. Concentration values were reported in units corresponding to the Immediately Dangerous to Life and Health (IDLH) as a parameter for toxicity. Vapor pressure was used as a parameter as well. Higher vapor pressures produced higher inhalation hazards. Each parameter was rated on a scale from 1 to 5.

The Hazard Index (HI) is the product of the four parameters and was used to identify and rank TICs.

 $HI = f \{(toxicity) \ x \ (physical \ state) \ x \ (geographical \ distribution) \ x \ (number \ of \ producers)\}, with a maximum value of 625$ 

The TICs were ranked in three categories as an indicator of their relative importance and to assist in hazard assessment:

- <u>High Hazard</u>, HI>81; widely produced, stored or transported TIC, very toxic and easily vaporized; 21 chemicals meet this criterion;
- <u>Medium</u> Hazard, 36<HI<80; TIC that may rank highly in some categories but lower in others; 46 chemicals meet this criterion;
- <u>Low Hazard</u>, HI<36; TIC might form a hazard in exceptional cases; 36 chemicals fall into this category.

Annex 3A includes tables that list the agents in these three categories. Other scorings lists have been produced by the Fraunhofer institute in Germany (Poster session 10<sup>th</sup> International Symposium on Protection against CBW Stockholm, June 2010)

"Heavy gas" models for either heavy or cold gases best describe the release of TICs. These models depict the maximum possible challenge, which are the optimal conditions for studying hazard distance and protection. However, these models do not predict the hazard area or account for complex terrain or dispersion in urban areas.

The experts modeled hypothetical releases and determined safe distances from storage sites for military operations. These methods were similar to those used by emergency services to assess a hazard area, for example, during a chemical HAZMAT incident.

## 3.2.3. Chemical warfare agents

Agents of interest to the former and present possessors of chemical weapons are well-known. Nearly all agents also appear in the schedules of the CWC, with a few exceptions, e.g., phosgene oxime. This compound is not very stable, and all attempts to stabilize the compound have been unsuccessful. Over the past years, several States have investigated organofluoro compounds as potential mask breakers. The general conclusion is that compounds that would penetrate carbon filters in appreciable quantities show limited toxicity and stability. As a result, the concentrations that must be created in the field must be appreciable before hazardous quantities will penetrate the filter. (This conclusion is similar to the one drawn in 1946: it was observed that fluorine compounds must be reactive if they are to display any toxicity. Reactivity produces reactions with the active groups on carbon filters or with the ambient moisture in the air. A "regular" gas mask filter will, therefore, be effective. (US review on chemical warfare activities during WWII, Washington, 1946)).

Lewisite is often cited as a CW agent, but low persistency led to its abandonment and destruction by the US in 1954. Lewisite hydrolyzes quickly in the environment. Considerable amounts of Adamsite are still stockpiled and are, therefore, regarded as a potential threat. Sulfur mustard and the more difficult to prepare nitrogen mustards are still warfare agents of concern. In addition to the mustard agents, the nerve agents of the G- and V-type are of great interest. Threats from G agents are not restricted to Tabun, Sarin, or Soman. All classical nerve agents defined in CWC's Schedule 1 lists should be declared under the CWC. Several States are said to consider psycho chemicals, such as glycolates (e.g., BZ) as a possible threat. Based on these guidelines, Table 3.1 summarizes all chemical warfare agents of potential interest.

Choking and blood agents must be released in large quantities to have a widespread effect. Incapacitants, in general, do not serve the purposes of terrorists. New nerve agents and the V-agents are difficult to synthesize. Therefore, G-agents and blister agents, particularly mustard, are the most likely CW threat agents.

| Type of Agent  | Chemical   | -             | -        | Number                   | of      | Common |
|----------------|------------|---------------|----------|--------------------------|---------|--------|
| choking/blood  | phosgene   | cyanides      |          | Compounds                | oro 00m | aounda |
| blister agenta | S mustard  | N mustard     | Louisito | 4-7 + perhuoro compounds |         |        |
| blister agents | S-mustaru  | IN-IIIUStal u | Lewisne  | 4 common, 8 rare         |         |        |
| G agents       | Sarin(GB), | Soman         | Tabun    | 9 different Nerve agents |         |        |
|                | GF         |               |          |                          |         |        |
| V agents       | VX         | Vx (R-33)     |          | 5 V agents               |         |        |
| nerve agents   | new types  |               |          | e.g., 3 GV ag            | gents   |        |
| other agents   | Adamsite   | BZ            |          | 6 types of incapacitants |         |        |

Table 3.1 Chemical warfare agents of interest according to Reference 3–10.

#### 3.2.4. Mid-spectrum agents

The first type of mid-spectrum agents is toxins that are produced by organisms and, therefore, belong to the class of agents of biological origin. The second type, bioregulators, are peptides present in the human system in extremely small quantities that are essential for regulating the system but are deadly when present in large amounts. With recent advances in synthetic organic chemistry, it has become possible to synthesize some of the toxins and bioregulators. It is expected that in the coming years many more if not all toxins will be synthesized. In that case, they will most likely be regarded as chemical warfare agents according to the definitions of the CWC. Some may be no more toxic than nerve agents. Others are up to four orders of magnitude more toxic and offer the option of clandestine delivery. In addition, the advances made in organic chemistry and biotechnology facilitates the production of (extravagant) toxins and bioregulators. Mid-spectrum agents that were formerly of minor importance but may easily be produced by gene technology (e.g., snake and spider toxins) may be of greater concern in the future.

The CWC schedules mention only two toxins: ricin and saxitoxin. On the internet, information about 4 such toxins may be found: Aflatoxin, Botulinum toxin, mycotoxins, and ricin. The Center for Disease Control and Prevention in the US (CDC) maintains a list of restricted agents, in which 12 toxins are listed.

It should be noted that all mid-spectrum agents are either solids or liquid suspensions. Therefore, it is highly likely that the atmospheric dispersal of these agents in a respirable particle size will not be very efficient (e.g., one order of magnitude less efficient than the dispersal of GBs). In addition, a respirator usually offers better protection against particulate matter than against vapors by up to one order of magnitude. Mid-spectrum agents, therefore, must have a toxicity that is 10 times that of GBs (100 times if a mask is available) in order to be of the same effectiveness. On the basis of these criteria, the most threatening mid-spectrum agents appear to be:

- Botulinum toxin,
- Tetanus toxin,
- Diphtheria toxin,
- Staphyllo entero toxin B (SEB),
- Staphyllo alpha toxin,
- Clostridium perfrigens epsilon toxin,
- Shigella toxin.

The use, however, of more potent toxins in large quantities (several tons) to break the protection afforded by a mask is assessed as not very likely.

Trichothecenes and ricin are often mentioned as threat agents, not because they are extremely toxic, but because trichotecene can be used for food poisoning and ricin is easy to produce. The toxicity of these compounds is, however, such that resort to nerve agents would be a better option. One type of trichotecene mycotoxin is said to act on the skin. There is, however, no proof that this is really the case. On the contrary, several observations indicate that they do not act on the skin. (See paragraph 3.1.2.h.) The aspects of opportunity or local availability may play an important role and overrule the logical argument of effectiveness.

## 3.2.5. Biological warfare agents

This class of agents comprises those agents that have the ability to self-replicate, either independently or in conjunction with a host organism. Their toxic action is dependent either upon the release of an extracellular toxin, the nature of which may be identical to those considered under the heading of mid-spectrum agents, or upon the invasive destruction of host tissues. The amplification factor available through self-replication and secondary infection introduces an enhanced hazard, which can in some instances represent an advantage, and in others a disadvantage as the amplification cannot be controlled. Conclusions regarding the significance of these two aspects in any particular circumstance will be subjective, as will be an assessment of the effects of several days' incubation period.

The previously identified five desirable characteristics of biological warfare agents are:

Simple to produce; Stable; Infective; Capable of acute incapacitation; In some circumstances, not easily defined, transmissible by secondary infection.

The CDC uses similar criteria to identify the most likely biological agents for use against the general public:

The public health impact in terms of morbidity, mortality, and propensity for contagion;

Delivery potential based on the ease of mass production and distribution, as well as stability of the agent;

Special preparation requirements based on whether vaccines or medications should be stockpiled, agent detection increased, or tools to identify the agent produced; Public perception of the agent and its capacity to generate fear.

These criteria still apply and can be used to select the most likely biological threat agents.

Several techniques may be used to disseminate biological warfare agents either directly into the human body through the respiratory tract, through the digestive tract (food, drinking water), by percutaneous transmission, or through transmission by other species (animals). Only dissemination through the respiratory tract is discussed as being the most important from a health hazard point of view, although there are many occasions in which civilian water supplies have been contaminated with pathogenic microorganisms. (It must be considered that at some point in time terrorists will attempt to use this route; fortunately, most modern water treatment plants have measures installed to counteract bacteriological contamination, e.g., disinfecting based on UV irradiation to counteract cryptosporidium contamination of the drinking water.)

In all, several features of biological agents influence their potential to be used as biological warfare agents, including infectivity, virulence, toxicity, pathogenicity, incubation period, transmissibility, lethality, and stability. (These terms are defined in ANNEX 3B.)

Based on the assumptions and estimations discussed earlier as well as the available literature, the most likely biological warfare agents are:

Eastern equine encephalitis virus Variola major virus Bacillus anthracis Brucella types Burkholderia mallei and pseudomallei Francisella tularensis Yersina pestis Coxiella burnetii

Some States and non-State actors have considered viruses such as Ebola and Marburg. Because their properties do not comply with all of the criteria listed above, they are not considered as likely threats. Advances in biotechnology may change this consideration in the not too distant future.

If one considers all possible genetic modifications, which are increasingly facilitated by advances in biotechnology, there seems to be an infinite list of possible biological warfare agents. "It is worth highlighting the ease of development of Biological and Toxin Weapons and the fact that humans appear to be increasingly susceptible to new diseases" (9). Nearly endless variations in properties can be obtained by genetic engineering.

"However, the [military] threat can effectively be reduced to those organisms which are the most robust, potent, quick acting, easy to make, store and deliver" (9). This statement is expected to be true when dealing with a rational opponent. Terrorists do not belong to this category of rational opponents, and the criteria related to availability may be the most relevant. In addition, the civilian population is not generally composed of healthy young males that typically form the military population. There will be a much wider distribution in sensitivity, particularly among the young and elderly who may be very sensitive.

It should be understood that biological warfare agents are living materials, one strain of which may be much more effective than another. Not only can the multiplication rate differ, but the potency of the toxin that is produced, its survivability in the atmosphere, etc., may vary significantly. Before introducing anthrax as a weapon in the previous century, the US scanned many strains until they found a very active one. Ken Alibek (13) was rewarded a prize in the former Soviet Union for identifying an even more infective strain of anthrax. Fortunately, most of that weaponized anthrax, both in the US and in Russia, has been destroyed and is awaiting destruction. Because of the long list of biological agents and the variability with respect to type, hazard assessment often follows a parametric approach in which three aspects of the threat are treated as parameters. The most hazardous agents are those that score highly in all three classes. These parametric aspects are:

Virulence and infectivity, in three classes: low, medium, or high. One particle of the most infectious compound leads to disablement.

Stability during dissemination, in three classes: low, medium, or high. This aspect describes the number of hazardous elements that may be introduced into the air.

Stability in the environment, in three classes: low, medium, or high. Biological agents can degrade during storage or during (explosive) dissemination. If the agent deteriorates quickly in the environment, the agent cloud will not travel very far downwind.

After dissemination as a spray, a suspension containing a biological warfare agent must evaporate to such a degree that floating particles are realized in the optimum size range of 1-5µm. Biological warfare agents can be disseminated by using explosive or spraying devices. It is very difficult to achieve effectiveness using the spraying approach, for example, with an aircraft. To be effective, the drops must not reach the ground before they evaporate to the required size. A less difficult method for disseminating is to spray using an aerosol generator. Again, producing a large fraction of particles within the required size range demands advanced technology. The easiest way to deliver an agent, using explosive devices such as bombs, is also the least effective method.

#### 3.2.6. CB terrorism

Non-State actors most likely will resort to the agent categories considered above, although less 'optimal' toxicity and other characteristics, such as the physical properties and purity, may be important considerations. The upper limits on the quantity of CB agent that a terrorist may use effectively include: 1. TICs in ton quantities in storage or during transport; 2. classical chemical warfare agents in kilogram quantities with the most hazardous release method being a vaporized form; 3. mid-spectrum agents in gram quantities; and 4. pathogens in milligram quantities, where both 3 and 4 are released as aerosols for maximum effect. The more toxic or more virulent the agent, the more hazardous it will be to produce it in significant quantities. In any case, during production, non-State actors must protect themselves physically or through prophylaxes. Consequently, CB defenses against such attacks are expected to be diverse. Because the issue of CB terrorism is relatively new, this field is still largely unknown. A clear defense concept, scenarios for planning purposes and qualitative and quantitative assessments are currently lacking or are of questionable validity (see the website for the US Homeland Defense). Often, the casualty-producing potential of an agent is derived from the ratio of grams of agent available to the lethal dosage by, e.g., injection. In comparison with the WWI statistics, this ratio overestimates the casualty potential by a factor of one million.

## 3.3 Quantitative hazard analysis

#### *3.3.1. Computer modeling*

Computer modeling of chemical and biological attacks will be discussed here because it is of paramount importance to estimate the dosages and concentrations of warfare agents that could be encountered during an attack, as well as estimate of the number of casualties that may accrue. This, in turn, provides valuable information to the military, first responders, and equipment developers regarding the scope of the threat for which preparations must be made. Particularly problematic for the modeling efforts is the incorporation of complex terrain and urban areas. Realistic modeling of an attack requires accurate source parameters (i.e., dissemination efficiency, influence of meteorological parameters on decay or loss of activity,

re-suspension by wind or movement) and the influence of terrain on the reduction of particle concentrations. Today, modeling efforts for CB cloud dispersal that account for the source parameters mentioned above are insufficient due to a lack of experimental calibration data.

Most models use a Gaussian approach to describe the dispersion of warfare agents in the atmosphere. Important input parameters are the source parameters, the meteorological conditions, the dispersion parameters, and the meandering path of the cloud. As far as is currently possible, the models have been validated using field experiments. The models are deterministic in nature, and the results represent the average of a large number of individual trials. The average describes the highest possible challenges in the field and, therefore, presents a worst-case scenario that assists the development of protective equipment. It should be noted that each individual release may deviate strongly from the average plume calculated using the model. The width of the plume depends on the actual dispersion parameters of the day, the effects of meandering are averaged, and the concentration fluctuations are not taken into account.

#### 3.3.2 Results of computer modeling.

The least disputable model calculations begin with a line source of liquid agent deposited, e.g., by an aircraft. At some distance downwind, the source can be regarded as a line of, for example, 1 kg agent/m. The agent will evaporate and will be transported downwind where a vapor dosage would be experienced, expressed in terms of concentration x time,  $mg \cdot min/m^3$ . As an example, assume that at a wind speed of 1 m/s, it will take 100 seconds for the agent to evaporate and form a cloud 100 m in depth. Due to turbulence, the agent vapor will not remain at ground level but will be distributed vertically. For simplicity, it will also be assumed that at the target site the agent is evenly distributed over a height of 3 m. Therefore, the original one kg of agent is initially assumed to be evaporated into a volume of  $300 \text{ m}^3$ . During the passage of the cloud, a man standing downwind will inhale about 30 liters of air in 100 seconds. Therefore, the man will inhale only 100 mg of agent. To achieve these ideal conditions, the weather must be very stable, the wind speed constant and low, and the loss of agent at the deposition site or during transport must be negligible. Somewhat further downwind, the agent will have been distributed over a thicker layer of air and the dosage will consequently be lower. The simplified calculations show that at least 10,000 times the effective dosage must be released to produce a casualty. Without going into detail, one can imagine that realistic military situations would require, on average, one million times the effective dosage to be released to cause one casualty. WWI demonstrated that attacking one another with large quantities of chemicals is feasible for determined armies.

As the simplified calculations indicate release in an enclosed environment requires far less than one million times the effective dosage per casualty. In the Tokyo Metro incident, several hundred grams to 1 kg of agent were released. The medical records indicate that around 1000 persons were exposed to Sarin with 13 fatalities. (The 4000 other casualties did not result from Sarin exposure). A lethal dosage for Sarin is on the order of 1 mg; a dosage that yields nonlethal effects is 0.01–0.1 mg (calculated from the dose-effect relationship and the breathing rate, see Chapter 4.) Therefore, the ratio between the released quantity and the effect amount times the number of casualties ranges between 10,000 and 100,000. Roughly, agent release in an enclosed space produces a dosage that is 10–100 times higher than that produced in the open air. Computer simulations confirm this ratio. A study by SIPRI (10) mimicked (Sverdlovsk) the release of anthrax spores in a shopping mall. Again, 10,000 times the effective dosage was shown to be required to produce a single casualty. Even though the
effective dosage applied in this study was too low (8000 instead of 55,000) and a steep doseeffect relationship was chosen, the indoor release was again shown to be 10–100 times more effective than release in an open space.

An intermediate situation between outdoor and indoor release occurred during the Bhopal incident. Most persons in the affected area were asleep and were exposed in their bedrooms. The 40 tons of methyl isocyanate that were released caused 3800 lethal and 200,000 nonlethal casualties. Roughly 10 kg induced lethality and 0.2 kg produced a casualty. In view of the toxicity of the compound, the quantity released was 10–100 times more effective than if it had been released in an open space. The relatively low ceiling of the inverted layer kept the concentration high at ground level.

It is appropriate here to point out a warning regarding the use of absolute numbers. Important to any CB incident is the surprise factor. When the surprise is complete, the attacks are likely to be more effective. In other words, the number of effective dosages required to cause a single casualty is reduced. On the other hand, when circumstances are less favorable for the attacker, the required number of dosages may increase considerably. The point here is that defensive measures must be in place to cope with 95%, 99%, or 99.9% of the challenges presented by the worst-case attacks. It should be stated that the release of classical TICs and classical chemical warfare agents as vapors is much more effective than the release of mid-spectrum and biological agents aerosols. The effectiveness differs by at least one order of magnitude and, in most cases, two orders of magnitude. In addition, the protection offered by a mask and inside rooms sealed with duct tape is much better against aerosols than against vapors. Overall, solids must be at least 100 times more toxic than classical chemical warfare agents as vapors.

# 3.3.3. Hazard area prediction

Directly linked to the computer modeling of attacks is the prediction of the hazard area. Currently, crude models use a triangle to indicate the hazard area imposed by a contamination point for wind speeds exceeding 2.5 m/s. Circles are used to indicate hazard areas at lower wind speeds. The data processing speeds of present day computers permit increased accuracy in the prediction of hazard areas. A given location may now be associated with a probability for contamination by a downwind cloud. Most models use a Gaussian approach to describe the dispersion of warfare agents in the atmosphere, leading to an average usually cigar-shaped hazard area. It should be noted that individual release conditions may produce strong deviations from the average plume calculated using the model. Therefore, the cigar-shaped hazard areas predicted by the Gaussian plume models are often misleading. In addition, the models do not consider complex terrain or built-up areas. In the latter case, clouds typically follow streets along the direction of the wind flow.

# **3.4. Future threats**

# 3.4.1. Classical chemical warfare agents

The classical chemical agents that are perceived as practical threats are limited: sulfur mustard and nerve agents of either the G or V type. It might be expected that States or non-State actors that wish to circumvent the CWC would look for newer nerve agents. A US 2001 proliferation and threat document (16) described the so-called "Novichoks": "[...] in 1992, Russian scientists familiar with Moscow's chemical warfare development program publicized

information about a new generation of agents, sometimes referred to as Novichoks. These scientists reported that these compounds, some of which were binaries, were designed to circumvent the CWC and to defeat Western detection and protection measures." This is, at least in part, far from correct. Nerve agents inhibit the cholinesterase enzyme, and detectors based on those enzymes will successfully recognize new agents as threats. Protection is based on physical processes that are non-selective and, therefore, protect to a certain extent against every agent. This is not to say that the protection cannot be overwhelmed by large concentrations, it just points out that the protection will work and larger quantities must be disseminated to improve effectiveness. Furthermore, it was claimed that the production of this class of agent could be hidden within "commercial plants". These agents are discussed in further detail in references 17, 18, and 18a. Recently, the Russian scientist Vil Mirzajanov published a book describing some of the compounds (18b).

#### 3.4.2. Mid-spectrum agents

Natural poisons often consist of chains of several proteins. Studies have shown that only a portion of the long chain is responsible for the toxic effect. It can be expected that the synthesis of the shorter chains will be possible in the near future. Similarly, the synthesis of complex bioregulators is likely. There is very little actual interest in producing or developing only the proteomic chain of a bioregulator, which is the portion responsible for its toxic effect, because the stability of the synthetic agent will decrease. To weaponize a bioregulator, it may be more worthwhile to develop more stable analogs than to develop more toxic analogs.

Production of toxins in genetically modified common microorganisms is possible. However, selection of microorganisms that are sufficiently robust to maintain the included alien gene is still a technological accomplishment not available to most States and Non-State actors.

#### 3.4.3. Biological agents

Biotechnology incorporates many different techniques. It usually comprises the use of cells or parts of cells in such a way to produce a desired product. The product can vary from specific toxins or pharmaceuticals to the seeds of plants that are resistant to diseases. The rapidly increasing knowledge of living cells has facilitated the control of complex processes in living cells, e.g., the transfer of properties from one organism to another (genetic engineering) (19). In addition, it may become possible to produce large quantities of mid-spectrum agents in genetically modified common microorganisms. The knowledge and technology may be readily obtained from the Internet.

Biotechnology will probably also be applied toward the development of new biological weapons, such as microorganisms that have a long storage life, are resistant to the forces and energies necessary to disperse them, are highly virulent, difficult to diagnose, resistant to antibiotics, and survive in the environment. Finally, it might become possible to improve the contagious properties of any infectious disease. This point is a complex matter, as evidenced by the fact that in the 1990s, up to 30,000 people worked in biopreparation in the former Soviet Union to improve the effectiveness of biological warfare agents. Biotechnology is expected to be a dominant technology in the coming decades, and will be exploited for hostile purposes, as have been other major technologies. "Biotechnology will not only allow us to devise new methods to destroy life but also to manipulate it– including such processes as thinking, development, reproduction, and the very means by which traits are passed from parents to offspring." (19) Even the human genome project may contribute to the development

of racially selective warfare agents. This would induce a radical change in the nature of warfare. These views are echoed by several experts. One such view is presented in a SIPRI fact sheet [20]. A close watch biotechnology developments is required to identify the development of hostile practices. Furthermore, a political barrier must be created, e.g., via the BTWC that prevents these developments. In his recent book "Bioviolence" (Cambridge University press 2007); Kellmann suggested the creation of a license system for all bio-related work.

The number of potential chemical and biological warfare agents will only increase. Agents do not disappear from the list, but their relative importance changes according to circumstances. Anybody, be it a State, a non-State actor, or an individual seeking CB capabilities, will use any material:

- available to them;
- that they believe they can effectively deliver;
- that they consider to have adequate toxicity;
- the use of which they perceive will give them an advantage or accomplish their goals.

# 3.4.4. Poisoning of food.

In the past, the food chain has been the target for chemical contamination. In some cases, the perpetrator's objective was to extort from food-producing companies substantial sums of money to prevent contamination from happening. On another occasion, the goal was to disrupt the export of certain countries' products, in this case oranges. Even if contamination were criminally accomplished as described, only a small number of casualties will likely be inflicted. Additionally, in most cases, companies involved in the production of food have taken measures to secure the production lines. Because of the relatively low casualty numbers and the precautions taken by the companies, food production will not be a prime target for terrorist groups.

Another criminal activity involving the food chain, which occurred in Austria, was the deliberate mixing of wine with automobile anti-freeze to enhance the appearance of the wine. The glycol in the anti-freeze affected the health of several people. In another criminal case, low-grade olive oil was mixed with high temperature resistant oils and lubricants and marketed as a cheap replacement for cooking oil. This occurred in Spain and Morocco. The tri-ortho-cresyl phosphate in the oils caused paralysis of the extremities (arms and legs). The victims of these sorts of activities are typically the less affluent classes in a society and are not the prime target for terrorists.

On regular occasions, outbreaks of mouth and foot diseases in cattle in Western Europe, bird flu in Asia, and pests amongst pigs have been attributed to terrorists, the main goal being to inflict economic damage on a society. Although such biological attacks against livestock are possible, and severe economical damage could result, it is not very likely that terrorists would achieve their political aims through these activities.

# 3.4.5. Poisoning of drinking water

All modern societies are vulnerable to poisoning of the drinking water supply with chemical or, more likely, biological agents. This is the case for both the civilian society and for military

installations. The drinking water supply consists of several processing steps, beginning with (1) a source of "raw" water that (2) is transported to (3) a treatment plant. A main piping system transports the water to (4) reservoirs, usually in water towers, from where it is distributed by gravity in a secondary piping system to (5) consumers.

The raw water source (1) is the most difficult to protect but, fortunately, also the most difficult to effectively contaminate. To begin with, chemical and/or biological contamination is, to a large extent, diluted by the large volume of water. Secondly natural processes, such as inactivation by sunlight, degradation of the pathogens, hydrolysis, and oxidation will further decrease the concentration of the contamination. Thirdly, a large fraction of the contamination will be removed in the treatment plant (3). Finally, only a small fraction of the water supplied to an individual home is consumed, with much of it boiled first.

Contaminating water during transport (2) to the treatment plant would reduce dilution and degradation due to natural causes. However, the other mitigating factors may still apply, and relatively large quantities of contaminant would have to be introduced into the system to be effective. The water treatment plant (3) would be a good place to contaminate drinking water, but any irregular activity will be quickly noted and action taken to prevent the spread of the contamination. Under any circumstance, it is wise to control access to water treatment plants.

The main water supply leaving the water treatment plant affords another opportunity to penetrate the system and introduce contamination, but the piping system is under a relative high pressure. In addition to the high pressure, time will pass before the water arrives at the reservoir from which it is distributed to the users. This allows the chlorine present in most supply systems time to react with any contamination present. As noted by a group of experts from the WHO, it is possible for an experienced drinking water engineer to penetrate the system, but relative large quantities of a chlorine-resistant pathogen are required to produce a significant effect.

The water reservoir (4) is the easiest point in the supply chain to introduce contaminants because the system is not under pressure at this point. Restricted entry to these reservoirs is, therefore, mandatory. Fortunately, water has an appreciable residence time in reservoirs, which allows chlorine, if present, to react with the contaminants. The system would still be vulnerable to chlorine-resistant pathogens, such as cryptosporidium. Some water supply systems have banned chlorine from the drinking water supply. Consequently, those water supplies are also vulnerable to the other pathogens, such as Shigella and Vibri cholerae. Most lists of potential water pollutants mention E-coli 157 (hamburger disease, resulting from the consumption of contaminated and under-cooked meat) and the Noro virus famous for its quick action and secondary infections. In some cases, persons on board cruise ships or the elderly in nursing homes became sick during the consumption of a meal in a dining room. Nausea arose very quickly and the affected people often vomited before they could reach a bathroom. The aerosol spread during vomiting was sufficient to cause several secondary casualties.

The pipe system carrying water to consumers may be penetrated by an experienced engineer. There is still pressure in the system, but this pressure is relatively low. The residence time of the water reaching the consumers is short at this point. Usually, military installations get water via a main supply line. Some chemical agents, including VX, are sufficiently toxic to do serious harm when introduced into the drinking water at this point in the supply system. More potent toxins that are resistant to hydrolysis will also work well. An Al Qaida manual mentions the use of ricin for polluting drinking water, but large quantities of a pure product are required to have an effect. The harm produced by Legionella bacteria in a water supply is well-known. Consumption of Legionella-contaminated water is not required, and taking a shower with Legionella-contaminated water can be dangerous. Fortunately, the growth of appreciable quantities of Legionella is rather difficult. Most of the biological threat agents mentioned as respiratory hazards also are hazards to the water supply, either through drinking or through showering. However, the poisoning of drinking water should be viewed in perspective. In the Netherlands, the consumption of non-boiled water is, on average, <0.2 L/day and the total use is 500 L/day. If a terrorist had access to a toxic compound with an effective toxicity of 0.1 mg/kg body weight, to be effective, the contaminated water would need to contain a concentration of 35 mg/L (a 70 kg person requires 7 mg for an effective dosage in 0.2 L water). Provided that there is no loss of active compound in the water, one still must inject 350 kg of a potent toxin into the main water supply to attack a small town. The G-agents are ten times less toxic, and 3.5 tons would be required for the same purpose. The reduction in toxin concentrations via dilution and water treatment is such that a raw water source must be contaminated with 350 tons of a potent toxin to affect a population slightly larger than that found in a "small town". Elderly people or those with depressed immune systems can fall victim to pathogens (for instance, the cryptosporidium incident in Milwaukee in 1993) much more easily than healthy young males, from which toxicity values are usually derived.

Although it is not easy to contaminate drinking water supplies effectively with toxic agents that affect typically the elderly and infirm, the psychological effect on the general population is usually very serious. Contamination of a water supply to a military installation would have serious consequence, because all personnel on the installation use the same water supply. Certainly, some critical personnel will fall victim to the contamination. It would, therefore, be wise to protect the water that is consumed without boiling by introducing filters/disinfectants that destroy pathogens and remove chemicals and toxins. Filters may also remove excess chlorine and organic compounds that have reacted with the chlorine. Systems in which water chlorination has been suspended should have facile reactivation capabilities.

# **3.5.** Threat and effect levels, threats that are not threats

The degree to which the release of an agent forms a hazard is determined by many factors, such as the terrain or meteorological conditions. Very important aspects are the quantity of agent released and the concentration or dosage that induces effects in man. An opponent cannot release unlimited quantities of an agent, and the effective levels, the dosage at which the agent produces an effect, are not infinitesimally small. An illustrative example is the statement made during the war in the former Yugoslavia that the release of 10,000 tons of chlorine would kill a large fraction of the population in Western Europe. Some threats can be excluded from the wide spectrum of potential threat scenarios. Examples are:

-The threat of chemical agent aerosols in liquid or solid form, or dusty agent to the skin;

- -The threat of biological agents to skin;
- -The threat of liquid TICs to skin.

More examples of excluded threats may be identified among biological contaminants of the drinking water, because agents usually cannot survive in treated water or in a purification system. Often, the threat of an agent is incorrectly assessed.

### 3.5.1. Threats of chemical aerosols to the skin

Small particles that can easily penetrate clothing are difficult to produce. To reach a production efficiency of 25% particles in the desired size range requires advanced technology. Often, improvised systems have an efficiency of only a few percent. Spraying particulate matter from a solution, e.g., from an aircraft, is nearly impossible. Larger droplets will evaporate during the free fall and, to be effective, must be the correct particle size upon reaching the ground level. Aircraft vortices and the speed of an aircraft will influence the drop sizes generated. Consequently, some drops will be too large and will be deposited on the ground, whereas others will be too small and will float in the air far above the ground level.

NATO AEP 38, in describing the characteristics of protective clothing, stated that the CW dosage delivered as an aerosol is at most half the worst-case realistic threat dosage in the vapor state. Once personnel wearing air-permeable protective suits are exposed to the aerosol cloud, the agent will penetrate the fabric, and harm is only caused by the amount that is deposited onto the skin. However, only a small fraction will be deposited onto the skin, and most of the challenge is carried away by airflow away from the skin. Air-impermeable clothing does not allow penetration of the aerosol, but the ingress of an agent due to the bellows effect (see chapter 6) allows even more aerosol to reach the skin. Elaborate experiments with human volunteers dressed in protective clothing have shown that the amount of agent deposited onto the skin is small (21). In fact, the deposition is so small that it only leads to minor effects in cases of exposure to realistic worst-case challenges. Skin exposure to chemical aerosols is not a significant threat. This conclusion may change if agents are introduced that are more toxic through the skin than, e.g., mustard or VX, or if more efficient aerosol disseminating techniques become available. However these agents most likely will also have an increased respiratory toxicity and will be more effective through this route, even when wearing a mask. (See biological aerosols.)

#### 3.5.2. Threats of biological agents to the skin

All biological agents are solids that may be dispersed as a liquid suspension. Once disseminated as an aerosol, they can challenge the breathing system protected by a mask and the skin protected by protective clothing. Only one biological agent is known to affect the skin, anthrax, which produces skin irritations and wool sorters disease. A mycotoxin, the T2 toxin, has been mentioned as a skin irritant, but it is doubtful that this is a real threat. (See section 3.1.2.h.) Although the T-2 toxin showed some toxic effects, it was assessed by the WHO as being far less toxic than most other toxins. It has been said that brucellosis and tularemia act via the skin, but the experts at the WHO only cite anthrax. For this reason, anthrax will be used in an example. The toxicity considerations hold for unbroken skin without lesions. It is, however, highly likely that lesions will facilitate the transport of biological aerosols into the human body. To facilitate appreciable transport of a biological aerosol, an open wound is necessary. Some soldiers may face the unfortunate situation of incurring a wound during a biological attack. First responders have the time to cover lesions to a sufficient degree before entering the contaminated zone.

A question to be answered is: what is the lethal inhaled exposure dosage (LD<sub>50</sub>) for a man protected by a mask? The LD<sub>50</sub> for anthrax is 55,000 spores inhaled. An active man inhaling 27.5 L/min of air containing 1000 spores/L for 2 min is exposed to the LD<sub>50</sub>. When protected by a mask offering a protection factor of 1000/10,000/100,000, 2 minutes exposure to concentrations of  $10^{6}/10^{7}/10^{8}$  spores/L results in exposure dosages of  $2x10^{9}/2x10^{10}/2x10^{11}$ 

spores-min/m<sup>3</sup>, which yield an inhaled  $LD_{50}$ . The study mentioned in the previous paragraph (21) permits calculation of the deposition rate onto bare skin from such a challenge. The predicted deposition rate is at most 0.001 m/min, which would result in a deposition of 200/2000/20,000 spores/cm<sup>2</sup>. In theory, the deposition velocity is two orders of magnitude lower, as predicted by Fedele (22). Exact values of the skin toxicity of anthrax are not available, but estimates are all above 100,000 spores/cm<sup>2</sup>. This leads to the conclusion that skin effects should only be expected if personnel are protected by a very good mask that provides a protection factor of 1 million, because lower PF masks allow death from respiratory exposure long before skin effects may appear. It is a different story when one actually handles anthrax-contaminated wool or is exposed to military grade anthrax powder, as in the case of the US anthrax letter incidents. In that case, the agent was transferred to the skin in a concentrated form.

# 3.5.3. Threats of liquid TIC to the skin

Exposure of TICs to the skin is an unlikely threat because the probability that the military or a first responder will be sprayed with TICs or will face liquid TIC contamination as a result of a "splash" is extremely small. In the vicinity of TIC storage, contamination by a spray mechanism may be feasible, but it is unlikely that it would produce effects. TICs with high volatility may quickly evaporate from clothing and, if outdoors, may be carried away by the wind, thereby forming only a very short-term vapor challenge. TICs with a low vapor pressure will generate minimal vapor, and simple textile layers will stop the liquid. Intermediate volatility TICs may generate sufficient vapor to affect the skin. However, these types of vapors are well-adsorbed by the active carbon in chemical protective suits.

#### **3.6.** Conclusions

- The threat agent spectrum may be divided into four categories: chemical warfare agents, TICs, mid-spectrum agents, and biological warfare agents.
- TICs can be used as chemical warfare agents when they have an  $LCt_{50}$  value of less than 100,000 mg·min/m<sup>3</sup>.
- According to the definition of TICs, phosgene and cyanides should be considered TICs.
- Mustard agents and the nerve agents of the G- and V-type represent the most important chemical warfare agent threats.
- It is possible that ways can be found to circumvent the verification regimes of the CWC, i.e., by developing warfare agents that are not in the CWC schedules. However, such developments would be covered by the General Purpose Criterion and, thus, be prohibited under the CWC. Challenge inspections could be used to verify if such developments are permitted under the CWC.
- Seven toxins have been identified as potential mid-spectrum agents. Syntheses of toxins will be facilitated by developments in organic chemistry and biotechnology.
- Concerning bacteria and viruses, eight organisms have been identified as potential biological warfare agents.
- Advances in microbiology and biotechnology may allow an extension of the number of potential biological warfare agents or enable the optimization of existing ones.
- The most hazardous method for disseminating bacteria and viruses is as a respirable aerosol. Three ways of disseminating respirable aerosols have been identified. The easiest but least effective method is explosive dissemination. Dissemination of

respirable aerosols from sub-munitions is assessed as technologically very advanced.

- The BTWC is not expected to have a verification regime any time soon. This is one of the reasons why good bio-defense capabilities are of the utmost importance to counteract bio-threats.
- The casualty potential of an agent used in war against unprotected military is not determined by the quantity released divided by the effective dosage. WWI statistics showed that, on average, a million times the effective dosage must be released to produce a single casualty.
- The casualty potential of an agent used in terrorist incidents is not known. Again, the casualty potential is lower than the quantity released divided by the effective dosage. When released in an enclosed space, the casualty potential is probably one to two orders of magnitude higher than the WWI statistical value.
- Chemical agent aerosols from realistic worst-case challenges are not sufficiently deposited onto the skin to form a health hazard.
- Biological aerosols form a very serious respiratory hazard but present no hazard to the unbroken skin.
- It is highly unlikely that military and first responders will be confronted with sprays or splashes of liquid TICs. Even if they are confronted, protective suits containing active carbon form a first line of defense.

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**Annex 3A review of toxic industrial chemicals, hazard index ranking** Reprinted from the US/UK/CAN ITF-25 Final Report, "Hazard from Toxic Industrial Chemicals", Page 16.

| HIGH                   | MEDIUM                   | LOW                             |
|------------------------|--------------------------|---------------------------------|
| ammonia                | acetone cyanohydrin      | allyl isothiocyanate            |
| arsine                 | acrolein                 | arsenic trichloride             |
| boron trichloride      | acrylonitrile            | bromine                         |
| boron trifluoride      | allyl alcohol            | bromine chloride                |
| carbon disulfide       | allyl amine              | bromine pentafluoride           |
| chlorine               | allyl chlorocarbonate    | bromine trifluoride             |
| diborane               | boron tribromide         | carbonyl fluoride               |
| ethylene oxide         | carbon monoxide          | chlorine pentafluoride          |
| fluorine               | carbonyl sulfide         | chlorine trifluoride            |
| formaldehvde           | chloroacetone            | chloroacetaldehvde              |
| hydrogen bromide       | chloroacetonitrile       | chloroacetyl chloride           |
| hydrogen chloride      | chlorosulfonic acid      | cvanogen                        |
| hydrogen cyanide       | crotonaldehvde           | diphenylmethane-4'-diisocyanate |
| hydrogen fluoride      | diketene                 | ethyl chloroformate             |
| hydrogen sulfide       | 1.2-dimethyl hydrazine   | ethyl chlorothioformate         |
| nitric acid fumino     | dimethyl sulfate         | ethylene imine                  |
| phoseene               | ethylene dibromide       | ethyl phosphonothioicdichloride |
| phosphorus trichloride | hydrogen selenide        | ethyl phosphonous dichloride    |
| sulfur dioxide         | iron pentacarbonyl       | hexachlorocyclopentadiene       |
| sulfuric acid          | methanesulfonyl chloride | hydrogen jodide                 |
| tungsten hevafluoride  | methyl bromide           | isobutyl chloroformate          |
| tungsten nevanuorite   | methyl chloroformata     | isopropyl chloroformate         |
|                        | methyl chlorosilana      | isopropyl chloroformate         |
|                        | methyl bydrazina         | n butul ablaraformata           |
|                        | methyl iceguanate        | nitrio ovido                    |
|                        | methyl isocyanate        | mulc oxide                      |
|                        | methyl mercaptan         | n-propyl chloroformate          |
|                        | n-butyl isocyanate       | parathion                       |
|                        | nitrogen dioxide         | perchloromethyl mercaptan       |
|                        | phosphine                | sec-butyl chloroformate         |
|                        | phosphorus oxychloride   | sulfuryl fluoride               |
|                        | phosphorus pentafluoride | tert-butyl isocyanate           |
|                        | selenium hexafluoride    | tetraethyl lead                 |
|                        | silicon tetrafluoride    | tetraethyl pyrophosphate        |
|                        | stibine                  | tetramethyl lead                |
|                        | sulfur trioxide          | toluene 2,4-diisocyanate        |
|                        | sulfuryl chloride        | toluene 2,6-diisocyanate        |
|                        | tellurium hexafluoride   |                                 |
|                        | tert-octyl mercaptan     |                                 |
|                        | titanium tetrachloride   |                                 |
|                        | trichloroacetyl chloride |                                 |
|                        | trifluoroacetyl chloride |                                 |

# ANNEX 3B: DEFINITION OF TERMINOLOGY FOR BIOLOGICAL WARFARE AGENTS

<u>Infectivity</u>: The infectivity of an agent reflects the relative ease with which microorganisms establish themselves in a host species. Pathogens with high infectivity cause disease with relatively few organisms, whereas those with low infectivity require a larger number of organisms. High infectivity does not necessarily mean that the symptoms and signs of disease appear more quickly, or that the illness is more severe.

<u>Virulence</u>: The virulence of an agent reflects the relative severity of disease produced by that agent. Different microorganisms and different strains of the same microorganism may cause diseases of different severity.

<u>Toxicity</u>: The toxicity of an agent reflects the relative severity of illness or incapacitation produced by a toxin.

<u>Pathogenicity</u>: The pathogenicity reflects the capability of an infectious agent to cause disease in a susceptible host. A sufficient number of microorganisms or quantity of toxin must penetrate the body to initiate infection (the infective dose) or intoxication (the intoxication dose). Infectious agents must then multiply (replicate) to produce disease.

<u>Incubation Period</u>: The time between exposure and the appearance of symptoms. This is governed by many variables, including the initial dose, virulence, route of entry, rate of replication, and host immunological factors.

<u>Transmissibility</u>: Some biological agents may be transmitted directly from person to person. Indirect transmission (for example, via arthropod vectors) may be a significant means of spread as well. In the context of BW casualty management, the relative ease with which an agent is passed from person to person (that is, its transmissibility) constitutes the principal concern.

<u>Lethality</u>: Lethality reflects the relative ease with which an agent causes death in a susceptible population.

Morbidity: The rate at which a susceptible population becomes sick.

Mortality: The rate at which a susceptible population dies from infection.

<u>Stability:</u> The viability of an agent is affected by various environmental factors, including temperature, relative humidity, atmospheric pollution, and sunlight. A quantitative measure of stability is the agent's decay rate (for example, "aerosol decay rate").

<u>Additional Factors</u>: Additional factors that may influence the suitability of a microorganism or toxin as a biological weapon include: the ease of production, the stability during storage or transportation, and the ease of dissemination.

The Center for Disease Control uses the following criteria:

- 1. The public health impact based on morbidity, mortality, and propensity for contagion;
- 2. Delivery potential based on the ease of mass production and distribution, as well as stability of the agent;
- 3. Special preparation requirements based on whether vaccines or medications should be stockpiled, agent detection increased, or tools to identify the agent produced; and
- 4. Public perception of the agent and its capacity to generate fear.

The CDC identified Variola major, Bacillus anthracis, Yersinia pestis, the Botulinum toxin, Francisella tularensis, and filoviruses/arenaviruses as biological agents of greatest public concern.

# 4. Toxicology and human toxicity estimates

### 4.1. Physiological and toxicological properties

A short description will be provided of the symptoms induced by an agent and of its mode of action, if known. Additionally, the rate of action, i.e., the rate at which symptoms become apparent after exposure, will be discussed. The rate of action may differ widely across chemical warfare agents. Knowing this rate of action is important for both the aggressor and the defender. The aggressor requires such information to plan his battle tactics and the defender requires such information for diagnosis and to determine the period of time in which medical countermeasures may be effectively taken. For most CWs, the rate of action is very rapid, seconds to minutes. Prolonged exposure causes more severe casualties. It is, therefore, of prime importance for emergency services to develop a doctrine and acquire protective equipment that enables rapid response.

A simple distinction between toxic and non-toxic compounds cannot be made. In 1654, Paracelsus wrote:

"Was ist dat nicht gifft ist: alle ding sind gifft und nichts ohne gifft. Allein die dosis macht das ein ding kein gift ist."

(What is not poisonous: all things are poisonous and nothing is without poison. The dose only determines whether a thing is not a poison.)

An important characteristic of chemical warfare agents, therefore, is the dose at which it causes a physiological effect.

This dose may depend on the route taken by the agent to enter the body. The three natural routes are inhalation, through the skin or eyes, or via ingestion. The air in the lungs is separated from the blood stream by a wall of two cell layers. On the skin, this layer is several thousands of cells thick. The thickness of the skin barrier varies widely over the body. In the case of the eyes, the nerve endings and blood vessels are very close to the surface. One would expect that thinner barriers facilitate the pace of effects. Gases, liquids, and solid aerosols can be taken up via the respiratory organs. Liquids, and in certain cases gases and aerosols, invade the body via the skin and eyes. Skin and eye effects, however, are usually local, although nerve and blister agents cause systemic intoxication. The ingestion route is less important from the perspective of chemical warfare.

When describing toxicity, Haber defined dosage as the concentration times exposure time, and assumed that a dosage was limited to this measure. Therefore, a low concentration exposure over a long period of time was assumed to cause the same effects as a short duration exposure at a high concentration. Haber himself was the first to recognize that the value of the formula was limited. Exposures to low concentrations of agents over long periods of time do not appear to cause the same level of harm. The equation was, therefore, corrected as (C-a) x t = Constant. Later, the toxic load concept was introduced, as explained below. As far as it is known, the toxic load concept holds for almost all CW agents and for most volatile TICs introduced via the respiratory route. Phosgene possibly constitutes the only exception. In the toxic load concept, dosage is described as Cexp.n x t = Constant, where the power n varies between 1 and 4. The toxic load concept has very important consequences for protection inside structures, such as houses, where lower concentrations and longer exposure times are less harmful, as made clear by the example below. An agent that is weakly adsorbed by the

active carbon in a gasmask filter is slowly desorbed and presents itself at a lower concentration to the respiratory system. This is an often omitted factor that enhances significantly the protective provided by a mask.

A hypothetical chemical warfare agent has an  $ICt_{50}$  value of 60 mg·min·m<sup>-3</sup> for a 10 min exposure. Assuming a toxic load exponent of 1.5, the  $ICt_{50}$  for a 2 min exposure would be 35 mg·min·m<sup>-3</sup>, which implies that the agent is apparently more toxic under these conditions. On the other hand, the  $ICt_{50}$  for a 60 min exposure would be 110 mg·min·m<sup>-3</sup>, implying that the agent was apparently less toxic under these conditions. This phenomenon arises because the body is capable of detoxifying the agent to a certain extent and at a limited rate. Consequently, if the body is exposed to a relatively low concentration of agent, it may detoxify a fraction of the toxicant before it reaches toxicologically relevant target. The important implication is that lower concentrations that are introduced upon penetration of filters or inside structures with sealed rooms are less hazardous.

The following doses are reported for chemical warfare agents, if available:

The median lethal dose,  $LCt_{50}$ , is the dosage (vapor or aerosol concentration of an agent multiplied by the time of exposure) that is lethal to 50 per cent of the exposed individuals. The  $LCt_{50}$  value may depend on the time of exposure. For a number of agents, the value increases with increasing exposure time. When intoxication takes place by inhalation, the  $LCt_{50}$  depends on the breathing rate and volume.  $LCt_{50}$  is expressed in milligrams times minutes per cubic meter.

The median incapacitating dose,  $ICt_{50}$ , is the dosage (vapor or aerosol concentration of the agent multiplied by the time of exposure) that is sufficient to disable 50 per cent of the exposed individuals. The  $ICt_{50}$  is also expressed in milligrams times minutes per cubic meter.

In some cases of intoxication via the skin, the  $LD_{50}$  value is also given. This is the dose, expressed in grams per individual or milligrams per kilogram, which is lethal to 50 per cent of the exposed individuals. For local skin effects, toxicity is expressed in  $\mu g/cm^2$ . If available, the exponent n in the toxic load equation is given as well.

For some agents, particularly the nerve agents and mustard gas, a comprehensive review of all human and animal test data has been presented. This review has become indirectly available after a thorough review of the toxicology committee of the US National Research Council. The data presented in that review were used by NATO to construct a table of percutaneous toxicities (See paragraph 4.7).

What follows are detailed quantitative figures associated with all classes of agents. These figures are important because, together with the quantitative threat data, they provide guidance for the required physical protection factors and for the minimum required sensitivity of detectors. They are essential references for those who set requirements for protective equipment or for those investigating detector sensitivity levels. Although it is shown that minute quantities can kill, in practice, much higher dosages must be created to achieve the desired effect. The protection factor required for a mask or clothing is not the released quantity of agent divided by the effect dosage, but the dosage that challenges the individual divided by the effect dosage.

# 4.2. Lethal agents

Lethal agents are capable of producing incapacitation, serious injury, or death when used in field concentrations.

#### 4.2.1. Choking agents

Choking agents injure an individual mainly in the respiratory tract. In severe intoxications, edema of the lungs develops, i.e., an abnormal accumulation of liquid in the lung tissue; blisters on the lungs may also develop, the walls of which may burst and release blood and liquid. Edema hinders the exchange of gas in the lungs. Victims may be choked by the agent due to a lack of oxygen.

Phosgene

| 3200 mg·min.  |
|---|
| $1600 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$  |
| immediately after exposure to a high concentration; a delay of 3 h or more after exposure to a low concentration.   |
| See the general description for choking agents. Full toxic effects are<br>not usually apparent until 3 or 4 h after exposure. Most deaths occur<br>within 24 h. |
|   |
| $3200 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$  |
| $1600 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$  |
| Immediately after exposure to a high concentration; a delay of 3 h or more after exposure to a low concentration.   |
| Diphosgene and phosgene induce similar lung effects. In addition, diphosgene has a slight lachrymatory effect.  |
|   |

# PFIB

The gas PFIB produces pulmonary edema and is generated when polytetrafluorethylene is pyrolized.

| Rate of action: | Possible delayed action.   |
|-----------------|--|
| Symptoms:       | See the general description for choking agents. Low doses may induce |
|                 | headache, coughs, substernal pain, and dyspnoea within the first     |
|                 | hours, and symptoms are aggravated at 6–8 h. pulmonary edema can     |
|                 | cause death 8–48 h after exposure.                                   |
|                 |  |

# Chloropicrin

| ICt <sub>50</sub> : | $50 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$                           |
|---------------------|--|
| LCt <sub>50</sub> : | $20,000 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$                       |
| Rate of action:     | As an inhalation poison, it has a somewhat shorter latency time than phosgene. |

Symptoms: Chloropicrin acts as a choking, vomiting, and tear gas agent. Lung effects are comparable to those induced by phosgene. The agent may also cause nausea and vomiting. It has a burning effect on the eyes and causes the flow of tears. It is irritating to the skin; as a liquid, it can cause blisters.

#### 4.2.2. Blood poisoning agents

The blood poisoning agents hydrogen cyanide and cyanogen chloride interfere with cellular respiration (the utilization of oxygen by the cells) due to interactions with the enzyme cytochrome oxidase. Symptoms may set in extremely rapidly. Their effects depend strongly on the concentration to which a casualty has been exposed because the agents are relatively rapidly detoxified in the body. The agents are considered to be of relatively low relevance to chemical warfare because of their high volatility in combination with a moderate toxicity.

Hydrogen cyanide

| LCt <sub>50</sub> : | largely depends on concentration; 2000 mg·min·m <sup>-3</sup> at 200 mg·m <sup>-3</sup> , 4500 mg·min·m <sup>-3</sup> at 150 mg·m <sup>-3</sup> . |
|---------------------|---|
|                     | Toxic load exponent is reported to be $>3$ .  |
| Rate of action:     | Very rapid.   |
| Symptoms:           | The first symptoms of mild intoxication are a feeling of weakness,  |
|                     | headache, confusion, and sometimes nausea. Respiration is stimulated.   |
|                     | During severe intoxication, breathing rapidly weakens after the initial   |
|                     | stimulation. Victims are unconscious within half minute and violent   |
|                     | convulsions begin. Arterial and venous blood has the same color   |
|                     | because oxygen is no longer absorbed by the tissues. This results in a  |
|                     | faint pale red color of the skin, a characteristic symptom of acute   |
|                     | intoxication. In less severe intoxication, the skin may become bluish   |
|                     | (cyanotic) due to insufficient breathing.   |

#### Cyanogen chloride

Cyanogen chloride is more volatile than hydrogen cyanide and, unlike the latter agent, can produce casualties in sub lethal doses. In addition to the effects caused by hydrogen chloride, cyanogen chloride has a powerful lachrymatory action and a choking effect. The agent penetrates the filter elements of a gas mask more readily than most other agents.

| LCt <sub>50</sub> : | $11,000 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}.$                   |
|---------------------|---|
|                     | Toxic load exponent n>3.  |
| ICt <sub>50</sub> : | 7000 mg·min·m <sup>-3</sup> ; median detectable concentration by its        |
|                     | lachrymatory effect, $12 \text{ mg} \cdot \text{m}^{-3}$ .                  |
| Rate of action:     | Rapid.  |
| Symptoms:           | Cyanogen chloride produces effects similar to those of hydrogen             |
|                     | cyanide. In addition, it has irritating and lachrymatory effects and acts   |
|                     | as a lung irritant after inhalation. Lung effects, e.g., edema, are similar |
|                     | to those caused by phosgene.  |

#### 4.2.3 Nerve agents

Nerve agents belong chemically to the group containing organophosphorus compounds or organophosphates. The first synthesis of a highly toxic organophosphate was reported in 1854 by the French nobleman and chemist Philippe de Clermont. Although the compound was handled with relatively simple equipment and was even tasted, according to the chemical practice at that time, the toxic properties of the compound were not observed. The first observations of the toxicity of organophosphates were made by Lange and von Krueger from the effects of the vapor on themselves during their synthesis work in 1932. In 1936, Schrader began research on this type of compound in the laboratories of IG-Farben to develop synthetic insecticides.

This work initiated the synthesis of a long series of biologically active organophosphates that continue to find applications as pesticides and as pharmaceuticals for the treatment of diseases such as myasthenia gravis and glaucoma. The classic biologically active organophosphates can be denoted by the general structure  $R_1(R_2)P(O,S)X$ , where  $R_1$  and  $R_2$  are either alkoxy, aryloxy, alkylmercapto, (alkyl)amino, alkyl, or aryl groups, and X is a residue easily liberated upon nucleophilic attack of the phosphorus, i.e., by fluoride, chloride, cyanide, phosphate, phenolate, or thiolate. Nerve agents contain a P=O moiety. In insecticides, this is often replaced by P=S, and X consists of a less reactive group than in the case of a nerve agent. Examples of organophosphorus insecticides are Malathion, parathion, and dichlorvos.

The first compound later referred to as a nerve agent, Tabun, was prepared by Schrader in 1936. Tabun and Sarin were produced for use as chemical weapons in Germany during the second World War. (For a full description of the history of nerve agents and the origin of the names Tabun and Sarin, see reference 18a in Chapter 3.) These agents, together with Soman prepared in 1944, belong to the group of G agents. A group of even more toxic nerve agents, the V agents, was developed in the 1950s. These agents are more stable and less volatile than the G agents and are, consequently, more persistent.

The toxicity of the nerve agents (and of the organophosphorus insecticides) is mainly due to their inhibitory effects on the enzyme acetylcholinesterase. This enzyme acts as a highly active catalyst for the hydrolysis of acetylcholine, a transmitter substance responsible for the transfer of nerve impulses across the cholinergic synaptic junctions. Inhibition of the enzyme causes accumulation of acetylcholine, which leads to over excitation or paralysis, depending on the type of cholinergic junction involved. Over excitation leads to the following symptoms: increased production of saliva, runny nose, difficulty breathing due to secretions in the respiratory tract, increased perspiration, vomiting, diarrhea, involuntary discharge of urine and defecation, cramps, and convulsions. The most critical effects are paralysis of the respiratory muscles in combination with the effects on the respiratory centre in the central nervous system. Nerve agents also affect the central nervous system and produce symptoms such as anxiety and stress. Death is generally caused by respiratory failure.

A typical symptom of intoxication by nerve agents in the form of vapor or aerosol is miosis, i.e., contraction of the pupil, induced at very low doses. Miosis impairs night vision and is accompanied by impaired accommodation. As a consequence, the direct effects of nerve agents on the eye are strongly incapacitating.

The nerve agents are much more toxic when absorbed by the eye than through the skin. Respiratory lethal doses and liquid agent in the eye kill in 1 to 10 minutes. After skin contamination, symptoms do not generally appear for at least half an hour. Death may be delayed for 1 to 2 h or even longer. Respiratory toxicity is believed to follow the toxic load concept with n=1.5 for the G-agents. At least five nerve agents have been weaponized: the G-agents Tabun, Sarin, Cyclohexyl Sarin, Soman, and the V-agent VX.

The nerve agents represent a chemical threat that is unsurpassed by any other group of chemical warfare agent. This is only partially due to their extremely high toxicity and their ability to rapidly induce toxic effects. They are stable, easy to disseminate, and relatively simple to produce from inexpensive starting materials. They may enter the body by inhalation in vapor or aerosol form and through the skin and the eyes as vapor or liquid. They have various persistencies, ranging from a low persistency for Sarin to the relatively persistent V-agents.

The nerve agents are listed as Schedule 1 Chemicals in the Chemical Weapons Convention (CWC).

| Route              | Definition            | Effect(s)             |
|--------------------|-----------------------|-----------------------|
| Inhalation and     | clinically noticeable | miosis, and/or tight  |
| ocular exposure to |                       | chest, and/or         |
| vapor              |                       | rhinorrhea, and/or    |
|                    |                       | headache              |
|                    | severe                | up to prostration and |
|                    |                       | convulsions           |
|                    | lethality             | death                 |
| Percutaneous       | clinically noticeable | slight ChE            |
| exposure to vapor  |                       | inhibition; local     |
|                    |                       | sweating or           |
|                    |                       | twitching             |
|                    | severe                | nausea, vomiting,     |
|                    |                       | cold sweating         |
|                    | lethality             | death                 |
| Percutaneous       | severe                | up to prostration and |
| exposure to liquid |                       | convulsions           |
|                    | lethality             | death                 |

Definitions of the effects of nerve agents

# 4.2.3.1. Tabun

Recommended human toxicity estimates: Inhalation and ocular exposure (10 min exposure): Clinically noticeable effects:  $ECt_{50} 0.5 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Severe effects:  $ICt_{50} 85 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Lethality:  $LCt_{50} 120 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ 

Percutaneous exposure to vapor (30 min exposure): Clinically noticeable effects:  $ECt_{50}$  2,000 mg·min·m<sup>-3</sup> Severe effects:  $ICt_{50}$  12,000 mg·min·m<sup>-3</sup> Lethality:  $LCt_{50}$  15,000 mg·min·m<sup>-3</sup> Percutaneous exposure to liquid: Severe effects:  $ID_{50}$  900 mg per 70 kg man Lethality:  $LD_{50}$  1,500 mg per 70 kg man

Rate of action: Very rapid Symptoms: see general description of Nerve agents.

4.2.3.2. Sarin

Recommended human toxicity estimates: Inhalation and ocular exposure (10 min exposure): Clinically noticeable effects:  $ECt_{50} 0.5 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Severe effects:  $ICt_{50} 40 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Lethality:  $LCt_{50} 60 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ 

Percutaneous exposure to vapor (30 min exposure): Clinically noticeable effects:  $ECt_{50}$  1,200 mg·min·m<sup>-3</sup> Severe effects:  $ICt_{50}$  8,000 mg·min·m<sup>-3</sup> Lethality:  $LCt_{50}$  10,000-12,000 mg·min·m<sup>-3</sup>

Percutaneous exposure to liquid: Severe effects:  $ID_{50}$  1,000 mg per 70 kg man Lethality:  $LD_{50}$  1,700 mg per 70 kg man

Rate of action: very rapid Symptoms: see general description of nerve agents.

4.2.3.3. Soman

Recommended human toxicity estimates: Inhalation and ocular exposure (10 min exposure): Clinically noticeable effects:  $ECt_{50} 0.3 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Severe effects:  $ICt_{50} 40 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Lethality:  $LCt_{50} 60 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ 

Percutaneous exposure to vapor (30 min exposure): Clinically noticeable effects:  $ECt_{50}$  300 mg·min·m<sup>-3</sup> Severe effects:  $ICt_{50}$  2,000 mg·min·m<sup>-3</sup> Lethality:  $LCt_{50}$  2,500–3,000 mg·min·m<sup>-3</sup>

Percutaneous exposure to liquid: Severe effects:  $ID_{50}$  200 mg per 70 kg man Lethality:  $LD_{50}$  300 mg per 70 kg man

Rate of action: very rapid Symptoms: see general description of nerve agents.

### 4.2.3.4. Cyclohexyl Sarin

Recommended human toxicity estimates: Inhalation and ocular exposure (10 min exposure): Clinically noticeable effects:  $ECt_{50} 0.3 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Severe effects:  $ICt_{50} 40 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Lethality:  $LCt_{50} 60 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ 

Percutaneous exposure to vapor (30 min exposure): Clinically noticeable effects:  $ECt_{50}$  300 mg·min·m<sup>-3</sup> Severe effects:  $ICt_{50}$  2,000 mg·min·m<sup>-3</sup> Lethality:  $LCt_{50}$  2,500–3,000 mg·min·m<sup>-3</sup>

Percutaneous exposure to liquid: Severe effects:  $ID_{50}$  200 mg per 70 kg man Lethality:  $LD_{50}$  350 mg per 70 kg man

Rate of action: very rapid Symptoms: see general description of nerve agents.

4.2.3.5. VX

Recommended human toxicity estimates: Inhalation and ocular exposure (10 min exposure): Clinically noticeable effects:  $ECt_{50} 0.1 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Severe effects:  $ICt_{50} 10 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ Lethality:  $LCt_{50} 15 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ 

Percutaneous exposure to vapor (min exposure): Clinically noticeable effects:  $ECt_{50}$  30 mg·min·m<sup>-3</sup> Severe effects: ICt50 60 mg·min·m<sup>-3</sup> Lethality:  $LCt_{50}$  200 mg·min·m<sup>-3</sup>

Percutaneous exposure to liquid: Clinically noticeable effects: 1.75 mg per 70 kg man Severe effects:  $ID_{50}$  2.45 mg per 70 kg man Lethality:  $LD_{50}$  2.8 mg per 70 kg man

Rate of action: very rapid Symptoms: see general description of nerve agents.

4.2.4. Toxins

4.2.4.1. Botulinum toxin A

It has been estimated that a lethal dose for a human being is  $1 \mu g$  when ingested and is even less when inhaled in aerosol form. The first symptoms of poisoning do not appear until 12 to 72 h after contaminated food has been consumed. The onset time is likely to be shorter after inhalation. Symptoms of poisoning are: nausea and diarrhea soon followed by headaches, dizziness, thirst, double vision, persistent constipation, and general weakness. Breathing becomes increasingly labored and muscles throughout the body increasingly weak. Convulsions and death may soon follow.

Botulinum toxins,  $LD_{50}$  below  $10^{-7}$  g/man for the most toxic variety, type A.

The compound is not incorporated into the Schedules of Chemicals contained in the Chemical Weapons Convention.

# 4.2.4.2. Saxitoxin

Symptoms of saxitoxin intoxication are tingling numbness in the lips, mouth, and tongue, muscular weakness, thirst, and prickly feelings in the fingertips. This is followed by increasing muscular incoordination, with ascending paralysis. Death is generally caused by failure of the respiratory muscles. Symptoms usually follow between 10 min and 4 h after the toxin is ingested. Intoxicated personnel surviving for 12 h have a good chance of recovering. Approximately 1 mg saxitoxin is lethal if ingested; the lethal dose by injection is about ten times lower. The LCt<sub>50</sub> value is estimated as 5 mg·min·m<sup>-3</sup>.

# 4.2.4.3. Ricin

The LCt<sub>50</sub> value for ricin is estimated to be 30 mg·min·m<sup>-3</sup>. The LD<sub>50</sub> in mice is around 4  $\mu$ g per kg, which is approximately the same as for most of the nerve agents. The physiological effects of ricin intoxication are delayed. After inhalation, ricin causes lung injuries similar to those produced by phosgene. Symptoms after ingestion of ricin are nausea and vomiting, cramps in the limbs, rapid respiration, and a rise in body temperature. Fatal cases pass into collapse followed by convulsions.

# 4.2.4.4. T-2 toxin

Taken orally or by inhalation, T-2 toxin gives rise to nausea, vomiting, diarrhea, dizziness, headache, and confusion. Inflammation of the gastrointestinal tract results in potentially fatal internal hemorrhages. The number of leukocytes and erythrocytes in the blood decreases steeply. In the fatal phase, hemorrhages appear all over the body with bleeding in the nose, mouth and intestines. The victims die from bacterial infections and by respiratory blockade due to swelling of the throat. The effects were discovered by Joffe et al. when studying food poisoning in the former Soviet Union during WWII. Grains harvested after the winter were contaminated with trichotecenes, one of them being the T-2 toxin. The toxicity of T-2 is markedly species-dependent. Vomiting is induced in cats at 0.1–0.2 mg/kg after oral dosing. Guinea pigs are unaffected at 0.75 mg/kg and start to show effects at a dosage of 2.5 mg/kg. Immunosuppression is observed in rhesus monkeys at 0.5 mg/kg and in mice at 20 mg/kg. In comparison with other toxins, T-2 is not very toxic. The T-2 toxin is said to cause skin inflammation at very low doses but again this is species-dependent. In one study with mice and rabbits, skin effects showed up in one animal but not in the other. The lethal dose for intoxication through the skin or after inhalation is probably one to two orders of magnitude higher than the lethal doses for other nerve agents. (Very little is known about the percutaneous toxicity, some believe that T-2 is not effective on the skin at all). Skin effects vary from erythema and edema to necrosis of the epidermis.

The WHO has evaluated the toxicity of T-2 in food (Evaluation of Certain Mycotoxins in Food. 56th report of the joint FAO/WHO Expert Committee on Food Additives, Geneva,

WHO, 2002 Technical report series No 906) but the respiratory and percutaneous toxicity remain unknown.

# **4.3. Incapacitating agents**

# 4.3.1. Staphylococcus enterotoxin B

As a rule, the toxin is not lethal but as little as  $25 \ \mu g$  can cause severe vomiting and diarrhea. It can be used to contaminate food or to intoxicate by inhalation after dissemination as an aerosol.

Food poisoning is characterized by the sudden and often violent onset of vomiting, diarrhea, and stomach cramps. Patients usually feel normal 24 h after intoxication. Symptoms generally show up within 2 to 4 h after oral or aerosol intoxication. Long-lasting effects or death are rare in humans. The ICt<sub>50</sub> for toxin aerosols in humans has been estimated as approximately 0.5 mg·min·m<sup>-3</sup>; the lethal dose may be two orders of magnitude higher.

# 4.3.2 Vesicants

Vesicants or blistering agents can produce skin injuries resembling those caused by thermal burns. They also severely affect the eyes. By inhalation, the blistering agents affect the upper respiratory tract and lungs, producing lung edema. In addition, the agents have a systemic effect when absorbed in the tissues. Protection from blistering agents is extremely difficult. One of these agents is mustard gas. Some arsenicals, including Lewisite, also belong to the group of blistering agents.

Vesicants are considered to be incapacitating agents, although their safety ratio is low (where the safety ratio is defined as the ratio between the lethal and incapacitating dosages; for nerve agents the safety ratio is very low, for tear agents the ratio is very high). During WWI, less than 2% of the total casualties inflicted by mustard gas were fatal, mostly due to secondary infections.

# 4.3.2.1. Mustard gas

| Route              | Definition            | Effect(s)          |
|--------------------|-----------------------|--------------------|
| Inhalation and     | clinically noticeable | conjunctivitis and |
| ocular exposure to |                       | ocular irritation  |
| vapor              | severe                | temporary          |
|                    |                       | blindness          |
|                    | lethality             | death              |
| Percutaneous       | clinically noticeable | erythema; itching  |
| exposure to vapor  | severe                | erythema and       |
|                    |                       | severe vesication  |
|                    | lethality             | death              |
| Percutaneous       | severe                | erythema and       |
| exposure to liquid |                       | severe vesication  |
|                    | lethality             | death              |

#### Definitions of effects of mustard

Mustard gas, also called sulphur mustard, or, in German, lost or senfgas, is called "the king of war gases". It was responsible for 1/5 of the chemical warfare agent casualties during the First World War and is still considered one of the most hazardous chemical warfare agents. Skin injuries caused by mustard gas are long-lasting and heal much slower than thermal burns. Mustard gas is simple to produce and meets most requirements as a chemical warfare agent, apart from its delayed effect. It slowly dissolves in water and its droplets tend to float for long periods of time on water surfaces, although it has a higher density than water.

Mustard gas is listed as a Schedule 1 Chemical in the CWC.

Rate of action: latency period up to 24 h; usually, symptoms appear 4–6 h after exposure.

Symptoms: The local effects of mustard gas on the skin range from itching and painful inflammation, resembling a first-degree burn, to the formation of large liquid-filled blisters. In the latter case, there is a considerable risk of infection. Mustard gas attacks moist areas of the body most severely, such as the neck, armpits, genitals, chest, or under the breasts. The agent irritates the eyes, and the eyelids may become swollen. Direct contamination of the eyes with liquid mustard gas may induce injuries to the cornea and the iris leading to permanent blindness. After inhalation, mustard gas acts on the lungs similarly to phosgene, and severe intoxication may lead to fatal lung edema. As an alkylating carcinogen, it may also induce systemic effects. The symptoms are reminiscent of injuries from radiation, such as nausea, and injuries to bone marrow, lymph nodes, and the spleen. The resulting drop in white blood cells renders a casualty very susceptible to infections.

<u>Recommended human toxicity estimates</u> *Inhalation and ocular exposure (10 min exposure):* Clinically noticeable effects:  $ECt_{50}$  25 mg·min·m<sup>-3</sup> Severe effects:  $ICt_{50}$  75 mg·min·m<sup>-3</sup> Lethality:  $LCt_{50}$  1,500 mg·min·m<sup>-3</sup>

 $\begin{array}{l} Percutaneous\ exposure\ to\ vapor\ (30\ min\ exposure):\\ Clinically\ noticeable\ effects:\ ECt_{50}\ 50\ mg\cdot min\cdot m^{-3}\ (moderate\ temperature)\\ 25\ mg\cdot min\cdot m^{-3}\ (high\ temperature)\\ Severe\ effects:\ ICt50\ 500\ mg\cdot min\cdot m^{-3}\ (moderate\ temperature)\\ < 200\ mg\cdot min\cdot m^{-3}\ (high\ temperature)\\ Lethality:\ LCt_{50}\ 5,000-10,000\ mg\cdot min\cdot m^{-3}\ (moderate\ temperature)\\ \end{array}$ 

Percutaneous exposure to liquid (moderate temperature): (Clinically noticeable effects:  $ED_{50} 1-2 \mu g \text{ cm}^{-2}$  skin, derived from vapor exposure) Severe effects:  $ID_{50}$  600 mg per 70 kg man Lethality:  $LD_{50}$  1,400 mg per 70 kg man

4.3.2.2 Nitrogen mustard

Nitrogen mustard is one of three well-known nitrogen analogs of mustard gas. The other two agents are ethyl nitrogen mustard (HN-1,  $C_2H_5N$  (CH<sub>2</sub>CH<sub>2</sub>Cl)<sub>2</sub>) and methyl nitrogen mustard (HN-2, CH<sub>3</sub>N (CH<sub>2</sub>CH<sub>2</sub>Cl)<sub>3</sub>). The compounds have toxicological effects similar to mustard gas. They are less stable in storage. Nitrogen mustard is the most stable agent in this respect.

It is more resistant to hydrolysis and oxidation than mustard gas, making decontamination more difficult.

Nitrogen mustards are listed as Schedule 1 Chemicals in the CWC.



A badly burned victim of a Mustard attack. If he survives, he will have an increased probability of developing skin cancer or blindness after some years. Unfortunately, there is little doctors can do.

| LCt <sub>50</sub> : | by inhalation, 1,500 mg·min·m <sup>-3</sup> ;  |
|---------------------|--|
|                     | by skin absorption, 10,000 mg $\cdot$ min $\cdot$ m <sup>-3</sup>                              |
| ICt <sub>50</sub> : | for skin injuries, 2,500 mg·min·m <sup>-3</sup> ; for eye injuries, 200 mg·min·m <sup>-3</sup> |
| Rate of action:     | latency period of 4 to 6 hours. Eye irritation may immediately appear                          |
|                     | in some cases.   |
| Symptoms:           | Skin effects, erythema, and blisters are induced as with mustard gas.                          |
|                     | Eye irritation may appear shortly after exposure. Mild skin injuries are                       |
|                     | caused by exposure to low doses (as low as 10 mg $\cdot$ min $\cdot$ m <sup>-3</sup> ). Lung   |
|                     | effects of nitrogen mustard are similar to those of mustard gas. After                         |
|                     | systemic absorption or ingestion, the agent depresses blood formation                          |
|                     | by inhibiting cell mitosis. Necrosis in the mucous membranes of the                            |
|                     | small intestine causes diarrhea.   |

4.3.2.3. Lewisite

Lewisite is both a blistering agent and a toxic arsenic compound. The effects of Lewisite on the eyes and the skin are immediately observable. Lewisite hydrolyzes very rapidly when dissolved in water or as a vapor in humid air. The arsenic-containing hydrolysis product is toxic but is nonvolatile. Lewisite is, however, of little importance as a chemical warfare agent due to its low stability in a humid atmosphere.

Lewisite (also named L-1) and the analogs (2-chlorovinyl) chloroarsine (L-2) and tris (2-chlorovinyl) arsine (L-3) are listed as Schedule 1 Chemicals in the CWC.

| LCt <sub>50</sub> : | by skin absorption, 100,000 mg·min·m <sup>-3</sup>                           |
|---------------------|--|
|                     | by inhalation, $1,200-1,500 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ |
| ICt <sub>50</sub>   | for skin injuries, 1,500 mg·min·m <sup>-3</sup>                              |
|                     | for eye injuries, less than 300 mg $\cdot$ min $\cdot$ m <sup>-3</sup>       |
| Rate of action:     | rapid  |
| Symptoms:           | The symptoms are similar to those produced by mustard gas. However,          |
|                     | as an arsenic-containing compound, Lewisite also affects the liver,          |

kidneys, and red blood cells. Lewisite induces immediate pain in the skin. Erythema begins within 30 min, and blisters are formed after 10 to 15 h. healing proceeds more rapidly than after mustard contamination. Liquid Lewisite causes an immediate burning effect on the eyes and permanent blindness if not decontaminated almost immediately. Lung irritation appears a relatively short period after inhalation of the agent. As a systemic poison, the agent causes pulmonary edema, diarrhea, general weakness, and low blood pressure.

#### 4.3.2.4. Mustard gas-Lewisite mixtures

Addition of Lewisite to mustard gas lowers the melting point, providing a low-temperature freezing mixture for cold weather operations. The mustard gas to Lewisite ratio in mixtures with the lowest melting point, the eutectic mixture, is 37/63% wt. The following properties apply to the eutectic mixture. Mixtures with higher mustard gas content may also be prepared.

| LCt <sub>50</sub> : | by inhalation, 1,500 mg $\cdot$ min $\cdot$ m <sup>-3</sup>           |
|---------------------|---|
|                     | by skin absorption, 10,000 mg $\cdot$ min $\cdot$ m <sup>-3</sup>     |
| ICt <sub>50</sub> : | for skin injuries, 1500 - 2000 mg $\cdot$ min $\cdot$ m <sup>-3</sup> |
|                     | for eye injuries, 200 mg·min·m <sup><math>-3</math></sup>             |
| Rate of action:     | rapid   |
| Symptoms:           | see mustard gas and Lewisite.   |

#### 4.4. Psychogenic incapacitating agents

Psychogenic incapacitating agents affect the mental state of the individual. Attention has been paid to the psychotomimetic agents, in particular, also called hallucinogens or psychodelic agents. Most of these agents are natural products. Examples include LSD 25, mescaline, psilocybin, and tetrahydrocannabinol (hashish).

Glycolates have been extensively studied in the US as synthetic psychotomimetic agents. These compounds are easy to manufacture at low cost. They are solids but can be disseminated as an aerosol. One representative of this group of compounds, BZ, was weaponized, albeit only in small quantities. BZ, however, is not regarded as a completely satisfactory chemical warfare agent. As is the case for all psychochemical agents, its effects on groups of people under combat conditions are difficult to predict, which is something the military commanders prefer to avoid. An interesting anecdote was related by Major General John Appel, former commander of the US NBC defense forces. When an accident took place in a workshop for filling shells with BZ, an aerosol cloud of BZ drifted through the camp. Despite a thorough medical examination, nobody appeared to be affected except for the commander, who had a lot of explaining to do at the Pentagon.

#### 4.4.1 BZ

| ICt <sub>50</sub> : | 110 mg·min·m <sup><math>-3</math></sup> (inhalation of aerosol)   |
|---------------------|---|
| Rate of action:     | Symptoms appear half an hour after inhalation; maximum effects in $4-8$ h.  |
| Symptoms:           | BZ can enter the body as an aerosol by inhalation or via poisoned food<br>or drink. It can be absorbed through the skin but shows effects after a<br>considerably longer onset time. After intoxication, peripheral |

symptoms appear first: parched nose, mouth, and throat, and dry, flushed skin. The victim may vomit, his head may ache, and his body temperature may rise, followed by blurred vision, dizziness, and disorientation. Coordinating various muscular movements becomes difficult, and visual and auditory hallucinations will occur. Memory may fade. Frequently, the victim suffers from great motor anxiety. The effects may last for 4 days and the return to normal proceeds only gradually.

# 4.5. Irritating agents

The ratio between the lethal dose and the effective dose of irritating agents is high, as for most incapacitating agents. Unlike the latter agents, however, irritating agents induce immediate effects upon exposure, and the effects disappear relatively rapidly after cessation of exposure. Two groups of irritating agent will be discussed: tear gases and vomiting agents. Tear gases are included in the police arsenals of many countries as riot control agents.

Irritating agents are not incorporated into the Schedules of Chemicals contained in the CWC.

# 4.5.1 Tear gases (lachrymators)

In addition to their use as riot control agents, tear gases are well-known from their use in the Vietnam War. They are also used as training agents. Tear gases cause instant eye pain, the flow of tears, eyelid cramps, and skin irritation. Some may also affect the respiratory system. When released indoors, they can cause serious illness or death.

4.5.1.1. Chloracetophenone (CN)

The solid agent is mainly disseminated as an aerosol. Although its volatility is relatively low, its efficacy is high enough for dissemination of an effective concentration. In addition to its lachrymatory effects, the agent is an irritant to the respiratory system.

| LCt <sub>50</sub> : | $7,000-14,000 \text{ mg}\cdot\text{min}\cdot\text{m}^{-3}$  |  |  |  |
|---------------------|---|--|--|--|
| ICt <sub>50</sub> : | $80 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$  |  |  |  |
| Rate of action:     | almost instantaneous  |  |  |  |
| Symptoms:           | Powerful lachrymatory effects and irritation of the respiratory system.   |  |  |  |
|                     | In higher concentrations, it is irritating to the skin, causing a burning<br>and itching sensation. High concentrations may cause blisters that are |  |  |  |
|                     | harmless and disappear within a few hours. The agent may cause  |  |  |  |
|                     | nausea.   |  |  |  |

#### 4.5.1.2 Bromobenzyl cyanide (CA)

| LCt <sub>50</sub> : | $8,000-11,000 \text{ mg}\cdot\text{min}\cdot\text{m}^{-3}$             |
|---------------------|--|
| ICt <sub>50</sub> : | $30 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$                   |
| Rate of action:     | instantaneous  |
| Symptoms:           | Irritation and lacrimation of the eyes with acute pain in the forehead |
|                     | and burning sensation of the mucous membranes.                         |

# 4.5.1.3 O-Chlorobenzylidenemalononitrile (CS)

CS is a water-insoluble white crystalline solid. It is disseminated either as a solution spray, as a cloud of dust or powder, or as an aerosol. The formulation known as CS1 comprises micronized CS powder mixed with 5% silica aerogel to reduce agglomeration. It remains active for up to 5 days when dusted on the ground. The formulation CS2 is CS containing the silicone water-repellent Cab-O-sil, which reduces both agglomeration and hydrolysis. CS2 may be persistent for as long as 45 days.

| LCt <sub>50</sub>   | $61,000 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$   |
|---------------------|--|
| ICt <sub>50</sub> : | $10-20 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ for respiratory effects  |
|                     | $1-5 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$ for eye effects  |
| Rate of action:     | very rapid   |
| Symptoms:           | Burning of eyes accompanied by copious flow of tears, coughing, difficulty in breathing, and stinging sensation in moist skin, runny |
|                     | nose, and dizziness. High concentrations may also cause nausea and   |
|                     | vomiting.  |

#### 4.5.2. Vomiting agents

This group of irritating agents causes coughing and vomiting in addition to irritation of mucous membranes. The agents are also called sternutators.

#### 4.5.2.1 Diphenylchloroarsine

Diphenylchloroarsine was used in the First World War and is also known as Clark I.<sup>\*</sup> \*Ernst Hemmingway, a Farewell to Arms.

| LCt <sub>50</sub> : | $15,000 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$                                      |
|---------------------|---|
| ICt <sub>50</sub> : | 12 mg $\cdot$ min $\cdot$ m <sup>-3</sup> (10 min exposure), but depends on the exposure time |
| Rate of action:     | very rapid  |
| Symptoms:           | Irritation of eyes and mucous membranes, sneezing, coughing,                                  |
|                     | headache, acute pain and tightness in the chest, nausea, and vomiting.                        |
|                     | The effects last for approximately 30 min after exposure to moderate                          |
|                     | concentrations and up to several hours at higher concentrations.                              |

#### 4.5.2.2. Diphenylcyanoarsine

The agent was used in the First World War and is known as Clark II.

| LCt <sub>50</sub> : | $10,000 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$                              |
|---------------------|---|
| ICt <sub>50</sub> : | 20 mg·min·m <sup><math>-3</math></sup> (5 min exposure); depends on the exposure time |
| Rate of action:     | rapid   |
| Symptoms:           | See diphenylchloroarsine. Diphenylcyanoarsine is more toxic than                      |
|                     | diphenylchloroarsine.   |

# 4.5.2.3. Adamsite

Adamsite was stockpiled during the Second World War. Its volatility is very low; hence, intoxication by its vapor is not possible. It can effectively be disseminated as an aerosol.

| LCt <sub>50</sub> : | $15,000 \text{ mg} \cdot \text{min} \cdot \text{m}^{-3}$                                 |
|---------------------|--|
| ICt <sub>50</sub> : | 22 mg $\cdot$ min $\cdot$ m <sup>-3</sup> (1 min exposure); depends on the exposure time |
| Rate of action:     | rapid  |
| Symptoms:           | Similar to effects of diphenylchloroarsine; the effects develop more                     |
|                     | slowly.  |

# 4.6. Herbicides

2, 4-D and 2, 4, 5-T, (di- and trichlorophenoxyacetic acid)

Technical preparations of 2, 4, 5-T may contain small quantities of the highly toxic 2, 3, 6, 7-tetrachlorodibenzo-p-dioxin, also known as dioxin. Dioxin is very toxic and has carcinogenic and teratogenic properties.

Like 2, 4-D, 2, 4, 5-T can be used as a salt or as an ester. The effects of 2, 4, 5-T on plants are similar to those of 2, 4-D. On certain plants, 2, 4, 5-T is more effective.

# **4.7. NATO effect levels for percutaneous exposure**

# 4.7.1. Percutaneous toxicity

The literature describing the percutaneous toxicity of mustard and nerve agents occasionally reveals conflicting data. As an example, exposure times vary from 1 min to 30 min for mustard agent on bare skin of the volar forearm, and the range of dosages that produce erythema are reported to fall between 3000 mg·min/m<sup>3</sup> for very short exposures to less than 100 mg·min/m<sup>3</sup> for approximately 10 min exposures, and in the 200–500 mg·min/m<sup>3</sup> range for approximately 30 min exposures. Explanations for this variability have been sought in the biological variations among test subjects (e.g., Caucasian or Negro), temperature or humidity during exposure, purity of the agent used during testing (laboratory or military grade), and clothing layers, e.g., summer uniform, foul weather dress, etc.

In addition, some confusion surrounds the apparent applicability of the toxic load concept to respiratory exposures but not to percutaneous exposure. One would expect that if for respiratory exposure the toxic load effect were due to detoxification by the human body, a similar process would function during percutaneous exposure.

The source of confusion may be that the kinetics of percutaneous exposure processes has often been overlooked. When vapor approaches the skin the first step in the kinetics is diffusion through a stagnant layer of air on the skin surface. The next step is absorption by the skin. The kinetics of these processes is controlled by the concentration gradient. The following step is penetration through the barrier formed by the skin. Analogous with heat transfer, four hypothetical skin layers are involved, forming two resistances and two capacities. Once the agent has passed these layers, it can be taken up by the blood stream The agents involved are all aggressive chemicals, and once they begin to penetrate a barrier (resistance) they may well open up the route for following agent molecules. Therefore, over the course of exposure, the penetration rate of absorption may increase. This would at least partly explain the absence of a toxic load effect during percutaneous exposure

Another effect of the two barriers (resistances) in the skin is that as an agent saturates the capacity of the surface of the upper layer of the skin it begins to penetrate this first barrier. For short exposures, if the source of vapor is removed the agent can diffuse along one of two pathways, one that penetrates the skin further and the other that penetrates the outside and thereby no longer contributes to the possible effect. The ratio between the barrier heights to the bloodstream and to the outside will determine how much agent is "lost" in this process. These kinetic processes may explain why very brief exposures require a far higher dosage to cause erythema than moderate or longer exposures. Assuming that the kinetics correctly represents the true processes, the kinetics also determines the toxic load effect. Such an effect would be observed for longer exposures > 10 minutes. Unfortunately, most exposures are in the 10–30 min range and it is very difficult to conclude whether a toxic load effect is present. To be on the safe side, it is, therefore, better to assume that the toxic load effect does not exist for percutaneous exposures.

As it is highly unlikely that additional controlled exposures of human volunteers to mustard or nerve agents will be executed in the future, the current values will be used to derive the protection factors required for skin protection. It may now be apparent that toxicity values and certainly percutaneous toxicity values are seldom definitive. Extreme care should be taken in handling those numbers for engineering purposes.

# 4.7.2 Recommended human toxicity estimates for percutaneous toxicity

Recommended human toxicity estimates for percutaneous exposure have been reviewed by a NATO group of experts. These values are based on data presented in a review made by US toxicologists of all available data. (See, for example, OPCW, Emergency Assistance Division, and Course for Creating Awareness Regarding Chemical Defense. Draft, The Hague, the Netherlands 2005.)

Exposure times are relatively short: several minutes up to ca.1 h. In the underlying reports and publications, insufficient data can be found that percutaneous exposure to these agents is accompanied by a toxic load exponent that differs from 1. This observation was independently made by NATO LTSS-024 in 2002. This implies that the acceptable dose does not increase with the exposure time.

The effect levels for vapor exposure are all in  $mg \cdot min \cdot m^{-3}$ . Those for aerosol exposures are derived from the amount deposited and the values given for liquid exposure. It should be remarked that HD is locally acting and sometimes an effect level per cm<sup>2</sup> is given. In addition, sensitivity varies widely over the human body, with the scrotum and armpit being the most sensitive areas.

Table 4.1. Percutaneous toxicity values

| Agent            | Effect                            | Vapor                | Liquid                |
|------------------|-----------------------------------|----------------------|-----------------------|
|                  |                                   | $(mg \cdot min/m^3)$ | (mg per man)          |
| Tabun (GA)       | negligible                        | 3000                 | No data               |
|                  | significant decrement in military | 7500                 | No data               |
|                  | performance                       |                      |                       |
|                  | danger of death                   | 12500                | No data               |
| Sarin (GB)       | negligible                        | 2000                 | 300                   |
|                  | significant decrement in military | 3000                 | 400                   |
|                  | performance                       |                      |                       |
|                  | danger of death                   | 8000                 | 500                   |
| Cyclohexyl Sarin | negligible                        | 350                  | 15                    |
| (GF)             | significant decrement in military | 750 <sup>a</sup>     | 30                    |
|                  | performance                       |                      |                       |
|                  | danger of death                   | 2000 <sup>a</sup>    | No data               |
| Soman (GD)       | negligible                        | 350                  | No data               |
|                  | significant decrement in military | 750 <sup>b</sup>     | No data               |
|                  | performance                       |                      |                       |
|                  | danger of death                   | 2000 <sup>b</sup>    | No data               |
| VX               | negligible                        | 30                   | $1.75 (0.05^{\circ})$ |
|                  | significant decrement in military | 60                   | $2.45 (0.14^{\circ})$ |
|                  | performance                       |                      |                       |
|                  | danger of death                   | 200                  | $2.80 (0.35^{\circ})$ |
| sulfur mustard   | negligible                        | 50                   | 200                   |
| (HD)             |                                   |                      | $(0.001)^{d}$         |
|                  | significant decrement in military | 200                  | 800                   |
|                  | performance                       |                      |                       |
|                  | danger of death                   | 1000                 | > 1000                |
| Lewisite (L)     | negligible                        | No data              | No data               |
|                  | significant decrement in military | 400-500 <sup>d</sup> | $0.005^{d}$           |
|                  | performance                       | (500 <sup>e</sup> )  | $(0.030^{\rm e})$     |
|                  | danger of death                   | 33000                | 930                   |

<sup>a</sup> Based on the potency ratio of GF to GB. <sup>b</sup> Based on the potency ratio of GD to GB. <sup>c</sup> When applied onto the skin of the cheek.

<sup>d</sup> In mg/cm<sup>2</sup>. Erythema for H (derived from vapor exposure) and vesication of the skin for L. <sup>e</sup> Serious corneal damage.

\*There seems to be a printing error in the NATO table, 800 mg per man exposed to one side corresponds to > 80  $\mu$ g per cm<sup>2</sup>. The values for Lewisite (5 $\mu$ g) in the same table are very much lower. It is generally understood that H and L are equally potent skin agents.

# INFECTIVE OR TOXIC DOSAGES FOR SELECTED BIOLOGICAL WARFARE AGENTS

#### 4.8. Viruses

Variola major virus (smallpox virus); the disease is highly contagious. The infective dosage is low, on the order of 10–100 organisms. Lethality is relatively high.

Venezuelan equine encephalitis virus, infective dosage about 10-100 organisms. The transmission between humans is low. Incubation period 2-6 days.

Viral hemorrhagic fevers (several forms like Ebola, Marburg, Junín, Machupo, Sabia, Flexal, Guanarito); infective dosage 1–10 organisms. The lethality is high to moderate, depending on the species. Transmission between humans is moderate.

#### 4.9. Bacteria

*Bacillus anthracis*, around 55,000 organisms present a  $LD_{50}$ ; the probit slope is very low with small numbers down to 1 organism being a lethal dosage. The disease is not contagious.

*Brucella abortus, Brucella melitensis, Brucella suis*+, infectious dosage as an aerosol 10–100 organisms. Incubation period of 1–2 month makes this an unlikely biological warfare agent. The diseases are not contagious.

*Burkholderia (Pseudomonas) mallei Glanders;* the infective dosage is assumed to be low. The transmission from human to human is also quite low.

*Burkholderia (Pseudomonas) pseudomallei Melioidosis,* Similar to Glanders, although the incubation period varies much more, from 1 day to even years.

*Francisella tularensis*\* 10 to 50 organisms form an infectious dosage. Although the incubation period can vary from 1–21 days it usually is 3–6 days.

*Yersinia pestis,* infective dosage 100–500 Incubation period is usually 2–3 days. Lethality is high.

### 4.10. Rickettsiae:

*Coxiella burnetii;* causes Q fever which seldom is transmitted from humans to humans. Only 1-10 organisms are required for an infective dosage. Incubation period, however, varies widely from 1-6 weeks. The lethality is very low.

# **5.** Detection

# **5.1. Introduction**:

In the area of detection, specific terminology is used that often leads to confusion. Therefore, the definitions of the terms used in this book are presented first.

# Definitions for detection:

**Alarm** – The visible and/or audible warning of the presence or impending presence of a chemical or biological agent.

**Detection** – The event by which the presence of a chemical or biological agent is established.

False Alarm Rate – The percentage of time that the detector produces a false alarm.

Hazard – The presence of an identifiable risk.

**Identification** – The unequivocal assignment of a specific chemical structure or biological identity to an agent.

Monitoring – The continued or periodic process of determining whether an agent is present.

**Point Detector** – A detector that samples the environment at its own location.

**Reconnaissance and Survey** – The identification of the presence, change, or absence of chemical agents or agents of biological origin, in an area, or on a route prior to occupation or use by troops.

**Remote Detector** – This refers to a *point detector* located some distance upwind of a target and designed to give that target advance (early) warning of attack.

**Selectivity/Specificity** – The ability of the detector to respond to the agents of interest and not respond to the background or non-hazardous agents.

**Sensitivity**- The ability of the equipment to detect sufficiently low levels of agent down to the threshold level for effects.

**Stand-off Detector** – A detector that interrogates the environment at some distance from its physical location.

**Warning** – The indication of the presence of chemical or biological warfare agents at a concentration that will cause casualties if the appropriate action is not taken.

# 5.1.1. CB Hazards Are Special

Detecting that something hazardous is in the air is the first step in taking evasive or protective actions. Almost all types of hazards are self evident, e.g., earthquakes, storms, floods, etc. However, chemical and, in particular, biological hazards are silent and in most cases do not have a signature until it is too late and the damage is done. For the chemical warfare agents used in WW I, timely detection was possible from the telltale odor of the agents. A "trained

nose" could detect those agents before they were present in hazardous concentrations. To develop a more rapid detection method, soldiers used white mice and canaries to detect the arrival of hazardous agents. With the development of the more potent nerve agents, which are very hazardous at concentrations that cannot be detected by smell, more advanced forms of detection became essential. Biological agents have no signature at all and must be detected by special means.

In the event of a chemical or biological (CB) incident, casualties will likely occur. The goal of detection is to operate systems that are capable of detecting and identifying all forms of CBW attack or hazard so as to minimize the effects on the health of individuals or the operational tempo of military forces. CB detection, used with monitoring and identification systems, can:

- Enable evasive actions or hazard avoidance wherever possible;
- Enable adoption of a protective posture;
- Guide medical countermeasures and treatment;
- Provide warning and reporting data for downwind hazard areas;
- Guide sampling and identification of biological and chemical agents, and;
- Act as part of the web of deterrence via the force protection it provides and reducing casualty numbers to less significant values.

Detection used to be directed toward chemical and biological warfare agents in military scenarios. At present, detection includes terrorist use scenarios as well, directed toward military or civilians. Detection also includes releases other than attacks, e.g., from industrial storage or transport tanks.

### 5.1.2. Important role for detection

In their report, experts of the WHO assigned a crucial role to detection in the response to a chemical or biological incident. The standard risk management steps in the response to a chemical or biological incident are:

- Identify the hazards (warning, detection);
- Evaluate the hazards to determine initial risks (detection and identification);
- Introduce risk reduction strategies (locate contaminated zones, monitor the hazard);
- Quantify residual risk and make a risk acceptance decision (detect residual contamination);
- Monitor the risk management program and repeat the process as required.

#### 5.1.3. Detection process

Detection is the process through which evidence is obtained regarding the types and quantities of chemical or biological agents in the environment. Basically, detection is carried out using a sensor that produces a signal that is observed by eye or by electronic means. Schematically, it consists of:

Sensor----- (Amplifier) -----Reader.

# 5.1.4. Various types of detection

Detection is of vital importance in reducing the risks and consequences of a chemical or biological agent incident, whether it is an act of war or terrorist activity. Detection determines the protective actions required, and it indicates to physicians which types of therapy should be applied. Therefore, detection may be needed for several different purposes:

- Alarm/Warning of an incoming attack/all clear using point, stand-off or remote detectors;
- Downwind hazard message;
- Confirmation of the attack;
- Reconnaissance and survey;
- Monitoring of the hazard;
- Sampling;
- Identification, e.g., to enact medical countermeasures;
- Verification;
- Identification of contaminated areas and the need for decontamination.

Detection, identification, and monitoring are closely interrelated processes by which a CB incident is detected, the agent identified, and the actual extent of contamination is delineated. Detection is an event sufficient to warn of a CB incident and allow adoption of evasive or protective measures. Detection should be sufficiently reliable to issue an alarm and a warning message. It should also be sufficiently reliable to ensure that the false negative rate, the probability of missing an incident, is acceptable for the operational purposes in military circumstances. Ideally, one would like to see a 100% detection of non-war incidents. The wearing of protective equipment is cumbersome and constrains military operability, so the determination that a hazard no longer exists is of equal importance. In the civilian sector, the proper functioning of protective measures (sealed rooms inside houses) relies on the "all clear" signal (opening up after the cloud has passed).

Identification is the process of determining the specific agents that were detected, and is necessary for ensuring that appropriate countermeasures are taken. Identification can take more time than detection.

The process of detection and, through that, hazard avoidance must be considered as a whole system. Each element above contributes to the other, constructing a concept of "cooperative" detection in which each element is critical to providing complete protection.

Different forms of detection require different methods and types of equipment. Warning is most important for the reduction of casualties because protective measures can be taken after a rapid warning.

# **5.2.** Characteristics of detectors

# 5.2.1. False negatives and positives

A sensor produces a signal, for instance, a color change in a chemical reaction that can be observed by the human eye. Essential for the sensor is that it always produces a signal when a hazardous compound is present and that it does not react to non-hazardous compounds, ideally producing neither false negative nor false positives signals. Given that perfect sensors do not exist, they should be sufficiently reliable to ensure that the false negative rate, the probability of missing an attack or incident, is acceptable for the scenarios under review.

Early sensors were based on chemical reactions. These reactions were highly selective, and false positives or negatives were greatly suppressed. However, the reaction worked only for

one type of agent and, in some cases, only for a particular agent. A complex battery of detection reactions was required to address all types of possibly hazardous agents. Later, detectors based on physical interactions were developed. Physical interactions are, by definition, general and non-specific in nature, and they will respond to any type of agent, including non-hazardous compounds. Both false positives and negatives may occur frequently. Ways to improve the signal to noise ratio for hazardous agents were sought, for example, through discrimination of the mass of the agents as in the case of the Chemical Agent Monitor (CAM) or mass spectrometer.



The CAM is a preferred agent detector based on ion mobility spectrometry. Once correctly calibrated, it can detect all types of agents. The current configurations are mainly optimized for nerve and blister agent detection, but as long as an agent can be ionized it can be detected by the CAM.

During inspection, communications may become complicated, and two CAMs and a small computer are required to record the findings.

The last decade has seen several major advances in CB detection and identification. Ion mobility spectrometry (IMS) has continued to be an important method for monitoring and, increasingly, for detection of CW agents. Alternative technologies, such as surface acoustic wave devices, flame photometric detection, and mass spectrometry (MS), have been developed for the detection of toxic industrial chemicals and CW agents. Further improvements to these devices are rapidly evolving with the advent of micro-electromechanical systems (MEMS). Composite systems that employ more than one detector technology are being examined as miniaturizes devices to make traditional laboratory technologies available for field deployment.

#### 5.2.2. Reversibility

Another required feature in detectors is reversibility, meaning that the signal should cease when the agent is no longer present. The chemical reactions used in detectors are generally designed for one-time use only. Each time a reaction is carried out, new reagents are required. Detectors based on chemical reactions are, therefore, non-reversible and will not automatically indicate that a hazard is no longer present. Detectors based on physical interactions all require that the agent be adsorbed onto the detector. The stronger the interaction, the stronger the signal, but also the more difficult it is to desorb the agent and to free the surface of the sensor for the next interaction. A balance must be struck between the
strength of adsorption and the signal such that reversibility is maintained, but the more common approach is to repeat the detection interaction frequently in intervals.

# 5.2.3. Amplifier

An amplifier is sometimes required to amplify the signal or to improve the signal to noise ratio.

## 5.2.4. Reader

The reader can be anything from the human eye to the most advanced electronic detectors, as used in mass spectrometers. The reader usually incorporates an instrument to analyze the signal. For the human eye, the brain accomplishes this task but for most physically-based detectors, this can be a very complex instrument that is difficult to use in the field and requires special skills.

## 5.2.5. Response times

Chemical Warfare agents can react very quickly with the human system and, therefore, quick detector response times are required. It is sometimes difficult to find the right compromise between response time, sensitivity, and selectivity.

## 5.2.6. Biological agents

Current biological agent detectors are almost exclusively based on biochemical reactions. Sampling of the agents, reaction with a biochemical, and development of the reaction all take time. As a result, biological agent detectors are relatively slow. Current advanced biological agent detectors have a response time of half an hour. Because of the large variety in biological agents, many different detection reactions are required, and usually full suites of reactions are combined in a single detector. Most emphasis in future work is on developing physical detectors for biological warfare agents. The first generation will be fielded in the near future. Until that time, the detection of a biological attack will be mainly through diagnosis of patients, in some cases many hours to days after the incident has taken place.

As a result of the Gulf War, the requirement for biological detection systems has been heavily emphasized. Major research and development programs among the nations involved have produced first- and second-generation biological detection and identification systems, including point, remote detectors, and stand-off biological detector systems.

BW agent point detection systems are now capable of continuous monitoring in battlefield environments. Fluorescence-based point detection technologies can discriminate between biological and non-biological aerosols in the atmosphere. Analytical methods for the identification of BW agents have been developed and automated, including aerosol sampling and collection. These fielded technologies remain principally wet techniques, such as immunological methods. Specialized reagents for a specific list of priority BW agents have been developed.



The enigma detection kit is one of the most advanced kits for detecting biological agents. However, it requires 20–30 minutes to produce a detection result. The military requires a much shorter response time, on the order of seconds, to take appropriate protective measures. Development of rapid response laser aerosol detectors combined with time of flight mass spectrometers seems to produce an ideal instrument. However, such an instrument may be too expensive for wide distribution among troops.

5.2.7. The detection identification continuum

Detection and identification represent a continuum. It must be recognized that continual improvements in the specificity of detectors will provide information equivalent to identification of a CB agent. Ongoing developments in identification methods that increase the speed and processing time can lead to identification systems that are sufficiently rapid to provide detection, alarm, and warning. Technological advances continue to narrow the gap between detection and identification.

5.2.8. Detection limit requirements as they relate to agent types

Detection limits and sensitivity can be different for different detection, identification, or monitoring equipment. The selection of a threshold level for a detection system is based on the human toxicity of the agent, the level of protection afforded by the personal protective equipment or the collective protection, and the risk one is willing to accept. Increased awareness of health effects from low-level exposures and the advent of occupational health considerations in operations other than war, as well as the no-risk concept for the civilian population, continue to drive sensitivity limits lower. It is vital to recognize that not all systems must have equal sensitivities. A combination of detectors can be as useful in providing the information necessary for detection, monitoring, or other applications.

As an example of the process leading to the NATO detector specifications contained in D/100 (a general document describing all aspects of detection, NATO Unclassified), the following excerpt is provided:

A trained person, properly equipped, standing alongside the instrument, and carrying out the correct drills in a timely fashion in reaction to its alarm shall not be exposed to more than:

| <u>Chemicals</u> :           | 1 ICt <sub>5</sub> (inhalation) accumulated over 4 hrs |
|------------------------------|--|
| <u>Biologicals, Toxins</u> : | 1 ED <sub>5</sub>                                      |

## **5.3. CBW detection and identification**

Situations involving CW detection are different from those involving TICs and are very different from those involving biological and toxin detection. Some remarks about the three situations are provided, with emphasis on chemical agent detection. Some of the instruments in use have been previously mentioned:

#### 5.3.1. Chemical agent detection

Chemical warfare agents, including vesicants and nerve agents (see Chapter 3), are a relatively diverse group of compounds. They are used as vapors or thickened liquids. Some nerve agents can be dispersed as aerosols.

Due to the diversity of the agents, there always will be several monitors and detectors involved to address the range of potential agents and the range of the required sensitivity. For example, a rapid response detector can be used on a large scale to provide primarily immediate warnings, and a high sensitivity detector can be used to determine the residual hazard level and for monitoring contamination levels.

#### 5.3.2. Alarm/warning/all-clear detection

The simplest form of alarm detection is the visual observation of a cloud of unusual color drifting with the wind at an unusual height as well as the functioning of delivery weapons or smoke producing devices. Next to be encountered might be unusual or telltale odors associated with agents that, in the case of nerve agents, may signal that it is too late to prevent exposure. Other observations include personnel with symptoms or sick and dead animals. Instruments have been developed with very short response times to warn of vapor and aerosol attacks. Some of these devices operate continuously and are often based on wet chemistry with enzymes. Replacing the enzymes and batteries must occur frequently. The enzyme systems are highly specific and respond to one type of agent only, mostly the nerve agents. It should be noted that many of the instrumental techniques, sometimes unrelated to CBW, that have been developed over the past 20 years also could be used in a CBW alarm mode. Passive infrared (PIR) and laser techniques (RSCAAL) have been developed that offer a remote detection capability.

A low-tech approach to warning against liquid attacks is the use of liquid detection paper that changes colors when in contact with a liquid agent. Some of these are reactive and can be made agent-specific.

An alarm should always result in a warning and a downwind hazard message to warn the personnel downwind from the attack. This includes the prediction of the downwind hazard area. Most models used for the calculation of the hazard contours from an agent release are deterministic in nature. They are based on averaged atmospheric diffusion parameters and calculate the average of 1000 or more releases to generate the well-known cigar-shaped plumes. The hazard contour of an individual release can be very different from this average. NATO has recognized this and uses template, the shapes of which depend on the wind speed.

These contours cover a much wider area than the average cigar shapes. The ideal would be to have a probabilistic model that indicates the probability of being inside or outside a hazard contour. These models require substantial computer power but are under development and will become available in the near future. Of equal importance is to provide an all-clear signal. For the military, this indicates that wearing of the cumbersome mask may be discontinued. For civilians, this signal is even more essential. After this notification, rooms should be opened up to flush out any agent that may have penetrated the protective barriers. The all clear signal can only be given by detectors that run continuously. Older types of detector are typically based on wet enzyme chemistry, and newer ones are based on physical techniques.

## 5.3.3. Confirmation/verification/identification detection

After the alarm has sounded, it is important to confirm the attack (false positive alarms are possible) to verify the presence of an agent and, for medical purposes, to identify the agent that has been used. Some forms of confirmation detection are based on manual detection methods that include the use of detector paper to locate and define liquid contaminated areas, detection tubes similar to those used in HAZMAT incidents, and special detection tickets developed for nerve agents. Some of these manual methods are highly specific and work against one type of agent only.

Many of the instrumental techniques can be used to confirm an attack or to identify the agent. Gas chromatography (GC) and mass spectrometry (MS) are two techniques that have been introduced. One of the most successful techniques has been the ion mobility spectrometer (IMS). Together with the reproducible generation of agent signals and extensive computational algorithms capable of processing the signals, this has become the instrument of choice for detecting nerve and blister agents. These two agents are the two most important agent groups that are hazardous below the threshold sensitivity level of the olfactory system. GC and IMS systems were initially developed as monitors to monitor the development of the hazard over time. Although they still have that function, many other applications, from alarm to all-clear and confirmation detection, have evolved. The latest versions include the capability of detecting TICs.



The Chemical Agent Monitor is a device, the function of which lies between a Geiger counter and a nose for smells. The form and characteristics of the detector facilitate use and adoption of the technology.



# 5.3.4. Toxic industrial chemical (TIC) detection

The recognition that TICs can be used as chemical warfare agents has increased the emphasis on chemical detection capabilities with a greater dynamic range for sample type (solid, liquid, aerosol, and vapor) and a larger set of reference data for identification. In cases of TIC incidents, one is always quite certain about the type of TIC that has been used. The locations where TICS are produced and stored are well-known, and only in the case of a hijacked transport vehicle might some uncertainty exist. Present day first responders, typically the fire brigades, have the capability to respond to HAZMAT incidents. The detectors used in HAZMAT responses are also very applicable to cases of TIC release. One common piece of equipment employed is the detector tube, in which an agent is collected in a sampling tube containing reagents. Usually a color changes indicates the presence of an agent.

#### 5.3.5. Toxins and other mid-spectrum agents

These comprise a variety of agents, from natural and manmade toxins, to bioregulators that can be used for a variety of medical purposes, to designer drugs. They are active in very small quantities and, therefore, require very sensitive, highly selective, specific detection methods. At present, detection emphasis centers on GC and MS techniques

Biological warfare agents represent a significant challenge for detection and identification systems. BW agents generally present a hazard as aerosols disseminated in the respirable range  $(1-10 \ \mu\text{m})$ . This form of incident is difficult if not impossible to detect. Because of their high infectivity (e.g. *Brucella* sp. are infectious in the 10–100 organism range), very low aerosol concentrations can be hazardous, necessitating systems with very low limits of detection. For some agents (e.g., anthrax) the probability curve for developing effects from a certain dosage has a very low slope, resulting in significant numbers of victims at dosages

tens or even hundreds of times below the  $LD_{50}$  level. For most CW agents, the slope is such that a dosage ten times below the  $LD_{50}$  produces hardly any effect.

Indicators for a biological incident that could result in a warning are varied and include:

- Intelligence information
- Observations by individuals of dusty clouds, special equipment, canisters, etc.
- Sudden outbreaks of unusual diseases; in particular, the time phased development of the disease may be noticeably different from that of a normal epidemic.

To achieve the necessary detection sensitivities, biological detection systems developed and demonstrated to date have utilized aerosol concentrators to increase sensitivity of both the detection and the identification modules at the cost of response time. They use the following technologies:

- Immunoassays
- Bioassays
- Genetic analysis, PCR techniques
- Spectrometric techniques
- Chromatography

The description of all techniques and their modifications used in bio-detection is beyond the scope of this discussion, therefore only a short treatment will be will be presented.

#### 5.3.6.1. Immunoassays

When an unwanted organism (pathogen) enters the body, the immune system will respond. The attacker is identified, absorbed by white blood cells and highly specific warrior cells, and antibodies are produced. For detection purposes, appropriate antibodies may be produced in the laboratory and marked (tagged) with a compound that can easily be detected. Tags are of various types: colored, chemiluminescent, bioluminescent, fluorescent, or radioactive. A technique that detects fluorescence in an immunoassay that uses enzymes is called an enzyme-linked Immuno sorbent assay (ELISA). These techniques are all extremely specific. The antibodies recognize only a single site on the pathogen. The large variety in pathogens and the variations, including genetic modifications within one type of pathogen, increases the number of required immunoassays.

5.3.6.2. Bioassays

Bioassays rely on the multiplication of pathogens in a certain medium. Once their quantity has risen to sufficient levels, determination of the type of pathogen can be attempted. Viruses can be detected only through their potential to kill other pathogens, e.g., the plague. A special group of biosensors employs receptor molecules immobilized on the surface of a sensor or chip to detect the binding of pathogens. The techniques involved are not without danger because live pathogens are cultured and used. This technique is also very time-consuming and only provides qualitative answers. Of particular importance is the chain of custody, from the sampling and transport of the samples to the analytical laboratory.

## 5.3.6.3. Genetic analysis and PCR

Every living creature, from humans to pathogens, contains hereditary material known as DNA. The building blocks of DNA are four types of chemical bases, A, T, C, and G that are ordered within a DNA molecule to form an enormous number of individual-specific and/or species-specific linear sequences. Each pathogen has a unique combination of these building blocks that can be used for identification purposes. The polymerase chain reaction (PCR) technique is used to amplify small quantities of DNA to facilitate the identification of the pathogen. Restriction fragment length polymorphisms (RFLP) are organism-specific variations in the DNA sequence that are recognized by cutting the DNA into fragments using sequence-specific restriction enzymes. The fragments are separated and analyzed by gel electrophoresis (a form of size chromatography) to yield an array of organism-specific bands (stripes). The bands characterize the pathogen type can be detected, e.g., to indicate whether the pathogen was slightly modified in a laboratory. The technique is labor-intensive and time-consuming.

## 5.3.6.4. Spectrometric techniques

Spectrometric techniques include radiation spectrometry, such as nuclear magnetic resonance (NMR) and Fourier transform infrared spectroscopy (FTIR). Both are techniques that can only be applied in highly sophisticated laboratories. Mass spectrometry is also applied in highly sophisticated laboratories, but for chemical agents detection, reliable field instruments are being developed, such as the German "Spurfuchs". Another particularly promising development is the miniature matrix-assisted laser desorption ionization time-of-flight mass spectrometer (MALDI-TOF MS). MALDI partially breaks apart the pathogens into charged fragments, which are then separated and detected by the TOF MS, as described below.



The Fox, which originates from Germany and carries the name "Spurfuchs", is armored an vehicle that is completely equipped to detect Chemical agents. The second generation can do wonders with TICs and is relatively easy to calibrate for new agents like Novichoks. The heart of the vehicle contains a mass spectrometer. As rapid response bio warning instruments become available, they can easily be incorporated into the Fox as well.

A technique developed for military purposes is the use of high-powered tunable lasers to detect scattered light from BW clouds at a distance, a stand-off detection capability.

There is a need for technologies that improve the initial detection of bio-aerosol events and a need to reliably differentiate between naturally occurring biological events and true BW agent attacks. This will require research to understand the signature of BW weapons systems and attacks, understand natural biological background phenomena, and the characteristics/impact of novel agents. The very promising MALDI-TOF MS technique is based on laser and MS technologies. A first laser detects the presence of particles for analysis. Detection triggers a second laser to split the particles into smaller molecular fragments. The fragments are analyzed using a TOF spectrometer. (A working laboratory model was presented during the Ninth Symposium on Protection against Chemical and Biological Warfare Agents, Gothenburg Sweden May, 2007.)

High quality detection and identification of BW agents requires that several parallel-stream analytical technologies be used to provide the information needed by authorities and medical personnel. No single test method is sufficient. Sample collection, processing, and the steps required of standard analytical techniques are still prominent problems in the operation of biological detectors, particularly for detectors that are liquid-based. The available systems now and in the near future are likely to be complex and very costly, limiting their wide distribution and use. Additionally, their relatively slow response time permits many people to be infected before a positive detection occurs and the appropriate alarm for evasive action is sounded. Although one should not put "all their eggs into one basket", it seems wise to rely on epidemiological information for the detection of biological incidents and work on therapeutically-based countermeasures that might also inhibit the infection in the initial stages.

# **6.** Physical Protection

# 6.1. Introduction

When personnel are challenged with chemical or biological agents, protection is mandatory. The first step in protection is detection, which produces an alarm and a downwind hazard warning. This is followed by the implementation of physical protections that aim to provide clean air or a clean atmosphere to the personnel involved. The environment in which personnel must operate may not be free of contamination. Decontamination or contamination control is a follow-up step in CB defense that deals with the "cleanup" of equipment, material, and terrain.

Air must be made free from vapors and particulates for the protection of the respiratory system and free from vapors for protection of the skin. Liquids from a spray attack are harmful to the skin, and protection from these drops is required. Physical protection involves the formation of a barrier between a toxic environment and an individual or a group of individuals.

Physical protection can be provided to the individual through a respirator and protective clothing or to a group of persons via collective protection. For both protective systems, the provision of clean air is essential. The respiratory system and eyes are the most sensitive for intoxication, followed by the skin. Another route of entry into the body is via ingestion of contaminated food or the drinking of contaminated water. These forms of contamination require special countermeasures that usually are not considered part of a chemical or biological defense system. The following subjects will be discussed:

- Removal of contaminants from air
- Respiratory protection
- Skin protection
- Collective protection

Physical protection places a heavy physiological load on the wearer. These physiological stresses are very noticeable to the respiratory system, such as the demanding task of exhaling against an overpressure. Heavy demanding workloads may result in insufficient breath to complete the task. Another load frequently encountered is thermal stress, resulting in increased perspiration usually associated with long-time slow heat built-up that depends on the work rate. It should be noted that both effects act on different human body control mechanisms. For one type of task, a mask could be the limiting factor, whereas for another type of task, the protective clothing, gloves, and boots may limit the endurance time. There are also minor limitations, such as reduced field of vision, loss of tactility, and restrictions to movement. The higher the level of protection, the higher will be the encumbrance. Unlimited protection, which is often requested, will result in the development of "space suits" or "Michelin man" systems in which it is very difficult if not impossible to move. Donning this these systems requires considerable time, and the rescue actions of first responders would be counterproductive. Therefore, individual physical protection always constitutes a compromise between the degree of protection and the physiological stress placed onto the wearer. One consequence of this compromise is that 24 hours of protection is futile if the system may be worn for only one hour before resulting in heatstroke casualties.

An important question is: How much protection should be provided and for how long? These questions are related to the efficiency (how much protection?) and capacity (how long?) of the

barrier between the human and the environment. Some examples will make this point clear. The first example discusses the efficiency and capacity of protective clothing. Suppose in an attack a Ct dosage of 2500 mg·min/m<sup>3</sup> of an agent is created, and the permissible exposure level to the skin is 25 mg $\cdot$ min/m<sup>3</sup>. In this case, the required protection efficiency for the skin is a factor 100. The airflow through the fabric due to the wind is 0.05 m/min, and the fabric is challenged with 125 mg/m<sup>2</sup> agent. This is less than 1% of the capacity of most suits. The protection efficiency is the critical factor in this case. However, when a suit is challenged with large liquid drops, the critical factor becomes the local capacity. The drop might be so large that over time, the capacity to adsorb vapor emanating from the drop may limit the protection time to a few hours. For the eyes and respiratory system, the permissible exposure level to vapor is usually lower (see Chapter 4), and 2.5 mg·min/m<sup>3</sup> will serve as a reasonable value for the example being discussed. This would indicate a required respiratory protection factor of 1000. In a recent Canadian study (1), it was argued that the worst-case feasible attack is a Ct of 5000 mg $\cdot$ min/m<sup>3</sup>. The protection factor of masks ranges from well above 10<sup>4</sup> for vapors to  $10^{6}$  for some aerosols. Consequently, an aggressor must use significant quantities of agent to reach his objective against a well-protected opponent. Because of the large quantities of agent required, except in very rare cases, the opponent may be much better served using weapon systems other than chemical or biological. The capacity of protective filters is usually very large. For this example, an attack on a person breathing 30 L/min would load the canister filter with 75 mg, which is less than 0.1% of the capacity. (The attack is expressed as a dosage C x t in mg·min/m<sup>3</sup>, the breathing rate is in m<sup>3</sup>/min, so dosage times breathing rate yields the load on the filter). A protected and trained army is unlikely to be defeated by a chemical or biological attack. However, the doctrine of use of the protective equipment should be adjusted to minimize the degradation in performance due to wearing the equipment, and forces should be well-trained to cope with the psychological impact of a CB attack.

# 6.2. Providing clean air

#### 6.2.1. Recirculation

The simplest form would be to put a plastic bag over the head and to run perpendicular to the wind outside the toxic cloud. Although the lungs will contain about 5 liters of clean air, the "protection "is very short-lived because one will suffocate within a few minutes. This should be considered only as a last resort if nothing else is available

#### 6.2.2. Recirculation with oxygen replenishment.

In some mask systems, the exhaled air is guided through a bed of chemicals in which carbon dioxide is absorbed and oxygen is simultaneously released, producing breathable air. These systems work well and have been in use as escape masks for miners and for soldiers passing in armored personnel carriers through waterways. The length of time that these systems are effective depends on the amount of chemicals, but is usually around 30 min.

#### 6.2.3. Air purification

Traditionally, air purification systems utilize filtration of particles and adsorption of vapors, usually combined in a single housing unit, to remove contaminants. In collective protection purification systems are incorporated into the environmental control system and pass air through high-efficiency particulate air (HEPA) and vapor filtration elements, in that order. The alternate filter order, i.e., vapor filter first, does not preclude the possibility of water in

the form of fog consuming the vapor filter and leaving no capacity for the toxic agents. In addition, liquid aerosols can penetrate the vapor filter, deposit onto the particle filter, and slowly escape via vaporization, which provides another argument against this alternate filter order. Every user would like to have a filter with an endless capacity, so one never must worry about replacement. Even if this were possible other limitations are in place. An aircraft designer is always looking for a low-weight small-volume filter system. An armored vehicle designer will aim at a small-volume easily-replaceable filter that may be incorporation into the existing air conditioning system. Finally, the most important part of the system, the filter canister for the individual mask, must be small in volume and weight with a low breathing resistance and must protect against TICS, CWs, and BWs of all sorts in concentrations and dosages that can be expected on the battlefield or in terrorist incidents.

#### 6.2.3.1. Aerosol filtration

Aerosol filters are made from non-woven structures of fine fibers. These fibers can consist of glass, organic, or polymeric materials. Filtration occurs by various mechanisms. Polymeric materials may be electrically charged to increase the filtration efficiency. Typically, HEPA filters remove at least 99.997% of 0.3–0.4  $\mu$ m particles, which are representative of the most penetrating particle sizes. The efficiency improves dramatically for both larger and smaller particle sizes. Adding filter depth can increase filtration efficiency, but this is at the cost of flow resistance and extra weight. Normally, a HEPA filter will be pleated to increase filter area and thus reduce flow resistance.

#### 6.2.3.2. Vapor filtration

Vapor removal relies on adsorption, either physisorption or chemisorption. Physisorption is a reliable mechanism for agents with boiling points above 100°C (e.g. nerve agents). Light vapors, such as HCN, cyanogen chloride, perfluoro compounds, several of the TICs, ammonia, and chlorine are not strongly retained by adsorbent media alone. There are two possible ways to address this problem; one is to remove the agents by a combination of adsorption and chemical reaction. The other is to invoke the concept of toxic load. Toxic load assumes that the dosage to create a certain effect is not a simple concentration-time product, as predicted by Habers' rule,  $C \ge t$  = constant. Rather, the product is not constant and is represented better by  $C^n x t$  =Constant. With n=1, this reduces to Habers' rule, but for most volatile compounds, n exceeds 1. For chlorine and HCN, the exponent n exceeds 3, whilst Sarin shows an exponent of 1.5. The consequence is that exposure to lower concentrations over a longer period of time is far less hazardous than exposure to the same dosage over a short period of time. The adsorbent in the canister reduces the peak concentration that normally would be received in a short time to a lower concentration over a longer time and, thus, reduces the hazard considerably. The concept of toxic load, in place of Habers' rule, is not well-incorporated in the assessment of the protection afforded by a mask. The toxicity of, for instance TICs, is seldom represented as a function of exposure time or concentration, For instance, NATO STANAG 2909 NBC commanders guidance on defense measures against TICs gives safe distances for TIC releases without incorporating the toxic load concept. Safe distances are, therefore, far too high and restrict the area of operations. It would be worthwhile to explore this concept further because it can form the basis for a significant contribution to the protection of the military against TICs using standard military canisters. Another feature of the toxic load concept is that it is practically the only principle that works for the protection of civilians inside closed rooms (see paragraph 6.5).

The adsorbent of choice is active carbon, composed of peat, coconut shells, or any other source with a relatively large quantity of aromatic hydrocarbons. By burning away hydrogen and some of the other elements, including carbon, and activating the product at high temperatures, an amorphous product results with many pores. The walls of the pores provide a large surface area for adsorption. Typically, an activated carbon has a surface area of over  $1000 \text{ m}^2/\text{g}$ . No other adsorbent has a similar capability. Molecular adsorption onto the walls of the pores may be considered as analogous to a bee (enlarged molecule) flying at supersonic speeds in random directions through the Godhardt tunnel in Switzerland. Each time the bee collides with the wall of the tunnel, it lingers for a while until it takes off in a random direction. It will take, on average, thousands of years before the bee reaches the other side of the tunnel. Flowing through a gasmask canister, a molecular-sized bee must pass thousands such tunnels. The higher the boiling point of an agent, the longer the time it rests after the collision with the wall. In practice, the molecular bees remain in the tunnels (pores) indefinitely.

Aluminum oxides and molecular sieves approach the capacity of activated carbon; however, they quickly become saturated with water vapor from the environment. Active carbon becomes saturated as well but at a slower pace and with weaker adsorption forces for the water molecules. As a result, most agents will displace the water adsorbed onto the carbon and remain adsorbed. Active carbon is amorphous and, therefore, a soft and brittle material. For use in canisters and protective clothing, it must be sufficiently hard to withstand the forces imposed during use. Often, the activation process of the carbon is controlled in such a way that a carbon with a hard shell results. Another way of dealing with this problem is to immobilize the carbon, thereby preventing friction between the particles.

Standard military active carbon, impregnated with salts to protect against HCN and cyanogen chloride, does not adsorb some TICs very well, although the carbon will lower the inhaled concentration and proportionally increase the exposure time. The higher the exponent in the toxic load equation, the lower the hazard will be. Industrial canisters (ABEK) are able to adsorb those gases in limited quantities. In certain TIC releases, the concentration of compound released may be so high that atmosphere is oxygen deficient, implying that filtration is pointless and the air supply systems are the only option. Canister penetrants are sometimes mentioned as a concern, however, except in the case of extremely high concentrations, no penetrants have been found to penetrate canisters sufficiently to yield hazards. In Canada, a special impregnation is used (TEDA) to stop these filter penetrants.

Important for the performance of vapor filters is the adsorbent volume, the surface area, and bed depth, which determines the breathing resistance. Following these aspects, the capacity and efficiency of the filter is determined by the porosity and pore size distribution of the carbon, the packing density, and the sensitivity for water vapor adsorption.

#### 6.2.3.3 Improved filters

Present day aerosol filters will stop biological warfare agents with an efficiency of better than 99.99%, meaning that only one in a million molecules will pass through. Because there are highly potent biological agents, it is argued that the efficiency must be improved. Mechanisms for improvement may include photo bleaching or the use of electric fields. Both require considerable quantities of energy and are applicable only to larger filter installations in collective protection. However, it is difficult to imagine that a first responder will ever face a high concentration of a biological agent. First responders should arrive at the scene of an

incident no later than 15 minutes after release, and for biological releases, probably after days or perhaps never. At worst, under any conditions, the fraction of agent that is still airborne will be low.

Filter media impregnated with biocide materials, such as iodine, can kill bacteria and viruses on the aerosol filter. For more than 25 years, electrets filters (filters composed of permanently charged fibers) have been heralded as a promising development. Regenerative filters with an endless service life are often called the most promising filters, but they have very high energy demands.

# 6.2.3.4. Endless filters

These include regenerative adsorption (e.g., pressure- and temperature-swing adsorption), membrane separation methods, and reactive methods, such as catalytic oxidation and discharge plasmas. These developments consume large quantities of energy and are applicable only for collective protection shelters. Membrane systems always must be combined with a carbon filter to remove the last traces of hazardous compounds. Catalytic oxidation and plasma discharge produces unwanted compounds during the destruction of agents and require additional active carbon filters. The most promising filter development is the pressure- or, alternatively, temperature-swing adsorption. These systems consist of two parallel filters that intermittently produce clean air in a high-pressure phase or are purged during a low pressure phase, with a fraction of the clean air flowing in the reverse direction. As indicated by the name, either the pressure or the temperature swings during each cycle.

## 6.2.3.5. Integration with environmental control systems

Most often, a COLPRO filter is an integral component within a host environmental control system (ECS). The environmental control system uses a compressor to compress and decompress the air, similar to the one used in a pressure swing adsorber. During the compress/decompress cycle, the air cools and water vapor condenses. Together with water vapor, often significant amounts of contaminants are removed, especially of the less volatile agents.

# 6.3. Respiratory protection

#### 6.3.1. Introduction

The protective mask has undergone development in various stages over the past century. The first military masks used in WWI were pieces of cloth impregnated with chemicals, used to cover the nose and mouth. This developed quickly into full-face piece respirators. In the first versions, the face piece was made from leather. Although far below the present standards, these masks did a good job of protecting the soldiers. Once protected, attacking the troops with gas was no longer effective. During WWII, rubber was introduced as the face piece material. This resulted in a much better fit and dramatically reduced leakage around the face. The rubber was not always resistant to the chemical agents, and the search for a resistant, flexible, and durable rubber began. Currently, most masks use a bromobutyl rubber material for the face piece. Better face seals are accompanied by the problem if eyepiece fogging. After the Second World War, most armies introduced the Tissot principle. By using an inner half mask, the relatively dry inhaled air was first directed over the eyepieces to dry them, with the added advantage of considerably reducing the dead volume inside the mask. The smaller

the dead volume of a mask, the more carbon dioxide from the exhaled air will be purged out of the mask and not be available when taking the next breath.

The protective mask or respirator has several problems, such as a restricted field of vision and, more importantly, physiological load. The mask wearer always must deal with the resistance to both inhaling and exhaling. Releasing inhaled air is usually a passive process; however, exhaling against an overpressure is difficult. It is, therefore, desired to keep mask wearing to a minimum. After an attack, the total protection provided to the mask wearer depends on advanced warning, the time required to don the mask, how well the wearer can hold his breath during masking, and the protection factor once the mask is donned. All sample calculations show that it is essential to don the mask as quickly as possible. In WWI, the allied forces developed the nine seconds rule. The German forces trained for two hours per day to don the mask in six seconds. In an effort to overcome this issue, in the late seventies, the UK developed a simple surgical mask, called a facelet, which could be worn all the time in anticipation of an attack. Most of the present day do-it-yourself masks are based on this principal. Although the facelet is no longer in use by the military, the degree of protection is such that it can be worn to prevent civilian casualties if confronted by a terrorist chemical attack.

For the military, requirements are more stringent. First of all, the mask must provide protection against all concentrations and dosages that can be expected on the battlefield. The mask should not degrade the ability to perform the mission, and the physiological encumbrance should be as low as possible. The mask should be completely compatible with the other equipment, especially with optical sights. To date, even the latest developments, displayed at the 9th CBW Protection Symposium in Sweden in 2007, demonstrate that the ideal has not been reached.



The *S10/FM12/M50/C50* series mask was developed by Avon, originally as the S10 for the UK market, The FM 12 was an improved version used in many countries by military and first responders. It is sometimes called a global respirator and is used by the OPCW inspectors as well. The mask evolved further and was adjusted to the US market by the development of the M50 and C50, the next generation in comfort and safety. To partly overcome the physiological encumbrance and to improve the protection factor, a powered air supply can be incorporated into the masks. Although this provides a comfortable way to breath, in most cases, the power required adds considerably to the weight that must be shouldered by the soldier. This principle is more appropriate for soldiers operating from powered systems, such as an armored vehicles or aircraft.

## 6.3.2. Face seal

Full-face piece respirators rely on a seal between the rubber and the skin to prevent leakage of contaminated air. This seal is either (usually) around the face or (seldom) around the neck. The pressure inside the mask is negative during inhalation, and air may leak in. Therefore, masks are designed with a reflected flange to make a seal between the mask and the face. This type of face seal usually provides protection factors over  $10^4$  for vapors. Usually, the protection factor for aerosol particles larger than 1 µm is much higher. Because the pressure inside these masks is negative (with respect to atmosphere), during the inhalation phase of the respiratory cycle any breaks or gaps in the mask seal will allow contaminated air into the mask. Blower-supplied respirators, on the other hand, maintain a positive pressure (with respect to atmosphere) within the mask during a large part of the respiratory cycle, thus ensuring that inward leakage through the seal is reduced. There is a short period of negative pressure during exercise that is difficult to compensate using a blower. A feedback loop that sets the power of the blower according to the pressure inside the mask is beneficial for reducing energy consumption, but the blower must respond very quickly. Often, a combination of a blower and increased expiratory resistance is chosen to increase the positive pressure. Therefore, blower-supplied respirators, in general, offer a higher level of protection than negative pressure respirators. Blower-supplied systems are used almost exclusively by platform users such as aircrew and combat vehicle crewmen. Because of the cost, logistics, burden, weight, and bulk of these systems, they have been deemed impractical for use by infantry and other ground troops. For these reasons, ground troops rely on negative pressure respirators to provide their required protection. The improvement of these respirators has been the primary focus of research during the past decade and will likely continue to be needed during the next decade.

#### 6.3.2.1. First responders

First responders to a chemical or biological incident often regard the incident as a HAZMAT (hazardous materials) incident. In this type of incident, contact with splashes of liquid and high concentrations of volatile agents cannot be excluded. For this reason, in any HAZMAT incident, the responders are equipped with totally encapsulating suits and an independent clean air supply. Under such circumstances, a face seal is irrelevant. However, donning those suits is very time-consuming, the mobility of the wearer is seriously restricted, and the physiological load is high. It has been argued that in the case of a terrorist CB incident the first responders will arrive not earlier than 15–30 minutes after the release of the agent. High concentrations of vapor or aerosol that might have been released in the initial phase have likely blown away or settled to the ground. A first responder will, therefore, likely experience low concentrations in vapor and aerosol in the aftermath of the incident, although some liquid residue at the point of release might be encountered. The "hot zone", in which first responders must operate, is in military terms often considered a decontaminated zone (see paragraph 8.7). The quantities of contamination may usually be handled easily by gloves and boots and do not form a particular hazard for the respiratory system. Alternatively, the contamination can be covered by foam or adsorbent materials. This argument leads to the conclusion that the face seal for a first responder operating in a CB incident should be at most as good as the face seal of a military mask.

# 6.3.3 Mask fit

To obtain a good fit, masks must be available in several sizes to account for varying facial structure and shape. Usually, 4 sizes will suffice for males of a particular ethnic background, with the number growing to 6 when males and females are considered. For large and diverse populations, such as are encountered in the US, more sizes are required to cover 95% of the population. There will always be a small fraction of the population that will not get an adequate fit with a full-face piece respirator.

A particular problem for the face seal is beard growth. The fraction of the male population with an excellent fit is reduced by more than 50% with beard growth of over 8 h for Caucasians. After 4 days, less than 25% of the original good fits can be qualified as adequate. People with a full beard never achieve a good face seal. Typically, the protection is three orders of magnitude below that for well-shaven men. Masks with a neck dam have been developed to provide protection to full-bearded people. Consequently, bearded terrorists are not likely candidates for handling CB agents, except in case that they posses and wear gasmasks with a neck dam.



Mask fit in the field, always a difficult problem, requires special instruments such as the TSI, which uses naturallyoccurring aerosols to ensure a good fit.

#### 6.3.4. Outlet valve

The outlet valve is another source of leakage of contaminants into the mask. Outlet valves are constructed with a flexible flap that seals to a metal or plastic seat. As soon as a negative pressure is applied, the valve closes, preventing air from passing backward into the mask. If the valve is not completely sealed, e.g., by dust or hairs on the seat of the valve, contaminated air may enter the mask. During exhalation, the valve is in contact with moist air and the flap

usually becomes wet. Under normal circumstances, this would improve the quality of the seal. Unfortunately, dust also adheres more easily. It is, therefore, of paramount importance to keep the outlet valve clean by flushing with water.

# 6.3.5. Fitting protective masks

The previous discussion of mask seals and sizing is based on results mostly obtained during laboratory tests. Data show that fitting a mask in field conditions results in a lower protection factor. The infectivity of some biological agents is such that 10-100 particles inhaled might be very hazardous. These small numbers require a high protection factor if an aerosol of the right particle size and of sufficient concentration is encountered. Protection factors of over  $10^5$  may be required, which is difficult to achieve in the field. Two things are of importance here. First, a high concentration of small aerosol particles will exist only for a short time. The challenge to first responders when they arrive on the scene, therefore, will likely be at least two orders of magnitude lower. Secondly, the face seal leakage for particles of a few microns is far less than for vapor.

# 6.3.6. Other facilities.

Other problems encountered while wearing a mask are communications, be it via radio or by direct speech from person to person. To address this issue, speech membranes have been introduced, often one in front of the mouth and the one on the side of the mask to make telephone use possible.

Yet another problem is the use of corrective lenses. Usually, prescription glasses can be mounted inside the face piece and sometimes contact lenses are prescribed. Compatibility with optical devices and binoculars is also an issue. Some face pieces have a flexible transparent section to see through to help overcome this problem. However, this is primarily a military problem. First responders mostly must deal with a restricted field of vision. As a result, training is required to adjust operations and procedures to such restrictions.



Drinking whilst wearing a mask is possible but requires

some training, especially to prevent contamination

of the connectors to the flask or mask.

Military procedures can require that a mask be worn for 24 h, sometimes including sleep sessions. For that reason, most masks are equipped with a drinking capability that allows for the intake of clean water or liquid food. In view of the changes in the CB threat in the coming decade, it is highly likely that the 24 h wearing time will be reduced to a few hours. Within a few hours, troops will evacuate to a toxic-free area.

## 6.3.7. Breathing resistance and filter canister

Although a mask in combination with a hood will almost certainly increase heat stress, breathing resistance against an overpressure (to open the outlet valve) is considered more uncomfortable. Breathing resistance mainly arises from the filter canister, and many efforts have attempted to reduce the resistance. Because the requirements have stayed the same over the years, only marginal improvements have been achieved. If the filter capacity could be reduced because previous levels of protection are no longer required, the goal of a low pressure drop, which reduces resistance, could come realizable. However, due to the increased biological threat, it may well be that the particulate filter must be improved, resulting in an even higher pressure drop. A powered air supply might offer a solution, but if not designed properly, it may contribute to resistance during exhalation of air, which is, again, uncomfortable. A microprocessor-controlled ventilator that supplies air during inhalation but stops during exhalation has been demonstrated. Even with the cited drawbacks, powered air supplies are usually regarded as more comfortable by the user and increase the field protection factor considerably.

# 6.4. A. Body Protection/Protective Clothing for Military

# 6.4. A.1. Background

Once troops were equipped with respiratory protection, it became ineffective to attack them with chemicals. In WWI 250 kg of HE ammunition was required to cause one casualty. That single casualty could also be caused by 100 kg of asphyxiating gases. With a protection factor of only 10, the amount of asphyxiating gas required to make one casualty became prohibitive. For that reason, research was begun to find agents that could circumvent respiratory protection. One line of research sought agents that are poorly adsorbed by active carbon. Although some researchers claim that they had found a mask penetrant, it is highly doubtful that they did. Even for a sufficiently toxic compound that is not well adsorbed by the carbon, the active carbon would still lower the concentration over a longer time period. In order to be hazardous, and agent would have to be present at higher concentration over a long period. As an example, only near a storage tank leak will the concentration of HCN be large enough to overwhelm the capacity in the canister. None of the agents mentioned in schedule 1 of the CWC can be regarded as filter penetrant.

Another line of research was aimed at finding an agent that would act through the skin. In 1916 Germany scanned 70 compounds for this purposes, and two were introduced, with one being very effective. This agent was named mustard, because of the tickling sensation it caused on the tongue, and it was code named HS for Hun Stuff, or HD for a distilled product. In the first three weeks of its use against British troops during WW I, mustard caused more chemical casualties than for the whole year of 1916. It worked as a liquid and also as a vapor, and it penetrated carbon filters as an aerosol. (The first respiratory protection did not have a particulate filter.) Later nerve agents were discovered that also acted very effectively through the skin, in particular agents with the code names GD, GF and VX.

The hands and feet of a soldier can come into contact with liquid agent when walking through a contaminated field or when touching contaminated equipment, e.g. carrying a rifle. For that reason, hands and feet are protected with material impermeable to agents, but also impermeable to water vapor and air. Because of the high degree of protection required for the hands and feet, these items contribute significantly to the discomfort of the total body protection.

The rest of the body of the soldier can in principal be protected by 4 types of garments:

- A1 type; air-permeable, reusable, as over-garment or instead of combat suit
- A2 type; air-permeable, disposable, as over-garment
- B1 type; air-impermeable, reusable, on top of underwear for decontamination purposes
- B2 type; air-impermeable, disposable, on top of combat suit, mostly poncho type.

Over the years several intermediate concepts have been developed, for example semipermeable fabrics that allow the transport of some water vapor through the fabric provided the climate conditions allow a sufficient water vapor pressure gradient between skin and the environment. In another case, the outer layer in the A2 type garment served as the liquid barrier and was separated from the active carbon layer, which served as a vapor barrier. The latter is worn as underwear.

Air-permeable protective clothing is almost exclusively designed as an over-garment. The trousers allow for donning the suit while wearing combat boots. It has been suggested to use the over-garment as a combat suit when used in a hot climate. The oversize design causes a lot of air to be enclosed in the suit, which adds to the insulation, and a bellows effect can pump appreciable quantities of air through the fabric and the closures. Understandably, this decreases the degree of protection. The bellows effect occurs as a result of the movement by a person dressed in an impermeable suit. The air entrapped in between the fabric and the body is pumped around due to movement. This causes pressure differences and air can leak in through seals and closures. This can be a very serious leakage, and impermeable suits provide only very little protection against vapor and aerosol. They are good for protection against liquid splashes.

An air-permeable material incorporates two barriers: an inner vapor barrier layer and an outer shell liquid barrier, which also reduces the vapor challenge from a liquid contamination. The working of the inner layer relies on adsorption and consists of immobilized activated carbon. Liquid chemical agents should not come into direct contact with the carbon because local saturation would occur. A second function of the outer layer is to allow evaporation of the agent to the environment, thus not challenging the carbon. A concept where evaporation could be enhanced by using a wicking outer shell has been abandoned. The agent wicks not only over the fabric but also into the fabric, thereby increasing the challenge to the carbon, although over a larger surface area. The outer layer of most of the current CB protective clothing consists of a water and oil repellent impregnated fabric of cotton, or mixed weaves of polyester/cotton (Europe) and nylon/cotton (US).

It should be noted that from a military operational viewpoint, chemical protection has always been seen as an add-on article. Senior officers do not want soldiers to disrobe/striptease in the field, and therefore the over garment concept became very popular. Only in case of a significant chemical threat is the protection worn. The military concept was to fight contaminated and win the battle first before protective equipment would be restored or replaced. This led to protection times initially of six hours and later of 24 hours. With the much lower present day probability of a chemical attack and the expected lower intensity, the degree of contamination will be reduced. Soldiers are also more likely to withdraw to a clean area to get new equipment.

The doctrine of protective equipment utilization is of great importance. Gloves, boots and mask are donned in the field when a chemical attack is imminent. Previously the overgarment was donned in the field as well, but recently the US has adopted another doctrine. During operations in Iraq, the chemical protective suit was used as a combat suit and worn open in anticipation of an attack The necessity to provide CB protection for use in a wide range of climates and over the full range of work rates is one of the main challenges facing clothing scientists today.

# 6.4. A.2. Protective characteristics

The starting point for protection is an understanding of the challenge level (Chapter 3) and the effect level (Chapter 4). During the cold war period, the challenge levels and the acceptable effect levels were quite high because a certain risk was accepted. In the new century, that risk is no longer accepted, and the maximum allowable exposure level is the just visible effect level. For mustard gas, the allowable exposure dosage is set at a 10-20 times lower value, 25-50 mg·min/m<sup>3</sup>, than during the cold war. In a Canadian study, it is mentioned that the challenge is also reduced (1). For vapor, it is 5000 mg·min/m<sup>3</sup> at a wind speed of 1 m/s and below. The challenge level is proportional to the inverse of the wind speed and therefore 1000 mg·min/m<sup>3</sup> at 5 m/s wind. Defining the challenge levels at a certain wind speed is of importance because the wind speed determines the wind pressure and the flow through the clothing. Aerosols in the right particle size are more difficult to produce, and the challenge levels are set at half the values for vapor (2). The allowable exposure levels have been set by NATO (see Chapter 4.7 or Review of Acute Human Toxicity Estimates for selected Chemical Warfare Agents, Committee on Toxicology National Research Council, National Academic Press, Washington D.C., US, 1997.)

The protective performance of clothing was tested using volunteers who were subjected to vapor or liquid mustard agent attacks. After WW II the large-scale troop trials became more and more unacceptable and arm tests with a swatch of material were performed. In rare cases the tests were done with nerve agents. Later, many laboratory swatch tests were developed using vapor, neat or thickened liquid from deposited or falling drops and drops under pressure. Although the swatch testing can provide a good indication of the protective quality of the fabric, it says little about the protective performance in the field. The overall performance of a CB protective garment in a field environment is not solely determined by the quality of the protective materials themselves.

Leakage through seams, closures, and interfaces between the garment and the auxiliary equipment, such as footwear, gloves, hood and respirator, is a significant part of the total amount of agent that penetrates the garment. When using the protective system under dynamic conditions, body movements and wind tend to increase this penetration.

Over the last ten years, several whole system tests have been developed, in which the entire protective system, including masks, boots and gloves, is worn by a marching mannequin and then challenged with live agent (UK) or volunteers when challenged with a CW agent simulant (US, Canada, CZ, NL). The most elaborate test in this respect is the one performed in South Africa (3). In this test, the degree of protection, using a simulant as well as the physiological load, is determined simultaneously in a field trial involving volunteers crossing

an obstacle course. Besides body temperature and heart rate, the time required to complete the tasks is also measured. Also, the volunteers are equipped with samplers that sample the vapor that has penetrated the suits either through the material or through the closures. Experiments show that the closures give the largest contribution to the ingress of agent.

# 6.4.A.3. Vapor protection

Agent vapor penetrates through the fabric as a result of the limited depth of the filter layer and the pressure caused by wind. Adsorption kinetics plays a vital role in this process.



The soldier on the right is very well protected by his chemical defense suit worn during operations. The other one is relying on a bag of flower which appears to be an excellent decontaminating powder



The chemical defense clothing does not prevent the soldier pulling from his weight. The system worn during the invasion of Iraq is the only suit actually worn during combat operations. A limited number of Iranian forces might have worn a foam type of suit during combat for protection from Iraqi CW attacks in the 1980s.

Usually the capacity of the carbon filter is very large in comparison to the challenging vapor dosage. Most of the present day materials provide a protection factor over 1000, which means

that an outside dosage of 5000 mg·min/m<sup>3</sup> is reduced to a value far below the level where initial agent effects are observed. Higher air permeability results in higher airflow and less efficient adsorption. However, when the maximum credible worst case challenge is expected to be 5000 mg $\cdot$ min/m<sup>3</sup> at a wind speed of 1 m/s as proposed in a Canadian study (1) then the challenge dosage at 5 m/s would be reduced to 1000 mg·min/m<sup>3</sup>, requiring only a protection factor of 40 to reduce the outside dosage to a value below the threshold dosage for effects. A second route of skin exposure is by ingress of agent through seals and closures. In human volunteer tests with simulant vapor exposure, it appears that the deposition near the closures (sleeves, hood) is about ten times the value found at places far away from the closures (middle back). This would indicate that the ingress is about ten times the penetration. Ingress for mannequins is always larger because it is more difficult to obtain a seal around a hard wrist or inflexible head (6). The ingress of agent differs considerably for air-permeable versus airimpermeable systems. In the latter, it is far higher due to the bellows effect always present when personnel are in motion. At the end of the day, it is both ingress and penetration that must be reduced sufficiently in order to provide protection. Selecting a fabric that prevents penetration is not sufficient because ingress must be sufficiently reduced as well.

## 6.4.A.4. Liquid protection

The surface tension of liquid CW agents is such that small droplets result when the agent is sprayed or explosively disseminated. The particle size distribution is from about 50 to 500  $\mu$ m with most of the mass in the 100-200  $\mu$ m range. The maximum drop size is about 1  $\mu$ l or 1 mg of agent. Present day protective fabrics do not have problems providing protection from these drops, either deposited, free falling or under pressure.

Thickened liquid CWA results in much larger drops. Usually the median diameter is 10 times larger (2 to 3 mm) than "neat" drops, but larger drops (5 to 6 mm) can occur close to the point of dissemination. The number of large drops however will be small and the area covered with the larger drops will be very small. In the majority of cases the contamination on personnel will be from drops equal to or smaller than 4 mm. In laboratory tests, the protection against the larger thickened agent drops is problematic because the protection time will be determined by the capacity of the filter layer. The suits in use by the OPCW inspectors have a filter layer containing close to 200 g/m<sup>2</sup> of active carbon, which is twice as high as for other filter fabrics. These types of suits offer the longest protection against the larger drops. The protection time decreases further when pressure is applied to these drops. It is therefore always wise to remove visible liquid contamination, preferably using a decontaminant and to replace the suit with a new one as soon as possible. The membrane-type suits are supposed to offer better protection against larger liquid drops but that is not guaranteed. The original membranes were not resistant to chemical agents and had to be additionally treated. This treatment is chemically based, and aggressive chemical warfare agents might destroy the agent resistant properties of the membrane. This must be thoroughly tested against the known threat agents but will remain uncertain for unknown agents.

## 6.4.A.5. Aerosol protection, Chemical Agent

Except for a limited filter efficacy of the outer layer, air-permeable protective materials do not provide any aerosol protection. However, the fraction of the penetrated particles that is deposited on the skin is very small, as most of the aerosol is carried away with the flow of air that caused penetration. Fedele (4) has derived the theoretical deposition velocity of a fine particle aerosol as 0.00001 m/min. In experiments, it proved to be 0.0001 m/min. (5). The

reason for this lower deposition velocity is that the particles are carried away by the airflow around the skin. Again, the deposition is about ten times larger near the openings. So from an aerosol challenge of 2500 mg·min/m<sup>3</sup>, only 0.25 mg/m<sup>2</sup> or 0.025  $\mu$ g/cm<sup>2</sup> are deposited, far below any known effect level. Even deposition 10 times higher near the closures does not form a real hazard. For details see chapter 3.

# 6.4.A.6. Aerosol protection, Biological agents

As with the CWA aerosols, BWA aerosols will be deposited on the skin in small quantities after penetration through the fabric. Almost no biological agents penetrate intact skin. There are one or two exceptions. One well known exception is anthrax, which causes wool sorter's disease. However, anthrax probably needs a partly broken skin to reach the living cells and interact. Some believe that trichotecene mycotoxins or "yellow rain" is another exception. Recent evidence strongly suggests that this is an artifact.( Meselsohn, M., Yellow Rain; The CBW Conventions Bulletin, Quarterly Journal of the Harvard/Sussex Program on CB Armament and Arms Limitation, Issue 12, Messages on the CBW discussion group website from the Swedish International Peace Research Institute, Jean Pascal Zanders (Editor), June 2002). Even if anthrax and trichotecene are a threat to the skin, both agents are much more effective via the respiratory route. The challenge dosage of Bioaerosols in particles.min/m<sup>3</sup> is not known, but for the skin it can be derived from the dosage that would be effective when wearing a mask. A million times more agent penetrates through the respiratory system than through 20 cm<sup>2</sup> of broken, open skin. Consequently there is absolutely no need to protect the skin from biological aerosol attacks unless a mask with a protection factor above  $10^7$  becomes available. Details are presented in Chapter 3 and Annex A to Chapter 6.

For biological aerosols, the amount that leaks through seal and closures is about ten times higher than the amount that can be deposited from penetration of the fabric, but this deposition from a high challenge dosage is still below the effect level.

Biological aerosol that is deposited onto the fabrics of the combat suit and protective garment can cause a problem. During doffing of the suit these particles can become airborne again and form a respiratory hazard. It would therefore be wise to build in a biocide that kills the deposited biological. Building in a filter to stop aerosols is counter-productive. Due to the higher resistance of the fabric, it becomes less permeable to air, and the pumping effect is increased. This results in a higher ingress of agent through seals and closures. An even more serious problem is that the aerosol filter stops the agent, and a larger fraction is deposited into the fabric and bonded with weak forces. So during doffing, the respiratory hazard might dramatically increase. Biological aerosols are not too great a hazard to the skin but can form a very serious secondary hazard to the respiratory system when doffing protective clothing.

#### 6.4.A.7. Protection against Toxic Industrial Materials

Toxic industrial chemicals can be hazardous to the skin as splashes of liquid or high concentration of vapors. It is most likely that if this occurs it will be during release from a storage site or transport tank and will threaten only those in the immediate vicinity. In cases such as this, the respiratory protection is inadequate and fully encapsulating impermeable suits will be necessary. This type of incident should be treated as a HAZMAT incident. CB terrorist incidents including those involving TICs will not result in bringing the First Responders in contact with liquid neither will they face a spray attack as might be

encountered by military personnel. Protection as provided to the military provides more than sufficient protection for the First Responder.

# 6.4.A.8. Air-impermeable clothing

For heavy duty purposes such as contact with large quantities of water during decontamination, some troops are equipped with air-impermeable clothing, usually suits made of butyl rubber. This type of clothing causes great heat stress and restrictions in movement that it will likely never be accepted as the general protective clothing for soldiers. In Eastern Europe and Germany, the disposable poncho was favored to prevent gross liquid contamination of the suits. The poncho does not provide any protection against vapor or aerosol. In order to overcome the heat stress, membrane systems were developed that are impermeable to air and agent but let some water vapor through. These systems will only provide proper protection when worn on top of active carbon-loaded underwear, however, the vapor protection afforded by permeable suits is still superior (see paragraphs 6.4.B.). The physiological load induced by the membrane-type of suits is significantly above the load induced by permeable suits. When membrane-type -suits are worn alone on top of underwear or with a carbon-loaded under coverall, and no masks, gloves or over boots, the endurance times are significantly below those of permeable suits. When worn in combination with all other protective equipment (mask, gloves and boots), the differences between the two types become marginal because the larger part of the physiological load comes from the mask, gloves and boots.

In addition, air-impermeable and membrane-type clothing have some serious drawbacks for military applications:

- They demonstrate the bellows effect. Significant quantities of air are drawn through the seals and closures. The ingress of air is about two orders of magnitude above the ingress in air-permeable suits.
- They are noisier than textile materials.
- It should be noted that all impermeable suits that are not flushed with clean air or have a built-in filter show no protection against vapor at the end of the day. It can be easily shown that the dosage inside equals the dosage outside after a period of time (See paragraphs 6.4.B). The lowering of the concentration due to absorption by the skin is disregarded here, but this is just what the system should prevent. The wearer may receive an even higher exposure if the suit is not "flushed out" before starting a rest period after activity and exposure. For this reason this type of suit only works well if flushed with clean air and preferably with a constant positive pressure. If a hole exists in the clothing, the bellows effect causes large quantities of air to enter the system, again much larger than with a hole in an air-permeable system.

# 6.4.A.9. Laundering and decontaminating suits.

In the 21st century, it has become unlikely that there will be frequent and intensive chemical attacks against protected military personnel. But being unprotected is a clear invitation for the use of chemicals. For that reason, suits have to be worn for extended periods in anticipation of an attack. This makes laundering mandatory because replacing the suits with new ones every time they get soiled becomes very costly. Laundering degrades the protective performance of clothing materials. The water and oil repellence of the outer layer can withstand at least ten washings but the active carbon layer is poisoned incrementally during every laundering cycle,

especially when rinsing is carried out with cold water. The soap residues might be responsible for an increased water vapor adsorption by which the kinetics for adsorption of agent is slowed.

Once a suit has become contaminated with vapor, aerosol or liquid CWA, or particulate of BWA, the question arises whether the suit should be decontaminated. Regarding vapor and aerosol there is more than sufficient capacity in the filter layer to withstand several major attacks. In the case of an attack with thickened liquid, the capacity of the filter layer at the location of the contamination (where the "drops" have landed) determines the penetration of agent. A legitimate question to ask is whether a suit can be safely worn continuously once contaminated. Unfortunately, decontamination of suits by laundering, steam treatment or chemical extraction never results in 100% decontamination. There will always remain a fraction of agent in the fabric. It is unlikely that this fraction will cause secondary exposure easily, but when subjected to a fuel spillage, potentially hazardous desorption is possible.

In reality, the only situations that require decontamination is after an attack with thickened agent or biological agent attack (secondary respiratory hazard). Large drop contamination on suits can be evaluated using pieces of liquid detection paper mounted on the suits. Most of the liquid will be removed by laundering, which will also work for biological aerosols that have been deposited in the fabric.

"Self-decontaminating" systems have been proposed. The idea is that agent is destroyed by a catalytic or enzymatic process. However, these processes are often highly selective, and an enzyme that works with one agent might not work with another. Furthermore poisoning of the enzymes or catalytic systems by decomposition products is highly likely and perhaps unavoidable. The local high contamination densities in a thickened chemical agent attack will exceed the capacity to decompose the agent. In order to work in a catalytic way, either the enzyme or the agent must be mobile, and the decomposition product must be released from the enzyme freeing the active site for the next reaction. As the catalyst/enzyme and the agent are fixed at a site, the reaction will be more one to one (stoeichiometric) than catalytic. Self-decontaminating materials regarding biological agents are more likely to be developed. It might be possible to impregnate broad spectrum, non-selective biocides onto fabrics. These products already exist in the medical arena, e.g. wound dressings. Currently the use of silver nanoparticles has been developed as biocides. However, these nanopartcles are so small that they themselves or the ions released from the particles might penetrate the skin. The biocides might start to kill the internal bacteria flora.

#### 6.4.A.10. Encumbrance of skin protection

As with the respirator, skin protection, gloves, boots and suits also cause much discomfort. The air permeability of modern CB clothing permits transport of heat and water vapor which make these suits relatively comfortable to wear when worn as a combat suit, without gloves and boots. On the other hand, when worn completely closed there is less ventilation and both the hands and head no longer contribute to the cooling surface. The use of the CB suit as an over-garment increases the number of clothing layers and ads to the insulation and physical load. This limits the wear time of a CB suit and the performance level of its wearer, particularly in situations where high work loads, high temperature and high relative humidity are combined. At ambient temperatures above 20°C during heavy work, heat strain related problems occur generally within one hour. Introducing work/rest cycles can mitigate the effect and more work can be performed, although at a slower pace.



Heat stress is often ascribed to the protective clothing. The pictures make it clear that there are many other contributing factors.

Several aspects other than heat stress have negative impacts. For instance, the gloves are thick and not very flexible. Dexterity is often poor, and the sweat accumulating in the gloves causes the skin to weaken, increasing susceptibility to abrasion. The over-boots make running more difficult, as the grip of the sole is usually different from that of the combat boot. The suits are often bulky and the equipment that must be worn externally is not always optimally designed for compatibility.

Protection and hindrance are two opposite aspects that affect the ensemble. More protection means more hindrance, and vice versa, so the problem is to strike the right balance. It should be remembered that it is not just the suit that causes the hindrance, as gloves and boots are cumbersome in certain tasks, and for many other tasks the respiratory hindrance of the mask is the limiting factor. Reducing the hindrance of one item without changing others will bring only relief in special cases. What is required is a system approach in which all the items are reviewed regarding the required protection factor, the required protection time and the endurance time.

For some highly demanding tasks, and when sufficient energy is at hand, cooling vests have been proposed. The vests are based on either ice cooling or on perfusion water systems. Using cool air from an air conditioner might be possible in some cases. Also the use of dry air will help evaporate sweat and cool the body. Again, these systems are energy-demanding and applicable only in special cases.

#### 6.4.A.11. Alternative concepts

A reduction of the number of clothing layers can lead to a decrease in the thermal load. One way to achieve this is the incorporation of vapor protection into underwear. With this undergarment concept, a better overall protection can also be expected. A skin-tight protective undergarment has the advantage that leakage through openings will be much lower in comparison to a loose protective over-garment because there is no airflow through and under

the clothing. The penetration of agent vapor through the undergarment is mainly caused by diffusive transport, and not by convective transport. Due to wear, sweat poisoning, and washing, deterioration of the carbon layer in a skin-tight undergarment will be worse than with the "layered" protection approach. The use of shorts and T-shirts loaded with active carbon might become an acceptable option, protecting the most sensitive skin areas compared to arms and legs, which are at least ten times less sensitive.

All protective functions combined in one layer and integrated in the combat suit is the ideal situation, but materials that would strike the right compromise between heat stress and protection are not yet available.

Over the past two decades, attempts have been made to introduce semi-permeable membranes into protective clothing. This membrane would be permeable to water vapor but impermeable to agent liquid, aerosol and vapor. The idea has not fully materialized. Under hot humid conditions, agent may still penetrate. Also, the thermal load of this system is too high in hot climates. Finally it should be noted that agent penetrating through closure produces at the end of the day the same dosage (C x t) with and without the protective layer. (See paragraphs 6.4.B)

Since WWI, the use of protective skin lotions has been suggested to replace gloves or to protect sensitive body parts. It is good to note that after WWI the remarks about these lotions included that they are impervious, thick and uncomfortable, greasy, short-lived and sometimes increase the effect of mustard agent. Modern lotions will not only reduce the rate of uptake by the skin but could also decompose the agent. Unfortunately, the decomposition will work only with certainty for a select number of agents.

Probably the best solution for reduction of the physiological load would be to custom-size suits to the wearer, or at least make suits adjustable in size. This may be achievable now for special personnel such as pilots. In the future, it may be possible to couple scanning machines directly to suit-manufacturing robots that will make a tailor-made suit for the individual.

# 6.4.A.12. Testing the encumbrance and protection of protective clothing

During and shortly after WW II, it was the custom to simultaneously test both encumbrance and protection by spraying a group of volunteers in full protection with mustard agent and measure the time they could continue to operate. End points were either too high of a body temperature or too severe the effects from mustard agent. Illustrative of these tests are troop trials in Australia where troops in summer uniform, while marching and carrying sandbags, were exposed to mustard vapor. After the exposure the troops had to cross a "Japanese style" obstacle course, constructed of sharp bamboo sticks. In the late 20<sup>th</sup> century, records of these exposures were made public. It showed how tough the Australians really were, but it also mentioned the high cancer rate for those taking part in the trials. Although these tests would provide absolute proof of the degree of protection and the degree of encumbrance, they are absolutely unacceptable from a humanitarian point of view. In recent times, activists have protested when animals are subjects for CW testing, cosmetics and even when used for medical purposes. It is therefore obvious that other ways of testing have to be developed. Choosing the protection that meets the requirements needs a lot of testing. The soldier is protected by an Avon FM 12 mask, German-made gloves and boots, both composed of butyl rubber, and a US-type Suit JSLIST produced in the US by Texshield or in Germany by Bluecher GMBH. These items have been selected by many countries. The suit is an outer shell of Nylon/Cotton 50/50 treated oil and water repellent. The inner shell consists of active carbon spheres glued to a textile carrier. These systems have a high carbon load and therefore a high capacity and protect well against the larger drops of thickened agent. Due to the high capacity the suit can withstand the poisoning induced by 7 weeks of wear and weekly laundering. A desert camouflage version has been used as a combat suit by US troops invading Iraq in 2003.





Testing is often performed through evaluation an pyramid. First, materials are pre-selected by swatch testing using CW agent. The next stage is subsystem testing, and the final stage system testing using agent and simulant on robotic mannequins and simulant on human volunteers. It is preferred to do the chemical agent protection testing (using simulant) and the physiological load and heat stress testing simultaneously in one test run, mimicking operational conditions much as as possible.

Large variations in tests and test criteria exist with each sponsor believing their test to be the (only) correct one. In particular, across testing protocols, there is a large variation in the

airflow through the sample. In practice wind will induce airflow through part of the fabric with airflow out of the system in the down wind area of the fabric. Close contact tests do not use any airflow through the sample (in theory the clothing is against the skin preventing air flow). Other tests do not allow for any agent to be evaporated to the environment and all agents are dragged through the fabric (convective flow test). Many examples for both vapor and liquid contamination are available where the flow is in between these two extremes. This confusion makes it difficult for procurement offices to produce technical specifications. One could say that every test has its merits, but it is often very difficult to understand the purpose of the test.

Testing protective clothing has two main purposes: first, to test the encumbrance, and second, to test chemical protection against vapor, liquid drops of various sizes, and aerosols. Biological agent protection is seldom tested. The reason is that there is only one biological agent that acts through the skin, and this agent is much more hazardous through the respiratory route. One would become a casualty through inhalation, even when wearing a mask, long before one would receive hazardous skin doses. In conducting encumbrance and protection testing, there is much confusion about what should be measured and how the measurement should be accomplished. The best method would be to test encumbrance and chemical protection in one field trial; however that is out of the question using human volunteers and chemical agents.

Encumbrance is usually investigated separately by having volunteers in full protective gear perform exercises either in a climatic chamber or in the field. In the more advanced tests, the heart rate, core temperature and skin temperature are monitored continuously. The endurance times, or the time to complete a set of tasks which is used as an indicator of the physiological load, strongly depends on environmental conditions such as temperature and humidity. The general outcome of trials is that the differences between air-permeable fabrics, membrane-based outer layers or completely impermeable outer shells are not significant. There are two important reasons for this finding. One is that the encumbrance due to the mask, gloves and over boots overshadows any differences between the suits. Another reason is that the suits are not evaluated on the afforded protection. It is known that membrane and impermeable suits in particular show a large bellows effect, and the wearer gets cooling from the air that is circulated within the suit. It is obvious that such a bellows effect does not contribute to protection in a positive way. Such tests should therefore be performed by carefully "duct" taping the seals and closures to prevent the ingress of air. It only makes sense to conduct encumbrance tests with systems that provide sufficient protection.

An alternative solution to this problem would be the simultaneous measurement of field protection factor and encumbrance under realistic conditions. One such test set up is available in South Africa were volunteers execute an obstacle course and carry out military tasks while being exposed to a simulant vapor. A less desirable alternative would be to run exposure tests in a climatic chamber normally used for clothing encumbrance trials.

During the last decade, protection from chemical agents has been analyzed using human volunteers exposed to a vapor simulant (for permeable/membrane and impermeable suits) and an aerosol. This approach works well for impermeable and membrane suits as long as the deposition onto the skin is used as the indicator for the protection. The ingress of aerosol and vapor are both physically governed processes and should therefore result in nearly identical protection factors. That this does not hold for permeable clothing is due to the unhindered penetration of the aerosol through the filter fabric, whereas vapor is adsorbed. It could be

argued that the adsorption kinetics of a simulant, typically methyl salycilate as a simulant for mustard gas, is not necessarily identical to that of the agent. By choosing a simulant with physical characteristics as close as possible to those of mustard, the differences in adsorption kinetics can be made negligible. If the result is not convincing, an experimental approach would be to use both mustard agent and simulant exposure on a mannequin and look for differences. If a significant difference is found, a correction factor may be applied in volunteer simulant tests. Mannequin and volunteer tests are carried out in the US, Canada, the Netherlands, the UK and South Africa. The Czech Republic and Switzerland have developed a capability for human volunteer testing.

To summarize, in the paragraphs above the following steps were considered:

- 1. Human volunteer- CW agent-Realistic field conditions
- 2. Human volunteer- Simulant-Realistic field conditions
- 3. Human volunteer-Simulant- Test chamber
- 4. Mannequin-Simulant- Test chamber
- 5. Mannequin-CW agent- Test chamber

The CW agent and simulant could be in the form of vapor or aerosol.

Liquid exposures are more complex due to the interaction between fabric and liquid agent. Liquid exposures are evaluated primarily through mannequin tests. The mannequin is suited up in full protection and exposed to liquid agent of various drop sizes. Currently, the only location where these kinds of experiments are performed is in Porton Down, UK. Most other laboratories involved in testing liquid agents use laboratory swatch tests. The US is attempting to establish a capability with robots that can perform military exercises.

Investigations regarding the protection against liquid drops are primarily carried out on swatches of fabric in a laboratory testing apparatus. Drop sizes vary from 1 to 30 mg of neat or thickened agent. The drops are applied via a syringe or free fall. Pressure can also be applied to the drop. Test cells in which the swatches are mounted range from 1.5 to  $100 \text{ cm}^2$ . Contamination densities range from 5 to 50 g/m<sup>2</sup>. Airflows involved are very different across the various experimental designs. In principle, there are three types of flow. In the first type of flow, air at a typical velocity of 5 m/s is directed to the "outside" of the fabric. The "wind" will cause agent evaporation and most of the challenge will be carried away. In the second type of flow, the "wind" will generate a pressure and a flow through the fabric. The exact flow can be calculated from the wind speed and the resistance of the fabric, but it is roughly 1/1000of the wind speed. When penetrating the filter fabric layer, where the agent is adsorbed, not all agent molecules are adsorbed in the shallow filter, and the skin is exposed to small vapor concentrations. Some tests use a flow underneath the fabric to carry away the agent that has penetrated (flow type 3). This agent is sampled, and the amount determined by chemical analysis making it is possible to calculate a concentration and ultimately a dosage. The problem with this method is that it is assumed that all agents that penetrate are absorbed by the skin. In practice, the rate of absorption by the skin is limited and agent that has penetrated diffuses in lateral directions over the skin and even back to the filter layer where a second chance of being adsorbed occurs. To overcome the problem of measuring every molecule that penetrates, the concept of testing with a polyethylene (PE) film was introduced fifty years ago. A PE film of approximately 15 micron thickness was supposed to mimic the absorption and penetration of the skin. Unfortunately there is a 100-fold variation across the body in the penetration of mustard agent through the human skin. So the PE film mimics just a portion of the human skin. The test method was validated using field trials in which volar forearms were exposed to liquid contamination. The validation tests are described in a report by Dawson and Gilchrist from the Edgewood laboratories. Unfortunately it is difficult to obtain the report, and it is equally difficult to understand the logic behind some of the reasoning. One statement in the report however is very obvious. A swatch was fixed to a volar forearm and contaminated with a 1 mg drop. The time until erythema was first detectable was recorded. Such a drop usually covers a small surface area, less than 0.1 cm<sup>2</sup>, but the generated vapor is spread due to movement of the clothing and due to diffusion. When the same test was done in the laboratory swatch test using a polyethylene film, the breakthrough in the same time span was around  $1 \,\mu g/cm^2$ . This was then used as the basis to arrive at the  $4 \,\mu g/cm^2$  criterion. This was adopted in all NATO documents in the previous century. Several aspects were considered before arriving at the 4  $\mu$ g/cm<sup>2</sup> criterion. One is that a starting erythema is generally not disabling. A second one is that the measurements were carried out in close contact without movement of the clothing. In practice the clothing will move over the skin and distribute the agent over a larger surface. Although the method has been validated, the functioning of the PE film is often not understood.

The function of the PE film is to control the kinetics of the agent penetration process in an attempt to mimic the effects on skin. Agent that has penetrated the filter will be transported toward and absorbed by the skin. The rate of these processes is controlled by the concentration gradient between the inner side of the filter and the skin surface. The rate of absorption by the skin is relatively slow, and therefore the concentration at the surface has a value greater than zero. The rate of transport to the skin is smaller than it would have been if agent was absorbed by the skin at an infinitely high rate and not all agent is absorbed immediately. Some fraction of the agent will diffuse back to the filter layer or spread over the skin surface and is no longer available for absorption on the small spot just below the drop. Efforts to determine the kinetics of mustard agent skin absorption have been attempted in only two instances. Both were carried out during WW II, with one in the US ( by Nagy et.al. Published in the JACS 1946), and the other by Columbine in the UK. These investigations show that the rate of absorption from a vapor source by the skin versus a thin polyethylene film is about the same. So, polyethylene film is a fair representation of skin. The concentration of agent at the upper side of the film is close to the concentration that the skin would experience. It is smaller than the concentration at the back side of the filter but definitely not zero. Knowing this concentration makes it possible to calculate an exposure dosage as a Ct product in the same way as it is computed in the percutaneous toxicity tables.

In order to make the conversion from the amount penetrated on one side of the film to the exposure dosage on the other side of the film, one must know the permeability of the film for mustard agent. This can be determined in a simple experiment in which the film is exposed to a known concentration of vapor during a known period of time and measuring the amount of vapor that penetrates through to the other side. The vapor dosage is usually expressed in mg·min/m<sup>3</sup>. The quantity that has penetrated in  $X \mu g/cm^2$  corresponds to  $10X mg/m^2$ . This last value divided by P, the permeability in m/min (a linear velocity), forms the conversion factor, 10X/P, for quantity penetrated on one side to the exposure dosage on the other side.

For the most common polyethylene films of 15  $\mu$ m thickness, P is on the order of 0.05 m/min. However, thicker films or films of a different quality will show a different value of P. For a correct interpretation of the test result, it is necessary to determine the value of P for the films in use. Using the value of P as given above, the minimal effect exposure dosage of mustard agent according to the NATO Table in Chapter 4 will result in a breakthrough criterion for the PE film of 0.25  $\mu$ g/cm<sup>2</sup>. This is considerably below the criterion of 4  $\mu$ g/cm<sup>2</sup> which was determined under the conditions of risk-taking and not for the minimal noticeable effect for the most sensitive skin.

It is generally considered that a test using a PE film and close contact between fabric and PE film will represent a worst case for agent exposure. An agent drop is exposed to the wind and is subject to evaporation. Agent penetrates and vapor comes in direct contact with the PE film (skin). Because the fabric is pressed against the skin (close contact, e.g. shoulders) there is no airflow through the fabric. The airflow underneath the fabric is irrelevant because the total amount that has penetrated is of most importance.

Unfortunately, there is no film available that mimics the absorption of nerve agent by the skin. Tests under many flow conditions have been developed. Some tests go to the extreme of forcing all agents from a contamination drop through the fabric (convective flow test in use in the US). Although this method might rank the fabrics according to filter capacity, it has little to do with the penetration of agent through a suit in the field.

Nerve agent testing is accomplished using Sarin (Germany), Soman (the agent of choice in the US), and in rare cases Cyclohexyl Sarin. Sarin is a remarkable choice because it evaporates quickly and most of the challenge is carried away with the wind. In addition the permissible skin exposure to Sarin according to the NATO Table (see 4.7) is very high. The use of Soman is also remarkable because there is no proven therapy to be applied to test personnel in the event of an accident. Also, a NATO group of experts noted that there are no human exposure data from which a negligible risk dosage can be determined. Finally the vapor pressure of Soman is considerably higher than that of mustard, and the persistence on the fabric of Soman is much shorter. Cyclohexyl Sarin, GF, was the agent of choice for conducting tests forty years ago because it had the "right" vapor pressure and reliable human data were known. The reason for abandoning GF is not clear. In any case, the human exposure data indicate that for effects the dosage of G-type nerve agents is considerably higher than the effect dosages of mustard agent. There is absolutely no reason for G-agents to penetrate the filter fabric in larger amounts than mustard. Adsorption onto the active carbon is a physical process, and the boiling point of the agent might determine the energy of adsorption. The G and H agents under consideration have fairly high boiling points and are thus well adsorbed by the carbon. In short, a fabric that protects against mustard agent also protects against G-agents. Tests with VX are often performed but the vapor pressure of this agent is so low that hardly any vapor is generated. The point of interest is now the penetration of liquid, possibly enhanced due to fuel spills, rain or sweat. Severe effects might occur at exposures of 2 mg per man. This value should not be converted to 0.2  $\mu$ g/cm<sup>2</sup> because VX, as is the case for G agents, is acting systemically, and the total exposed skin surface must be considered. The varying permeability of the skin over different body sites should be considered as well.

Reviewing protective clothing requirements of various nations, it becomes obvious that the authorities responsible for these specifications have not always received the best advice regarding the test methods and the criteria. Unfortunately, as the scientists continue to argue about the correct test method it is no wonder that the "requirements" authorities become confused.

# 6.4. B. Body Protection/Protective Clothing for First Responders

## 6.4. B.1. Background

A review of CBRN training exercises images will reveal a vast array of protective clothing. Fully encapsulating clothing with clean air supply, the "Michelin man" suits in yellow, green, blue, red or orange, are quite common. White plastic suits often duct taped around the wrists and ankles are another common sight. Only a very few services, such as the London Metropolitan police, use blue suits in which the outer shell is based on a membrane. The suit is air tight, but in non-rainy conditions, it allows some water vapor to pass through the membrane and is said to be resistant to toxic compounds. Ingress of agent is still possible through seals and closures, and an active carbon inner layer has been added to the fabric. The reason for this wide array of protective clothing can probably be found in the quick fix solutions that were adopted when chemical and biological terrorism became a reality. The fire brigades adopted the approach used for chemical HAZMAT incidents. It comprises a fully encapsulating suit with clean air supply and a SCBA. The police force, while preventing contamination of a crime site, often use the white plastic suits in crime scene investigations. The reverse was also thought possible. The white plastic suits also have a function when dealing with liquid spills. Plastic suits have protected medical personnel from contamination of bodily fluids when treating Ebola patients. Almost all military taking part in CBRN exercises are dressed in standard protective gear. (See paragraphs 6.4.A) A relative small group of military, such as those involved in decontamination operations, are dressed in butyl rubber suits, sometimes over undergarments containing an active carbon. With this broad spectrum of protective clothing, the preferred protection for first responders is a legitimate question. The following paragraphs will attempt to answer that question. As it is a relatively new area, references to some newer studies will be provided.

As has already been stated, the challenge faced by first responders is only a small fraction of what soldiers might experience in a CB attack. Most of the vapor and aerosol will have dissipated by the time that the first responders arrive at the scene. It is extremely unlikely that they will face a liquid spray attack. Liquid splashes as the result of puddles will usually only occur in the close proximity of a storage site or truck loaded with agent. First responders will respond to the release site of a biological agent only in the case that the release is announced or pre-attack intelligence is available. Covert biological attack will be detected through the development of casualties and may be days after the release that a previous hot spot might be detected. The signature of a CBRN terrorist incident is very different from a HAZMAT incident. Overall the protection that is good for the military is more than sufficient for the first responders. At present, there is no generally accepted estimate of the challenge level to be encountered by first responders. It can be safely said that the challenge level will be significantly lower than those estimated for the worst case military levels. Vapor and aerosol levels will be at least one to two orders of magnitude lower. Only those who have to operate in the hot zone might receive some liquid contamination if a second device is set off. The challenge levels are such that the military suits provide more than sufficient protection.

The allowable skin exposure levels for first responders are not established. IDLH (Immediately Dangerous to Life and Health) or AEGL (Acute Exposure Guideline Levels) levels are given in publications of the US National Academy, but they apply to respiratory exposure. A zero exposure level has been cited which requires an infinitely high protection factor (the protection factor is the quotient of challenge level and allowable exposure level).

At present, it is best to accept as an allowable exposure level a value of half the minimally noticeable effect level.

Tests for impermeable fabrics are prescribed by the National Fire Brigades Association based on methods developed by ASTM (American Society for Testing and Materials). Unfortunately the only types of clothing regulated are impermeable types defined by Levels A, B and C. Level A addresses a fully encapsulating suit used with an independent clean air supply and a SCBA, Level B is slighter lower in protection but still is combined with a SCBA. Level C is a coverall that can be used with a filter type gas mask and offer the lowest protection. Testing protection afforded by the suits is principally accomplished by exposing the suits to an aerosol for some period of time (up to 15 minutes) and subsequently



In any exercise several types of impermeable suits are used, from Levels A and B down to Levels C and D. Sometimes the first responders facing the least challenge wear the highest protection level whereas those rescuing casualties are protected with plastic suits offering poor protection.

measuring the concentration inside the suit. Protection factors are defined as the ratio in concentrations outside the suit to that on the inside of the suit. The British Standard uses similar techniques. Four issues warrant mention:

- If the challenge level to first responders is one or two order of magnitude lower than that to the military, why is a SCBA necessary for respiratory protection and Level A or B suits for skin protection?
- Does the first responders heavily reliance on impermeable suits imply that the military in the permeable suits are not well protected?
- The higher the required protection level, the longer it takes to don the ensemble. Valuable rescue operation time is lost. Higher protection levels are more costly and thus less widely available.
- Level C types of impermeable suits offer less vapor and aerosol protection compared to air-permeable suits. Protection offered by C level suits rapidly degrades with the wear time.

# 6.4.B.2. Permeation/Ingress

When impermeable materials or membranes are properly designed, there is no permeation of agent through the film or membrane. However it is difficult to find thin films that will block all agents under all circumstances. Some materials provide excellent protection against H, G and V agents but provide just marginal protection against L. Most plastic films, with one or two exceptions, provide better protection against V than against H. The protection provided by one type of material, e.g. butyl rubber, can vary over a wide range depending on the type of rubber, the fillers and the curing of the rubber. Some films and membranes loose their protective properties when saturated with sweat. Sweat composition varies from person to person, and some sweat components can be very aggressive. Organic solvents like those present in petrol, oil or lubricant can destroy the protective properties of membranes and impermeable materials. A well-known example of this is butyl rubber used in the construction of gloves. The materials are capable of stopping mustard agent for days but after contact with diesel fuel the protection time drops below one hour. It is therefore mandatory that the impermeable/membrane systems are tested for all possible agents under all possible user conditions. Most of the materials are produced in batches and the quality of the product can vary from batch to batch.

The most significant problem with impermeable systems arises from the bellows effect. Body motions in an impermeable suit will create a vacuum, and air is forced into and out of the system. Contamination is transported with the air. The situation is identical to the leakage of contamination into a sealed room.

# 6.4.B.3. Theory of protection in a sealed space, e.g. a room or a suit

The differential equation describing vapor/aerosol penetration into non-filtered sealed enclosures has been solved by Helmbold (7). Although the original paper describes the ventilation kinetics in collective protection shelters, the same model holds if the enclosure is a room in a house, possibly sealed with duct tape or clothing, permeable or impermeable, surrounding an individual.

The basic differential equation is very simple:

- Due to leakage there will always be a small flow [F] of outside air into the enclosure with a volume [V]. This air will be loaded with the outside concentration of vapor or aerosol [C<sub>o</sub>].
- As long as the exposure lasts, contaminated air will penetrate, and a concentration  $[C_i]$  in the enclosure will slowly build up. If the exposure time [t] is long enough, the concentration inside the enclosure will approach the outside concentration. During the exposure phase, the concentration in the room is increasing according to  $C_o(1-e^{-F/V.t})$ .
- When air flows in, the same amount of air must flow out. The outgoing air will be loaded with the concentration of contaminant as inside the enclosure  $C_i$ . After termination of the exposure, the concentration in the enclosure  $C_i$  reduces in time according to  $e^{-F/V.t}$ .

The exposure is described as a dosage, the concentration x time integral. The most striking result of this model is that the dosage received inside the enclosure approaches the dosage received outside as the residence time approaches infinity. Dependent on F and V, infinity is not too far away. The protection factor of the enclosure is the ratio of the dosages received

outside to that received inside the enclosure.

The following example calculations show how the protection factor of impermeable systems rapidly degrades over the time the suit is used. In responding to a hazard, it is not unlikely that the first responder initially seeks out the hot spot and experiences an exposure of 5-10 minutes, for example. He might spend another 50 minutes scanning the surroundings. During the exposure phase the concentration inside the enclosure is increases according to  $C_o$  (1-e<sup>F/V.t</sup>). After termination of the exposure, the concentration in the enclosure C<sub>i</sub> reduces in time according to e<sup>-F/Vat</sup>. This simple model, the general solution provided by Helmbold, shows that given time, the concentration inside will approach the concentration outside. The time required depends on the ratio F/V. Unfortunately the rate at which the hazardous compound will be cleared from the enclosure also is an exponential dependence on the ratio F/V. As a result, the dosage (Cxt), to which the wearer is exposed approaches the dosage that would have been received without the enclosure. In the above simple model, it has been assumed that the mixing inside the enclosure is fast. This is a reasonable assumption considering the likely body motions and the bellows effect of the clothing.

The concentrations as a function of time both inside and outside are given in the figure below. The dosage is represented by the total area underneath the two curves. It is obvious that the two areas underneath the curves become equal when the time is long. The simple model also assumes that the only route for lowering the concentration is ventilation of the suit and that the ventilation remained the same after exposure has ceased. This is obviously wrong as skin will absorb agent and thus additionally lower the concentration. In view of the low ventilation rate from the inside of the suit, it would be a better assumption that all the agent that has leaked in will be absorbed by the skin. The determining parameter is then the amount of agent that has leaked into the system. This does not address the issue completely because both for locally acting agents like mustard and systemically acting agents like the nerve agents, the penetration rate into the skin depends on the site of the bodily skin involved. Some skin parts (groin) are very sensitive whereas others (palm of hands) are very insensitive.



6.4.B.4. Protection factors degrade with time

The model discussed above makes assumptions about the inner volume and the inward flow as well as the concentration of agent. It is however quite easy to measure the protection factor over a 5 minute exposure, actually determine the inward leakage, and from there derive the degradation in time using the model. A very well duct taped C type of suit showed in a 5 minute exposure test with a stimulant with a concentration of 500 mg/m<sup>3</sup> simulant a
protection factor of between 1500 and 2000. Using the model and with the assumption that nothing is absorbed, the following dosage and protection factors are found inside the suits at the indicated times.

After the initial high protection factor in a six-hour working period, the protection factor has degraded nearly two orders of magnitude. This degradation will always occur.

| Time (min) | dosage inside | Protection Factor |
|------------|---------------|-------------------|
| 5          | 0.027         | 1800              |
| 30         | 0.25          | 230               |
| 60         | 0.51          | 110               |
| 120        | 0.98          | 60                |
| 180        | 1.41          | 40                |
| 240        | 1.80          | 30                |
| 300        | 2.15          | 25                |
| 360        | 2.47          | 23                |

Protection factors of 1800 are attainable only in cases where the inward flow is extremely low. Duct-taped Tyvek® suits only achieved a protection factor of 100 in a 30 minutes exposure test indicating that the inward flow was on the order of at least 0.02 l/min. If no duct tape was used, the protection factor dropped to values around 25 indicating an inward leakage of about 0.1 l/min. The amount that leaked in under these conditions would be 12.5 mg. While the test would not be representative of VX because the vapor concentration is too high, it could certainly have simulated GD or GF. Severe effects are expected at exposures to 200 mg/man (see Chapter 4) so the 12.5 mg might induce minimal noticeable effects. The skin around the neck and cheek is much thinner and leakages around the hood are therefore much more hazardous.

In the above case, if the agent had been mustard the 12.5 mg corresponds to less than 1  $\mu$ g/cm<sup>2</sup> when divided evenly over the surface of the body. It is however, obvious that certain parts of the body, close to the suit openings, will be exposed to more agent and serious erythema might develop.

Another example of the low protection factor offered by plastic (taped suits) is given in the diagram below from an experiment carried out in the Swiss Spiez laboratory in cooperation with the German Army. (Karola Hagner of the WIS in Munster was the project leader and kindly gave permission to use the diagram)

In a two hour test, the dosage received close to seals and closures for a well taped Tyvek suit worn by a human volunteer was 10-20 times higher than the exposure dosage for the well protected sites far away from seals and closures. Once the inside of the suit is cleaned using a filter blower unit, the protection dramatically increases. Actually what the experiment clearly tells is that level A and B types of impermeable suits provide good protection, comparable to or slightly better than the permeable protective garments. On the other hand, the impermeable suits that are not equipped with a system to clean the inside provide very poor protection.



The inward leakage for a good Level C suit with an initial protection factor of 25 is such that an appreciable quantity of agent reaches the skin and effects might become visible. The amount of agent that reaches the skin is about half when an additional active carbon layer is worn. Over a six hour working shift, the protection factor will drop to a value of 2. These modeling values have been substantiated with test data accomplished by Lindsay (8) and from measurements in South Africa (9). In both cases, the protection factors were on the order of 2 or below.

As shown in the paragraph 6.4.A., permeable suits offer excellent protection against vapor. A vapor dosage challenge of 5000 mg·min/m<sup>3</sup> (1) is reduced to a 500 times lower dosage. Protection against C and B aerosols is irrelevant because the amount deposited onto the skin is small. Protection against neat liquid drops is very good, and protection against thickened liquid drops is regarded as sufficient. Since the likelihood that first responders will be contaminated with thickened liquid CWA is extremely small, the military-type suit offers sufficient protection and is better than the level C type of suits. The protection afforded by permeable suits is regarded sufficient against a far higher challenge than a first responder is likely to encounter. The level A and B type suits offer protections comparable to the military type suits but require a much longer donning time. For first responder casualty mitigation when time is a factor, it would appear that military-type suits might offer the best option.

An advantage of air-impermeable clothing is that showering can easily wash down the external chemical and biological contamination in comparison to more complex decontamination accomplished during the removal of a permeable suit. Elimination of

external contamination reduces any secondary contamination during suit removal. It must be noted that the majority of contamination deposited on the skin and underwear is the result of ingress of agent through seals and closures. This is significantly more for impermeable clothing than for permeable clothing. Simply decontaminating the outside barely reduces the re-aerosolisation hazard.

It must be stressed once again that it is difficult to achieve effective CB protection using impermeable clothing. Illustrative is a whole system test conducted in South Africa. (Eloff, C.G. and Claassen, N., Field Evaluation of Chemical Protective Suits, Protechnik Laboratories Report no B65.6/1/2004, Centurion, South Africa, 2004.) Volunteers wearing impermeable protective clothing accomplished three trials through an obstacle course. Each trial required 15 minutes with a 10 minutes rest period between trials. The volunteers were exposed to methyl salycilate vapor during the navigation of the course. The physiological load experienced in their trials was not significantly different from that of a permeable suit, perhaps because mask, gloves and boots contributed to the load. The protection offered by the impermeable suits (membrane type) was very poor. One of the likely explanations was that during rest periods, the volunteer did not move, generated no pumping effect and did not pump out the contamination that had entered during exposure. The results might have been more realistic if the suit would have been ventilated during rest periods. This was not considered an option in a contaminated area. Despite a very elaborate duct taping job, it did not appear possible to maintain a seal. The duct taped seals often detoriated while navigating the obstacle course.

The interpretations of field tests as described above are always hampered by the fact that many parameters can have an influence on the result. Controlled exposure tests in which the exposure is carefully controlled as well as the temperature, wind and humidity, with human volunteers in an exposure chamber might be more meaningful.

In such a test, four volunteers underwent two trials in an exposure chamber, once wearing untaped permeable and once wearing impermeable suits. In addition, four volunteers underwent two trials wearing taped permeable and impermeable suits. There was no special provision for taping the mask. The results indicate that the leakage around the mask hood seal is definitely smaller than that at the sleeves and legs, but it is not known how the ingressed agent distributes over the body. Tests on four volunteers with a special sealed mask were run as well. Due to the variation in the test, the fact that unexpected exposures were observed, and the artificial nature of the exercises during the exposure, it seems wise not to rely too much on the absolute value of the protection factors that were measured. The relative scale results will be presented in Table 6.1. The lowest PF observed is labeled "X" and all other will be multiples of X. In the figures below, the values of the PF are averaged over 8/8/4 tests, untaped/taped arms and legs, taped on mask as well for filter and, barrier type suits.

As could be expected, the un-taped suits resulted in the lowest protection factors. The lowest value is taken as X. The first column represents an un-taped suit. The second column presents data for a suit taped around wrists and ankles. The third column presents data for a suit taped at the wrists, ankles and mask.

The value of X in this case is below 10. In none of the mannequin or human volunteer tests, a higher value was observed. Taping helps improve the protection factor but not by more than a factor 2. Additional taping around the mask improves the result further, but the protection

factors remain in the range of 4-11 X. These protection factors are in general too low to provide military with sufficient protection against vapor.

Table 6.1 Relative Protection Factors for Impermeable Suits in Three Configurations, Averaged Over Different Body Parts.

| Body Part            | Not Taped | Taped Wrists/Ankles Tape | ed/Wrists/Ankles/Mask |
|----------------------|-----------|--------------------------|-----------------------|
| Necks                | 3X        | 2.2X                     | 4 X                   |
| Shoulders, Neck back | 4X        | 6 X                      | 8 X                   |
| Shoulders front      | 6X        | 5 X                      | 8 X                   |
| Bodies               | 7X        | 9 X                      | 11 X                  |
| Lower arm            | Х         | 5 X                      | 2.5X                  |
| Upper legs           | 5X        | 13X                      | 9 X                   |
| Lower leg, front     | 3X        | 4 X                      | 5 X                   |
| Lower leg, back      | 2X        | 6 X                      | 5 X                   |

Whether the protection factors are sufficient for first responders is dependent on the expected vapor exposure levels. In this respect, it is of importance to note that military accept a small risk in their operations whereas first responders operate under a zero risk principle. Consequently, accepted agent effect levels are somewhat higher for military personnel than for first responders. For equal challenges the required protection factor for a first responder is higher than for a soldier.

Table 6.2 presents protection factors for filter-type suits in three configurations averaged over different body areas. The first column presents an un-taped suit. The second column represents a fully taped suit. The third column represents a filter-type suit with improved seals and closures, and the reference protection factor is the same as that cited for the lower arm in an un-taped impermeable suit, X (see Table 6.1).

Table 6.2. Relative Protection Factors for Filter Type Suits.

| Body Part<br>Design  | Not Taped | Taped Wrists, Ankles and Mask | Improved |
|----------------------|-----------|-------------------------------|----------|
| Neck                 | 80X       | 40X                           | 60X      |
| Shoulders, Neck back | 270X      | 850X                          | 500X     |
| Shoulder front       | >1000X    | 150X                          | 300X     |
| Body                 | 400X      | 190X                          | 300X     |
| Lower arm            | 40X       | 140X                          | 150X     |
| Upper legs           | 130X      | 260X                          | 400X     |
| Lower legs front     | 800X      | 250X                          | 400X     |
| Lower leg back       | >1000X    | 730X                          | 600X     |

The lowest PF observed for the filter-type suits is generally above 50X. In some cases, no agent was detected inside the suit, and the protection factor was reported as above 1000X. The PF for taped and un-taped configurations does not differ very much for the filter-type suits. Obviously the adsorption onto carbon is effective in reducing the ingress of agent. For the improved design suit, the protection factor is above that of the un-taped suit but in the same range as the taped suit. Whether such a design is required again depends on the expected

exposure levels. For the military, the PFs meet the essential required protection level and often even the desired protection level.

# 6.4.B.5. What is the preferred protective clothing for first responders?

In the beginning of paragraph 6.4.B, a legitimate question was raised as to what should be the preferred protection for a first responder. In answering that question, the results of the tests presented above speak for themselves. The primary weak points in impermeable suits are the openings, especially around the hood/mask, at the sleeves and the legs, but others contribute as well. Taping improves the protection factor but certainly not by two orders of magnitude. At most taping improves protection by a factor 2. Due to the decreased bellows effect and the presence of active carbon, the permeable suits provide better protection against vapor by 1.5 to 2 orders of magnitude.

It might be possible to improve the seal of impermeable clothing, however, with the absence of air exchange, the cooling due to the air exchange and the loss of water will be retarded. The suits will definitely become more uncomfortable, and the endurance time will be reduced. Another point is the integrity of the seals can they be maintained during operational use.

# The results support the military choice for permeable suits.

For first responders, the most likely exposure will be to low residual vapor concentrations, being lower than those for the military. The required or desired protection factor for first responders will be in the same range, indicating that a permeable suit might be the best choice. It is a general misconception that one could penetrate the protection of permeable clothing by challenging the clothing with an aerosol, a so-called dusty agent. A summary of an earlier publication makes it clear that this is not the case. See Annex B to Chapter 6.

# **6.5 Collective Protection**

There are many different types of collective protection shelters, some are in vehicles or ships, others are man-transportable and some are hardened underground structures. The purpose of collective protection for the military is to provide toxic-free areas that allow relief from the burden associated with wearing individual protection. Sometimes military functions cannot be performed wearing individual protection, and a shirtsleeve environment is mandatory, e.g. command and control centers, medical facilities and complex weapon platforms. All these functions have one thing in common: a closed space kept under positive pressure through the supply of clean air. Another type of collective protection exists that does not provide the clean air positive pressure feature. Applicable mainly to the civilian population, this protection is provided by closing windows and taping openings to improvise a sealed enclosure to the maximum extent possible. The latter type is often practiced to protect civilians in case of a HAZMAT incident in a developed area or in the case of fires with the possible release of dangerous compounds. Unfortunately, the protection that is provided in a sealed room is not as good as many people think. This type of collective protection will be discussed first, followed by a description of the many types of collective protection in use with the military.

# 6.5.1. Sealed room

Ingress of agent into a sealed room cannot be prevented. Experiments have shown that the volume of an ordinary room is exchanged with fresh air about ten times per hour. By taking

some precautions, it can be reduced to one air exchange per hour. By carefully taping all openings and selecting the room that is situated in the downwind direction, the exchange rate can be reduced to 0.1 times per hour. Usually the high concentration section of an agent cloud passes the house in a few minutes, and only a small amount of agent will leak into the room. However, as long as the room stays closed, the concentration that has leaked into the room will linger and be reduced very slowly because of the low exchange rate. After some time, the dosage, expressed as C x t inside the room approaches the dosage that would have been received outside the room. The protection provided in a sealed room rapidly degrades as time passes. Fortunately there are some effects that can mitigate the low protection factors. The most obvious would be to shorten the time of residence in the room as much as possible. As soon as the agent cloud has passed the windows can be opened to let fresh air enter the room. This would require an adequate detection capability of the first responders. Another mitigating effect can be found in the adsorption of agent onto the walls and furniture of the room with the result being a further lowering of agent concentration. Aerosols will not be easily adsorbed but the tortuous path encountered in leaking into the room might filter out a significant portion of the aerosol. By closing the windows and taping the openings, one can keep out of the house most of the smoke resulting from an exterior fire. Perhaps the best mitigating effect can be found in nature itself. Most volatile compounds, if not all, follow the toxic load concept. This concept theorizes that harmful dosage is not C x t = constant (Habers rule) but it depends on concentration and exposure time according to  $C^n x t$  =constant. Chlorine and HCN show exponents n above 3, where a nerve agent such as Sarin shows a toxic load exponent of 1.5. As a result lower concentration over prolonged exposure times are far less hazardous than short term exposures to high concentrations.

It should be remembered that protection inside houses and sealed rooms is not very good, and even non-existent when the source is of long duration. When the source releases agent at a slow rate over a period of hours, the concentration inside the room will approach the concentration on the outside, and the protection factor is just slightly above 1.

A somewhat unorthodox method of providing better protection in a sealed room is to fill the collection container of a vacuum cleaner with active carbon and have the vacuum cleaner operating inside the room, cleaning the air while recirculating. The same can be achieved with a kitchen recirculation hood equipped with an active carbon filter.

## 6.5.2 Theory of protection in a sealed room

The theory of ventilation kinetics in collective protection shelters has been described in paragraph 6.4.B.3. Here, some relevant sample calculations for a simple scenario will be given. A possible scenario is a short duration cloud present 5 minutes (at a wind speed of 3 m/sec, such a cloud has a length of nearly 1 km). For simplicity it is assumed that the concentration throughout the cloud is a constant 1000 mg/m<sup>3</sup>. During the exposure phase, the concentration in the room will increase according to  $C_o(1-e^{-F/V.t})$ . After termination of the exposure, the concentration in the enclosure  $C_i$  reduces in time according to  $e^{-F/V.t}$ . As time passes, the dosage inside will approach the dosage that would have been received outside, see figure in paragraph 6.4.B.3

## 6.5.3. Remedies

The simple model shows that the protection inside a sealed room, sealed in an improvised way, e.g. with duct tape, is relatively poor. For short exposures to contamination, this

approach might provide some protection. However, it is usually very difficult to warn people of the onset of a short duration cloud. The cloud will pass in a few minutes and travel down wind dependent upon wind speed.

Some remedies that might enhance the protection inside houses include "opening" up the room as soon as the cloud passes, and/or residing on the highest floor of the building. These two suggestions are official practice in the Netherlands. (Green Book, Ministry of Social Affairs and Employment, the Netherlands)

A controversial option would be to switch on the air conditioning. Contrary to general beliefs, this might offer a better reduction of the exposure than sealing the room because the compressor will condense water and simultaneously agent from the air.

The most important contribution to protection inside sealed rooms is found in the toxic load concept as described in Chapter 4 and the preceding paragraph. Many volatile industrial compounds show a value of n as large as 3 or even higher. This means that when the concentration inside the house is 1/10 of that outside the house, the exposure time must increase 1000 times in order to arrive at the same effect. So in most industrial accidents with short duration exposures staying inside an improvised sealed room offers a fair protection.

Less volatile agents usually have lower values of n, but recently it has been established that nerve agents also follow the toxic load concept with a value of n in the order of 1.5 for exposures in the range of 1-100 minutes.

## 6.5.4. Collective protection with clean air supply

A basic problem for collective protection is to keep the area free of toxic agents when in use in a contaminated environment. In order to achieve this, entry/exit systems have been developed consisting of contamination control areas and airlocks. The first step in a contamination control area is to remove liquid contamination either by decontamination or by removal of the outside layer of clothing. This is termed the liquid hazard area. The next step is to prevent vapor from being carried in by persons entering. This is termed vapor hazard area. After each step an airlock is established to flush the area. Entering personnel remain in the airlock for a prescribed time to reduce as much as possible the contamination that is brought into the toxic-free area. Small amounts of agent might still be adsorbed for instance by body hairs and be brought into the toxic-free area. It is therefore essential to flush the toxic-free area with clean air and in order to prevent ingress of contamination to maintain positive pressure. The air filter should be in excellent condition to prevent any ingress of contaminant. It might be beneficial to have additional recirculation filters or any other type of adsorbent installed in the toxic-free area to scavenge traces of contaminant that have ingressed into the system.

It is clear that the procedures described above are very complex and can only be applied in full to hardened collective shelters such as command posts and other fixed site locations. These rear area targets only face attacks with persistent agents, which have by definition a low vapor pressure. The problems of keeping the toxic-free area clean would be greatly reduced if the contamination is mapped and the contaminated area was avoided as much as possible.

At the other end of the collective protection spectrum are the vehicles used on the battlefield, such as tanks and armored vehicles. When the vehicle is sealed before the attack, the filter installation will maintain a positive pressure and keep the inside relatively clean. However, when the vehicle is moving at high velocity, the increased air pressure will enhance the ingress of agent and the protection factor which might be sufficient while motionless will be reduced significantly.

# 6.5.5. Protection factors

The dosage received by personnel inside a collective protection can be derived from a complex differential equation which includes the penetration of the filters, penetration of the construction, transfer of contamination by entering or exiting personnel and the removal of agent by the flow of clean air or recirculation filters. Solving this differential equation yields that the dosage C x t = mg of agent ingress / air flow in  $m^3/min$ . For a well-constructed and well-filtered shelter, the main route of agent ingress will be by entering personnel. As an example, if 100 people enter a shelter and the contamination on each is reduced from the original few grams/man to 1 mg/man with an air flow of 10 m<sup>3</sup>/min, each person will experience a dosage (C x t) of 10 mg·min/m<sup>3</sup> assuming that all of the "off gassed" agent is desorbed while the person is in the shelter.

For the case of biological agents, the same differential equation can be applied by replacing "mg of agent" with "number of particles". Defining the protection factor becomes somewhat more difficult. It is highly unlikely that all the particulate matter on personnel will become air-borne again. In addition the infectivity of a biological agent strongly depends on the particle size, the smaller particles being by far the most infective. The particles outside the shelter are likely to be of a larger particle sizes and distribution than those transported inside. Relatively little is known about these aspects and defining protection factors is certainly not based on science.

# 6.5.6. Encumbrance of collective protection

The atmosphere of an inhabited collective protection will quickly degrade if the clean air flow is reduced appreciably. Carbon dioxide builds up, oxygen is depleted, odors accumulate and fungal growth is possible. Temperatures might increase to unacceptable levels. Without the facilities to maintain the air quality, occupation of such a shelter can be likened to that in a submarine.

Problems are reduced for systems with built-in collective protection, such as ships and armored vehicles. In the past, filter exchange was often mentioned as a point of major concern. But with the present day threat picture, it is unlikely that filter exchange will ever be necessary except for perhaps operations in a dusty or dirty environment.

The primary role of collective protection is to provide rest and relief to the soldier in a chemical or biological environment. This requires the transportation of liners, tents, air filtration units, entry-exit locks, as well as the storage of equipment, air conditioning, connection hoses, power generators, lighting etc. This requires the use of trailers, trucks and drivers, which are all desperately needed for other wartime tasking. The overall consequence is that the logistic burden is too high, and this form of collective protection suffers from the "leave at port syndrome" or "leave at the first available dumping ground" mindset.

Another serious problem is the time required to safely enter and exit the collective protection. Even with two air locks, one for ingress and one for egress it will take more than one hour to offer rest and relief to a single platoon. This does not account for the personnel required to establish and maintain the system.

# 6.5.7. Alternatives.

In view of the changing geopolitical situation, military personnel are likely to operate typically under UN mandate in the world's "hot spots" as opposed to on their home territory. Chemical or biological attacks will likely be incidental, if occurring at all, and will be localized to relatively small areas. In these theatres of operations (Afghanistan, Iraq, Bosnia, Africa), there are no hardened shelters on fixed sites. Air operations to a large degree may commence from aircraft carriers, which have colpro facilities. If operations are carried out from land bases, mobile, light-weight, possibly improvised collective protection must be provided. Command posts might be established in containers equipped with filter installations and air conditioning. For collective protection, the most demanding tasks occur with field hospitals and rest and relief stations. If possible they should be established in a toxic-free area were collective protection is not required. When that cannot be guaranteed collective protection is required. It should be noted that the availability of individual protective equipment forms a deterrent for the use of chemical or biological weapons. When protective equipment is used properly by trained troops, mass casualties will be prevented and most of the CB attacks by the aggressor will not be worth the effort.

## 6.6 Buddy care

Although not a part of physical protection, there is one protective measure applied by the individual or his buddy. When the symptoms clearly indicate a nerve agent attack, auto injectors will be used to inject atropine and other medicines into the bloodstream, giving the



When a soldier has collapsed during a nerve agent attack, his buddy will use up to three auto injectors containing a cocktail of basically atropine and valium. Sometimes complex oximes are added which help in the treatment of the effects of certain nerve agents. Arriving at the battalion aid station, the victims get a complex and timeconsuming decontamination before they enter the toxic-free environment of the medical treatment facility. The entering procedure could be speeded up by covering the victims with an active carbon containing blanket, which will effectively adsorb the gases that emanate from the patient.

victim more time to reach a battalion aid station for further treatment. The contents of the auto-injector will be described in the next chapter.

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# Annex A to Chapter 6: The Threat of Biological Aerosols to Skin

To illustrate the possible threat, anthrax is used as an example:

The first question to be answered is what will be the exposure. The answer is that the maximum relevant exposure is equal to the dosage that kills when wearing a gasmask. As equation:

Exposure dosage = respiratory  $LD_{50}x$  field protection factor of mask.

Recent studies in the US have shown that the respiratory  $LD_{50}$  for anthrax is 55,000 spores inhaled.

A concentration of 1000 spores/l or 1 million spores/m<sup>3</sup> is required to be exposed to an  $LD_{50}$  dosage if not protected by a mask, assuming an inhalation rate of 27.5 l/min. and exposure time to a cloud for 2 minutes. The exposure dosage expressed as C x t = 2 million spores.min/m<sup>3</sup>. If a mask is worn, the required dosage increases proportionally to the protection factor offered by the mask

Protection Factor mask  $10^3 10^4 10^5$ Dosage required  $2x10^9 2x10^{10} 2x10^{11} sp.min/m^3$ 

A mask offering a field protection factor of 10,000 makes the required exposure dosage as high as 20 billion spores.min/ $m^3$ 

## For exposure to this dosage, how much is deposited onto the skin?

Elaborate experiments in the US with the Man in Simulant Test (MIST) and at TNO in the Netherlands have shown that deposition onto the skin is low. The deposition is expressed in terms of a linear deposition velocity

Particles/ unit skin surface area = Exposure dosage x linear deposition velocity or  $sp/m^2 = sp.min/m^3 x m/min$ 

For a standing man just below 1 m<sup>2</sup> skin is exposed

Linear deposition velocity for aerosol penetrating through NBC fabric = 0.001-0.0001 m/min Number of spores deposited on 1 cm<sup>2</sup> skin becomes:

| Field Protection Factor | Mask | $10^{3}$ | $10^{4}$ | $10^{5}$ |
|-------------------------|------|----------|----------|----------|
| 0.0001 m/min dep. Rate  |      | 20       | 200      | 2000     |
| 0.001 m/min dep. Rate   |      | 200      | 2000     | 20,000   |

## How much is hazardous on the skin?

There are only estimates of this value, most likely it is >100,000 spores/cm<sup>2</sup>, so a very high respiratory protection factor is required before exposure to anthrax clouds become a skin problem. There are very good therapies for skin anthrax. No victims of the skin exposures in the US letter incidents died or showed effects five years after exposure.

## The effect of an aerosol filter layer.

Exposing protective clothing to an aerosol results in the following effects. A small fraction is deposited onto the outside of the garment, but the major part is transported through the fabric, with an airflow dictated by the wind pressure. Again a small fraction is deposited onto the skin but the majority of the agent leaves the suit on the downwind side. In this situation, the respiratory hazard during doffing comes from the agent deposited onto the outer fabric.

The process changes somewhat when an aerosol filter is built into the clothing. Agent is still deposited onto the outside, but the larger fraction of the agent is now taken up by the filter and is held by weak physical forces. The dosage to the skin is now smaller but so is the airflow. It is difficult to predict what happens at the lower airflow but it is not unlikely that a larger fraction albeit from a lower concentration is deposited onto the skin. It is certain that the major part of the challenge is now in the filter. During doffing, this can generate a serious respiratory hazard.

Under all circumstances, a filter layer will result in decreased air permeability. It has been demonstrated that the amount of vapor that penetrates through the fabric and is absorbed by the skin is 10% or less of the amount that entered the suit through seals and closures. Increasing the air resistance of the clothing results in a stronger bellows effect, pumping in the clothing, and therefore an increased ingress of agent through seals and closures. The aerosol entering this way is less susceptible to the airflows inside the suit and deposits onto the skin with a higher efficiency. To keep the aerosol deposition onto the skin small, it is mandatory to have a fairly high air-permeable fabric.

# Annex B to Chapter 6: The Myth of Dusty Agent.

From time to time, a special form of CW agents, liquids absorbed into fine dust, is mentioned as particularly hazardous. Some believe that the agents in this form result in more severe effects than the undiluted liquid agents. An example of such publications is the October 2002 report from Eric Crody, Senior Research Associate at the Centre for Non-proliferation Studies. It is a five page report entitled "Dusty Agent and the Iraqi Chemical Weapon Arsenal" with an impressive 30 references.

However, except for the first one or two references, news items in journals and the New York Times, no other reference has anything to do with dusty agents. Neither have the colorful pictures. Unfortunately the most important remarks such as" *Not only do dusty agents increase the amount that can be spread across an area, they can also frustrate and defeat chemical-protection measures*" are not referenced and for good reasons. They are absolute nonsense. Dusty agent is 50% non-agent and aerosols are more difficult to disperse in the atmosphere than liquids or vapors. It would be quite a miracle to create higher concentrations with an aerosol 50% less agent than with liquids or vapors of 100% agent. Another myth mentioned in the same sentence is the frustration and defeat of chemical protection. Based on conclusive scientific experiments, it will be shown that dusty agents do not frustrate or defeat chemical protection.

For all dusty agents, such as blister or nerve agents, a few general facts hold:

- The agent is diluted by a solid so at most 50% of the payload is real agent.
- The agent does not change its physical characteristics. Due to the forces involved in ab(ad)sorption, the vapor pressure might be somewhat smaller.
- No toxicological studies of dusty agent have been carried out. Nowhere in the serious literature is an enhanced toxicity mentioned. A US report on toxic effects from blister and nerve agents does not mention any particular toxicity for dusty agents (Reutter/Wade, see also Review of Acute Human Toxicity Estimates For Selected Chemical-Warfare Agents, Committee on Toxicology National research Council, National Academic Press, Washington D.C., US, 1997.) The recently distributed toxic effect levels as provided by NATO do not mention any particular toxicity for dusty agents. Nowhere is a toxicological process mentioned that could explain the enhanced toxicity. Dusty agents could form a particular hazard because the fine dust containing agent are in the form of an aerosol and will penetrate the active carbon filter layers in protective clothing. In this respect it should be remembered that it is not the amount that penetrates but the amount that penetrates leaves the protective clothing on the downwind side.

Searching the literature of the past 200 years regarding mustard agent, dusty mustard has been mentioned in the past 70 years. The first mention discusses the eastern desert of Libya (1937/38). Italian chemical officers note that the persistence of mustard is considerably increased when the mustard is mixed with fine desert dust. This is not amazing. Besides a slightly lower vapor pressure, the evaporation in the hot desert is reduced mainly because the agent is covered with dust. This information was exchanged with the Italian ally of those days, Nazi Germany. The Germans procured a load of dust from the Italians. In 1943 experiments with the mustard loaded dust were undertaken in the Kaiser Wilhelm Institute in Berlin. The archives (of the Wehrmacht in Freiburg Germany) did not reveal how the

experiments were carried out or whether it was test animals or humans that were exposed to the dusty agent. If the exposures did take place, they would comprise the only toxicological study. In 1944 the experiments were terminated for a remarkable reason. Those conducting the experiments were dressed in impermeable, rubber-type of suits and developed serious effects (erythema and blisters) around the wrists.

After the defeat of Nazi Germany, the US Army Medical Corps was tasked to interview German personnel who were involved in chemical and biological warfare. What happened subsequent to those interviews is unclear, but some 25 years later the topic of dusty agent appears again, and is stated with certainty that the dusty agent is more hazardous than pure compound. In retrospect it is not unlikely that the analyst who came to this conclusion assumed that impermeable suit provided good protection and that the minute amounts of agent which ingress into the system must be very potent to cause blistering of the wrists.

In recent publications (Oudmayer, CBDE Scientific Conference Aberdeen, November 2006, Medema 5th SISPAT conference in Singapore November 2006) an alternative explanation for the 1944 findings became apparent. Due to the bellows effect in all impermeable types of clothing, there is a serious ingress of agent along the wrists and the ankles. Applying extra duct tape improves the protection factor somewhat but it remains low. In none of the realistic tests with human volunteers was a protection factor above 10 detected for the wrists. When working with appreciable concentrations of dusty mustard aerosol, it is highly likely that blisters will occur at the wrists because of the ingress of agent and sweaty skin. On the other hand, the active carbon-based permeable suits showed very good protection factors of at least 1 if not 2 orders of magnitude better than the impermeable suit.

The message for today's military and first responders who might be involved in chemical incidents is: Do not trust your impermeable suit unless there is a mechanism at hand to clean the inside air. Even then the common military active carbon based suits are preferred.

# 7. Medical Countermeasures

# 7.1. Introduction

To protect against chemical warfare agents, physical protection measures, such as an NBC mask and protective clothing, are the first choice since prevention of intoxication should be the primary aim. Medical countermeasures are required if physical protection is absent or inadequate.

The recognition of a chemical victim can pose a problem. In WW I, medical officers had difficulty establishing whether they were dealing with victims of chemical warfare. This resulted from a lack of experience with such cases. Recognition by medical personnel of the signs and symptoms of chemical agent intoxications is essential for the adequate treatment of victims. Victims of intoxications by cyanide or a nerve agent require prompt medical treatment as such poisoning may rapidly lead to death. Intoxications by lung-damaging agents or mustards may be accompanied by a quiescent period before the nature and severity of the intoxication manifest themselves. To overlook this possibility presents a considerable danger to the victims, as they may be intoxicated with a potentially lethal dose.

On the battlefield the use of chemical agents can be suspected if there are victims without obvious wounds, or when a patient is incapacitated more than expected from a wound. Furthermore, a sudden unexplained increase in the number of patients should give rise to the suspicion of chemical warfare. In order to establish whether or not the patient is a chemical casualty, several facts must be established, e.g. whether the patient was wearing full protective gear, whether chemical agent detection equipment responded, whether bombardments were performed and a spray, mist or smoke was observed.

The next step is to identify the agent. This is based on the subjective effects the patient experiences, e.g. irritation of the eyes, feeling of tightness in the chest, difficult and rapid breathing, sudden runny nose, and muscular weakness. In addition, the possible existence of a delay between exposure and the onset of symptoms has to be established. Important aspects are whether the experienced effects persisted after using a gas mask, whether the patient has used an auto-injection device, and if so, the results this has had on the effects.

An estimation of the dose of the agent is made by evaluation of the duration of the exposure, whether the patient was resting or exercising and whether he was in the open field or under cover.

Based on the information received, decisions on the treatment are made.

In modern chemical warfare, it should be recognized that diagnosing chemical victims on the basis of signs and symptoms will be difficult if both the patient and the medical personnel are fully dressed in protective gear. In order to overcome this problem, various analytical-chemical and immunochemical in vitro diagnostic tests have been developed to assess the identity and extent of exposure to several CW agents. This is accomplished by analyzing blood, urine and/or skin samples from the victim. Currently, these tests are applicable in a well-equipped field laboratory. In the coming years, efforts will be made to modify the in vitro diagnostic tests into field kits, which can be used on the fields, closer to the patient.

There is a risk that medical personnel may become contaminated by the patient, therefore, decontamination of the patient and use of the appropriate physical protection measures by the medical attendants is essential.

The situation after terrorist use of CB agents against civilians might be even more complex. Physical protection and detection systems will in most cases be absent during the first stages of the incident. Consequently a larger number of victims might result, and the primary method to mitigate effects of CB will be adequate medical response. More so than on the battlefield, it will be of importance to understand the symptoms caused by the various agents and how to treat them. Medical personnel should have readily available information at hand that makes it possible to discriminate between the agents and to apply the proper therapy. This is important because it is likely that the identity of the agent will be unknown during the first hour after the release. As part of the counterterrorism program, hospital laboratory facilities should be established that can quickly analyze blood, urine, and skin samples from victims to identify the agent and support the staff in choosing the proper therapy. A close network should be established between the laboratories in order to obtain information by the most expeditious path. Adequate quantities of therapeutic means should be available. Most chemical agents act rapidly and minimizing time to treatment is a critical factor. As an option to time-consuming decontamination, protecting medical staff with a light oronasal mask and goggles should be considered. Decontamination is important but could be postponed until a first diagnosis and therapy has been applied to the victim. An alternative approach would be to cover the victim with an activated carbon-containing blanket and cover head hairs with an activated carbon containing-cap in order to adsorb the gases given off by the contaminated victim.

The importance of identifying the agent in order to apply correct therapeutic measures was demonstrated in the Moscow theater incident. The medical staff was not informed about the type or identity of the agent that was used and could do little to save the patients. Information became available at a later time but too late for several of the victims. Another example is the treatment of Iranian mustard agent casualties in Europe in the late 1980s. In one case it was assumed, without any proof, that the casualties were exposed to a mixture of mustard and T2 toxin. The therapy for a T2 toxin poisoning worsened the condition of the casualties.

In this chapter, medical countermeasures against the various types of chemical agents, as well as some riot control agents, will be discussed. For some agents, specific antidotes are available, whereas for other agents, only a general treatment of the life-threatening effects is possible.

Some information is provided on medical countermeasures against several biological warfare agents (mainly toxins), which are considered to be relevant threat agents.

The information presented below is for medically oriented personnel and might be somewhat lengthy for the layman.

# 7.2. Lung Damaging Agents (Choking Agents)

Lung damaging agents include chlorine, phosgene, PFIB and chloropicrin. The mechanism of action and pathophysiology are described in Chapter 4, "Toxicology and Human Toxicity Estimates".

## Signs and symptoms

The onset and intensity of symptoms depend on the concentration of the inhaled toxic vapor and the duration of exposure. During and directly after exposure, symptoms which may occur are pain in the

upper airways during breathing, pain in the nose and eyes, a feeling of tightness in the chest, coughing, running nose and lacrimation.

The patient may be symptom-free for several (2-24) hours. After that, the symptoms of lung edema develop, i.e., cough, shortness of breath, rapid shallow breathing and cyanosis. The patient feels uncomfortable and restless as the shortness of breath increases. The patient may develop shock-like symptoms. Since lung edema is a life-threatening condition, the patient may even die.

## Treatment

The patient should be removed from the contaminated environment. In view of the possibility of a long quiescent period, the patient should be kept under observation for 24 hours. During this period absolute rest is required, and the patient should be kept warm. Transport should occur in a semi-seated position.

Hypoxia is treated by oxygen supplementation, if necessary via intubation and artificial ventilation. Oxygen supplementation shortly after the exposure may decrease and delay the development of lung edema. Coughing worsens the prognosis and can be suppressed with codeine (30-60 mg). Sedatives are not recommended. Restlessness is usually an indication of hypoxia and should be treated as such. The use of atropine, barbiturates, analeptics and antihistamines is contraindicated. Antibiotic therapy is needed at a later stage when bacterial bronchitis or pneumonitis develops after intoxication. Prophylactic administration of antibiotics is not recommended.

For the treatment of lung edema, corticosteroids have been recommended, however their efficacy has not as yet been proven. When steroids are administered shortly after intoxication, the severity of lung edema may be less than without treatment. Corticosteroid therapy is indicated for moderate and severe intoxications by lung damaging agents. Either dexamethasone or beclomethasone can be used. High doses should be administered by inhalation, as soon as possible after exposure. In severe cases of lung edema, the corticosteroids should be administered by intravenous infusion.

There are indications that antioxidants such as tocopherol may have beneficial effects in the treatment of lung edema when combined with high doses of corticosteroids. Research is underway to investigate the potential of this new approach.

## Prognosis

Due to the long quiescent period, a prognosis is difficult within the acute phase of the intoxication. If patients under treatment survive for more than 48 hours, they usually recover without residual effects. In more severe cases, lung function may take several years to normalize. Furthermore, the patient may suffer from chronic bronchitis and emphysema.

## **7.3.** Cell Toxicants (Blood Poisoning Agents)

Typical representatives of this class of chemical warfare agents are hydrogen cyanide and cyanogen chloride. The mechanism of action and pathophysiology are described in Chapter 4, "Toxicology of Chemical Warfare Agents".

## Signs and symptoms

The onset and intensity of symptoms depend on the concentration of the inhaled toxic vapor and the duration of the exposure. At high cyanide concentrations, the depth of breathing increases within a few seconds after inhalation. This effect is very powerful, since the patient usually cannot willingly hold his breath. After approximately 30 seconds violent convulsions occur. Respiratory arrest may occur within one minute after exposure, with subsequent cardiac failure within several minutes followed by death.

Inhalation of lower concentrations of cyanide produces symptoms which initially resemble those of hyperventilation: increased respiratory and heart rate, dizziness, headache, weakness of the legs and nausea. Convulsions and coma may follow which could last for hours or even days.

## Treatment

The patient should be removed from the contaminated environment. Rescue workers must wear appropriate respiratory protection.

The treatment of cyanide poisoning is aimed at rapid elimination of the cyanide ion from its target enzyme, cytochrome oxidase, by offering alternative binding sites. This is accomplished either by administration of cobalt (II) ions or by inducing the formation of methemoglobin in the blood. Oxygen supplementation is helpful as a supportive treatment but is usually insufficient for survival without administration of antidotes.

Cobalt (II) ions bind directly to cyanide, both to the free toxicant and to the cyanide bound to cytochrome oxidase, to form a stable complex. In this way, the cyanide-inhibited cytochrome oxidase is reactivated. The complex formed is eliminated by urinary excretion, turning the urine cherry red. Free cobalt ions are toxic by themselves. Therefore, cobalt ions bound in complexes are used as antidotes, e.g. hydroxocobalamine and dicobalt edentate (600 mg). Both antidotes have to be administered intravenously.

The binding of cyanide ions to methemoglobin is stronger than to cytochrome oxidase. Furthermore, the induced amount of methemoglobin in the body can largely exceed that of cytochrome oxidase. Consequently, cyanide can be liberated from cytochrome oxidase and bound to the methemoglobin in the erythrocytes.

Methemoglobin is formed from hemoglobin by nitrites, such as amyl nitrite and sodium nitrite, and by 4-dimethylaminophenol (DMAP). Amyl nitrite is available in ampoules for administration by inhalation. After breaking the ampoule in gauze, the gauze should be held close to the mouth and nose. A more effective way to administrate amyl nitrite is via artificial respiration. Concurrent oxygen supplementation should be avoided considering the explosion risk. Sodium nitrite has to be administered intravenously (300 mg in 10 ml in 3 minutes). Since sodium nitrite will cause hypotension upon standing, the patient should remain in a horizontal position. The patient will likely display a slight degree of cyanosis, as a sign of a therapeutically adequate level of methemoglobinemia, which is in the range of 30-40 %. Methemoglobinemia levels higher than 70 % are lethal. At methemoglobin levels above 50 %, pure oxygen or antidotes such as toluidin blue or methylene blue should be administered. Administration of DMAP (250 mg, slowly injected intravenously) leads to a more rapid formation of methemoglobin than via the use of nitrites. Intramuscular administration should be avoided since muscular necrosis may occur. Currently, DMAP is the first choice antidote against cyanides in most nations.

The scavenging of cyanide ions is in itself not sufficient for adequate treatment. The next step in the treatment is the enhancement of the detoxification of cyanide by the endogenous enzyme rhodanase, which converts cyanide into thiocyanate if adequate sulfur donors are available. This is accomplished by administration of compounds which can function as a sulfur donor, such as sodium thiosulfate (50 ml of 25% solution intravenous infusion over 10 minutes).

## Prognosis

Without treatment, intoxicated patients will die within several minutes. When patients under treatment survive the first 4 hours after intoxication, full recovery is likely. However, if tissue hypoxia has existed for a long period of time, temporary or permanent damage to the central nervous system will have occurred.

# 7.4. Vesicants

## 7.4.1. Sulfur and Nitrogen Mustards

## Signs and symptoms

After exposure, a quiescent period of several (1-12) hours occurs, depending on the severity of the exposure. After this period, a burning pain in the eyes is felt. Furthermore, spasms of the eyelids, lacrimation and conjunctivitis are prominent. The cornea may be damaged, possibly leading to temporary blindness.

After the quiescent period, erythema is observed on the affected skin area, accompanied by swelling. Intense itching is experienced. When the erythema disappears, increased pigmentation of the affected area is observed. After 12 to 48 hours blisters may form. The blisters are filled with a clear, light yellowish fluid. They are easily ruptured, leading to erosive lesions. Lesions may become necrotic, usually followed by infections. Lesions are painful. The healing process is slow, and may take several weeks to months. Most sensitive areas are those with a relatively high temperature and humidity, such as the genitals, axillae and the skin between the fingers.

After the quiescent period, effects on the respiratory tract manifest: irritation and congestion of the mucosa in the nose, throat, trachea and bronchi; a burning pain in the throat; hoarseness and loss of voice; and coughing, first dry and then productive. The airways may become obstructed as a result of increased secretory activity and by fragments of necrotic mucosa, causing impairment of breathing. Infections of the lower airways are likely to develop.

Effects on the gastrointestinal tract are nausea, vomiting and diarrhea. Vomit and feces may be bloodstained. Systemic effects are comparable to those caused by radiomimetic compounds: headache, nausea, anorexia, leucopenia, thrombocytopenia and anemia. High doses of mustard may cause convulsions and cardiac irregularities.

## Treatment

A specific treatment for intoxications with mustard does not exist yet. Therefore, the treatment is aimed at:

- General life support by supplemental oxygen, artificial respiration, supplemental fluid and food, as well as cardiovascular support; enhancement of the healing of mucosal and skin lesions; and
- Prevention of infections, both locally and systemically, since these impair healing and pose a threat to the weakened patient.

Eye pain should be treated with a systemic analgesic, since local anesthetics may have an adverse effect on the healing of eye lesions. Infections of the eye are prevented by application of antibacterial preparations, e.g. 1% chloramphenicol ointment. In patients with corneal lesions, atropine is applied to prevent adhesions between the cornea and the iris via dilatation of the pupil. Sterile Vaseline can be applied to prevent the eyelids from sticking together. Irritation of the eyes is diminished by sheltering them from light. For this purpose, sunglasses should be used.

Patients are placed on metal lined sheets to prevent lesions from adhering to bedclothes. Itching is relieved by applying cooling lotions, e.g. calamine lotion. Erosive lesions of ruptured blisters are covered with sterile saline compresses. In case of infections, 0.5% chlorohexidin can be added to the saline. On dry lesions, 1% silver sulfadiazine cream can be applied to prevent infections. Hair is washed with a betadine shampoo. Pain is treated with systemic analgesics, preferably paracetamol. Acetylsalicylic acid is not recommended in view of the likely occurrence of thrombocytopenia. Opiates are contraindicated since they cause respiratory depression.

Treatment of respiratory tract lesions is aimed at clearing airway obstructions and enhancing the mucosal healing. The patient should remove the mucosal fragments from the airways by coughing. Supplemental oxygen is beneficial, and artificial respiration should only be used in extreme situations. Bronchial spasms are treated by parenteral administration of xanthenes such as theophyllin, and ß-sympaticomimetics such as terbutaline. Bacterial infections are treated with appropriate antibiotics.

Systemic effects are treated by sustaining the metabolic function and supplying fluid and electrolytes. Infections should be treated immediately and intensively to prevent sepsis. Cardiovascular shock is treated with intravenous fluids and administration of dopamine and/or noradrenalin and high doses of corticosteroids. However, the efficacy of corticosteroid administration is questionable.

The development of a causal antidote for sulfur mustard poisoning is hampered by the lack of understanding of its mechanism of action at the molecular level. However, in this age of 'genomics', new tools have become available to elucidate the process of blister formation. Very recent results obtained via proteomics of human keratinocytes exposed in vitro to sulfur mustard suggested that enzymes called 'matrix metalloproteinases' are involved in the blistering process. Inhibitors of these enzymes, such as Ilomastat, actually prevented blistering of human skin in vitro. Hopefully, a causal antidote against sulfur mustard will be found in the near future.

## Prognosis

Most mustard victims will survive. Eye lesions are generally healed within two weeks, whereas the skin lesions and the respiratory tract recover within two months. Victims may experience a long period of depression and anxiety. Long term effects include visual impairment and scarring of the skin. Patients with severe lung damage suffer from chronic bronchitis and narrowing of the bronchi.

Sulfur mustard is considered to be a primary carcinogen. It needs to be stated however that the incidence of cancer in soldiers acutely exposed to sulfur mustard in WW I was not significantly different from that in the non-exposed population. Repeated and/or prolonged exposure to relatively low concentrations of sulfur mustard, such as of workers in production facilities, did lead to a significantly higher incidence of cancer, mainly of the upper airways.

Reason for concern are the reports of Iranian soldiers exposed to sulfur mustard in the mid 1980s during the war with Iraq and became blind some ten years later due to blurring of the cornea, most likely due to an autoimmune reaction.

## 7.4.2. Lewisite

## Signs and symptoms

Contact with the eyes causes immediate pain, spasm of the eyelids and lacrimation. The cornea may become fogged after several hours. After severe contamination, necrosis of the cornea may occur, with possibly permanent damage or even blindness. Upon contact with the skin, a stinging pain is felt within 10-20 seconds. The pain intensifies after several minutes, as the compound penetrates the skin. Within 30 minutes, erythema of the affected area is observed, and painful blisters may develop within 12 hours. After approximately one week, the pain becomes less severe. Lewisite-induced blisters are filled with a yellowish, opaque fluid. These blisters heal faster than those caused by sulfur mustard.

Upon inhalation, the patient experiences coughing and sneezing. Symptoms of systemic effects of Lewisite are headache, pain in the chest, weakness and restlessness. In severe cases shock can develop, followed by death.

## **Treatment**

The immediate irritation of the eyes, airways and skin will prompt the victims to respond, using available means to decontaminate. Consequently, the exposure duration will be limited and decontamination and treatment can be started directly. This is a marked difference from contamination with sulfur mustard.

The treatment of Lewisite intoxication is aimed at the removal of the toxicant from its toxicological target, the coenzyme lipoic acid. This can be accomplished by administering an excess of antidote that contain two adjacent thiol groups. The most prominent antidote is 2, 3-dimercaptopropanol, better known as dimercaprol or British anti-Lewisite (BAL). The affinity of Lewisite for BAL is higher than that for lipoic acid. BAL forms a stable complex with Lewisite, i.e. a five-membered ring structure with the arsenic atom. This complex is highly water soluble and is excreted in the urine.

Since Lewisite penetrates the skin and the eyes very rapidly, local treatment is only effective within the first few minutes after contamination. The use of 3% BAL eye ointment can prevent severe damage to the eyes. Intense pain in the eyes may require systemic administration of morphine for relief. Antibiotics are used to treat bacterial infections of the eye. Sterile petroleum jelly can be applied to the eyelids in order to prevent them from sticking together. The skin is treated with 10% BAL ointment, preferably prior to the onset of vesication. After the ointment has been applied to the skin, it should remain there for at least 5 minutes before it is washed off with water. BAL ointment can cause an itching or stinging sensation. BAL ointment is unsuitable for prophylactic use, since dermatitis can occur upon frequent use on the same skin area. BAL is chemically incompatible with silver sulfadiazine, which is frequently used in the treatment of skin burns, e.g., caused by sulfur mustard.

Systemic effects of Lewisite poisoning are treated by deep intramuscular administration of 10% BAL dissolved in oil. The recommended dose is 3 mg/kg, 6 times a day for 2 days and 4 times on the third day. Administration of BAL produces a variety of side effects: tachycardia; hypertension; nausea and vomiting; burning sensation of the lips, mouth, throat and eyes; sweating; restlessness and anxiety. Very high doses may cause convulsions and coma. The side effects are dose-dependent and usually reversible.

Other dithiols, e.g. 2, 3-dimercapto-1-propane sulfonate (DMPS), 2, 3-dimercaptosuccinic acid (DMSA) and N-(2, 3-dimercaptopropyl) phthalamidic acid (DMPA), have been studied as alternatives to BAL in the systemic treatment of Lewisite poisoning. These new compounds are water soluble, orally applicable and less toxic than BAL. DMPS appears to be more effective against Lewisite than BAL. Currently; many countries have already replaced BAL with DMPS as the antidote of choice for Lewisite poisoning.

## Prognosis

There is no insight into the long term effects of Lewisite poisoning. It is anticipated that patients who developed substantial necrosis in the airways or lung edema will suffer from chronic bronchitis and lung fibrosis.

## 7.5 Nerve Agents

Typical examples of this class of chemical warfare agents are Tabun, Sarin, Soman and VX. The mechanism of action and pathophysiology are described in Chapter 4, "Toxicology of Chemical Warfare Agents".

## Signs and symptoms

Upon exposure to relatively low concentrations of nerve agent vapor, the first sign of its action is observed in the eye, i.e. constriction of the pupil (miosis). The eye turns red and ocular pressure is experienced. Pain is experienced from the spasm of the pupil, accommodation becomes difficult, the eyelids twitch, and a frontal headache occurs. Local respiratory effects are rhinorrhea, chest tightness, coughing, and occasionally wheezing. At this level of intoxication, symptoms may disappear in approximately one day.

After exposure to high concentrations of nerve agent vapor, the symptoms increase in intensity and more symptoms develop. The effects of nerve agents are the result of action on the muscarinic and nicotinic receptors, and on receptors within the central nervous system. Typical symptoms caused by

stimulation of muscarinic receptors are excessive bronchial and upper airway secretion; airway obstruction and respiratory distress. Breathing becomes difficult, which is accompanied by audible wheezing and cyanosis. Salivation, sweating, lacrimation, nausea, vomiting, diarrhea, cardiac arrhythmia, involuntary defecation and urination occur. With increased hypoxemia and cyanosis, the victim experiences exhaustion, collapse and becomes unconscious. Nicotinic effects are muscular twitching, fasciculation and cramps. As a result of vasoconstriction, blood pressure increases slightly and the skin becomes pale. The twitching and fasciculation may spread to the whole body. This phase is followed by a generalized muscular weakness, which includes the respiratory muscles. First, respiratory movements become labored, shallow and rapid; next they become slow and finally intermittent.

The central nervous system effects are restlessness, anxiety, headache, shaking, and difficulty in concentrating. Generalized convulsions may occur occasionally. The patient may become comatose, reflexes disappear, and intermittent respiration develops. Again, generalized convulsions occur. Central respiratory depression occurs, which impairs respiration even more. Circulatory centers may also be depressed, leading to a reduction of the heart rate and decreased blood pressure.

Death is caused by anoxia, which is a result of airway obstruction due to secretion and bronchiconstriction, weakness of the respiratory musculature as well as the central depression of respiration. The sequence of events: cessation of respiration, unconsciousness, fall of blood pressure and cardiac arrhythmias leads to death.

## **Treatment**

The treatment of nerve agent poisoning can be **supportive**, **symptomatic**, and **causal**.

**Supportive** treatment comprises assisted or artificial respiration and oxygen supplementation. In severely intoxicated patients, artificial respiration must be continued until the patient can breathe by himself, which may take several days or even weeks.

**Symptomatic** treatment consists of the administration of high doses of atropine in order to block the muscarinic effects of the intoxication. Every 10 minutes, 2-4 mg of atropine sulfate must be administered intravenously or intramuscularly, until an adequate level of atropinization is reached. Indications that this level is reached are the drying of the nasal and oral mucosa and the increase in heart rate to approximately 90 beats per minute. The administration of atropine has to be continued for several days or weeks, depending on the severity of the intoxication. Since high doses of atropine make the ischemic heart more susceptible to arrhythmias, ECG monitoring is recommended. An overdose of atropine will cause euphoria, hallucinations, anxiety and delirium. Therefore, keeping the patient under close observation is necessary. An important effect of atropine is the inhibition of sweat production, which increases heat stress. Care must be taken to avoid hyperthermia.



Auto injectors have been introduced for self medication or buddy help in order to give nerve agent casualties a better chance of reaching a medical aid station Miosis is treated by applying drops of atropine into the eyes, which reduces miosis, pain in the eye and headache. The beneficial effect of this procedure is questionable, since it is believed by some that accommodation problems of the eye are increased by these eye drops. Currently there are no adequate drugs available for symptomatic treatment of the nicotinic effects of nerve agent poisoning.

Anticonvulsants are also used for symptomatic treatment. Convulsions may cause brain damage in severely poisoned patients. The anticonvulsant of choice is diazepam, which has proved to be beneficial in animal studies. Diazepam in an auto injector has recently been approved by the FDA as an antidote against nerve agent poisoning. A drawback of diazepam is its very poor solubility in water. Diazepam is administered intramuscularly at an initial dose of 10 mg. Depending on the condition of the patient; additional doses of diazepam are administered at 4 hour intervals. Some countries use the water soluble prodrug of diazepam, avizafone, in auto injectors as anticonvulsants. In addition, midazolam is being studied as a promising alternative for diazepam.

Since the toxic effects of nerve agents are the result of acetylcholinesterase (AChE) inhibition, **causal** treatment is aimed at the reactivation of the inhibited enzyme. This reactivation can be achieved with oximes (RCH=NOH), which are highly nucleophilic compounds that, in many cases, are able to cleave the covalent bond between the enzyme and the phosphyl moiety.

The therapeutic value of oximes has been shown both in animal studies with nerve agents and in patients intoxicated with organophosphorus pesticides. Examples of oximes are pralidoxime and obidoxime. The efficacy of these oximes depends on the nature of the organophosphorus inhibitor. AChE inhibited by Sarin or VX is easily reactivated by the aforementioned oximes. For AChE inhibited by Tabun, however, obidoxime is more effective than pralidoxime. Unfortunately, these oximes are not effective for Soman poisoning. This is the result of a process called 'aging' of the inhibited enzyme. In this process, the phosphyl moiety on the inhibited AChE loses an alkyl group, after which a very stable agent-enzyme complex remains. This complex resists reactivation by oximes. The relatively new oxime, HI-6, offers some protection against Soman and Tabun in primates, in combination with atropine and diazepam. Strangely enough, the AChE in the blood of these animals was not reactivated after administration of the oxime. Therefore the efficacy of HI-6 is, at least partly, the result of a mechanism other than enzyme reactivation.

An alternative to oxime therapy, especially in those cases where aging occurs, is pretreatment with a 'reversible' AChE inhibitor. Carbamates (such as pyridostigmine and physostigmine) carbamoylate the active site of AChE by covalent binding. As a result, the enzyme is inhibited, as in after nerve agent poisoning. In contrast with the phosphylated enzyme, however, the carbamoylated enzyme spontaneously reactivates with a half-life of 15-30 minutes. An excess of AChE is present in the body. Therefore, a fraction of this AChE can be inhibited without influencing the performance of the subject. This fraction is approximately 30 %, when measured in blood. When at this stage nerve agent poisoning occurs, the nerve agent will inhibit the remaining 70 % active AChE completely, very rapidly and irreversibly. In time, as the concentration of the nerve agent in the body decreases, active AChE is gradually released by decarbamoylation, in sufficient amounts to sustain life. This pretreatment has to be combined with atropine treatment, in order to suppress the symptoms of the nerve agent poisoning. In animal studies, pretreatment with Carbamates has proven to be effective, at least in terms of survival after nerve agent intoxication. After pretreatment with pyridostigmine the surviving animals were highly incapacitated, which is an important drawback when translated into a military operational scenario. Since pyridostigmine is a quaternary compound it cannot pass the bloodbrain barrier under normal circumstances, and consequently cannot protect AChE in the brain. Physostigmine is capable of protecting brain AChE. Pretreatment with physostigmine is associated with much less incapacitation in animals surviving nerve agent intoxication. Unfortunately, pretreatment with physostigmine will cause more side effects than with pyridostigmine, in terms of behavioral effect, probably as a result of penetration of physostigmine into the brain. These centrallymediated side effects are ameliorated by co-administration of a low dose of scopolamine.

Soldiers are equipped with automatic injectors (auto-injectors), filled with atropine, oxime and in some cases an anticonvulsant, for self-aid or buddy-aid in the event of nerve agent poisoning. Various designs and compounds are used by different nations. Some countries have chosen separate injectors for atropine and oxime, whereas others have chosen combined injectors. Most nations have incorporated either pralidoxime or obidoxime as a nerve agent antidote in their auto injectors for military use, whereas only a few countries have chosen HI-6 for this purpose. This situation is likely to change in the near future, since more and more nations are considering introduction of HI-6.

The first auto-injector(s) should be used upon appearance of the first signs of nerve agent poisoning. If the symptoms increase or even do not disappear in the following 10 minutes, the second dose of atropine and oxime should be injected, and if necessary, 10 minutes after that, the third dose. Soldiers are equipped with auto-injectors in order to enable them to resume their task after mild intoxication. Furthermore, time is gained to transport the victim to medical aid facilities after moderate or severe poisoning. There is always a risk of the unjustified use of the auto-injectors, e.g. as a result of panic at a false alarm. Effects of atropinization will become manifest in that case: wide pupils, increase in heart rate, dry mouth and heat stress. The latter effect can be dangerous when wearing NBC garments. It is interesting to note that initiatives are ongoing concerning registration of various atropine/oxime auto injectors by the FDA and/or its European equivalent.

Many NATO countries have introduced pyridostigmine bromide as a pretreatment drug against nerve agent poisoning. In situations where there is a likely threat of chemical warfare agents, the soldier ingests a tablet containing 30 mg of pyridostigmine bromide three times a day, for adequate protection throughout the day. During Operation Desert Shield/Desert Storm, this pretreatment was used by, amongst others, United States soldiers. About half of them complained of gastrointestinal problems, e.g. cramps, loose stools and flatus, whereas 5-30 % complained of urinary urgency and frequency. These side effects are obviously very inconvenient under NBC conditions. It is not clear to what extent the stresses of the anticipated combat, sleep deprivation and life in the field contributed to the intensity of the side effects. Furthermore, pyridostigmine has become a suspect in relation to "Gulf War Syndrome", but it needs to be stressed that no proof of this relationship has been found. Several nations are currently considering replacement of pyridostigmine with physostigmine/scopolamine, in view of the better performance of the latter combination in terms of protection against post-exposure incapacitation.

## Prognosis

Patients surviving the critical period will probably recover without residual effects, unless brain damage has resulted from convulsions or periods of hypoxia. Nerve agents may cause organophosphate-induced delayed polyneuropathy, manifested by weakness of the legs, and to a lesser extent of the arms, and muscle twitches. In severe cases, progression to paralysis may occur. Recovery may require several years.

Recently, some data have become available with respect to the follow-up of victims of the Tokyo subway attack with Sarin in 1995. Many of these patients still suffer from neurological and psychological effects, which suggest that the effects of nerve agent intoxication are more persistent than previously assumed.

## 7.6. Psychogenic Incapacitating Agents

An example of a compound in this class of incapacitating agents is BZ (3-quinuclinidinyl benzilate).

## Signs and symptoms

In small doses, BZ causes sleepiness and diminished alertness. In the first few hours after exposure, typical symptoms are increased heart rate, dizziness, vomiting, blurred vision, confusion and sedation. After 4-12 hours, the patient is unable to respond adequately to the environment and to conduct normal body movements. An increase in activity follows accompanied by unpredictable behavior with delusions and hallucinations. Within 2 to 4 days after exposure, the symptoms gradually disappear.

## <u>Treatment</u>

Symptomatic treatment will usually be sufficient. A friendly attitude towards the patient is required. Restraint may be necessary in order to prevent patients harming themselves. When the ambient temperature is above 25°C the risk of heat stroke can be avoided by cooling the patient. Physostigmine (2-3 mg intravenously) is indicated as the antidote for those patients whose lives appear to be in danger. Repeated injections, followed by a slow intravenous infusion, may be required to 'titrate' the symptoms away. Quaternary cholinesterase inhibitors, such as pyridostigmine and neostigmine, cannot be used as antidotes, since these compounds do not cross the blood-brain barrier and therefore cannot antagonize the central effects of BZ.

## Prognosis

Patients will recover within several days without residual effects.

## 7.7. Irritating Agents

## 7.7.1. Lacrimators

Examples of lacrimators or tear gases are CS (orthochlorobenzylidene malonitrile), CN (chloracetophenone) and CA ( $\alpha$ -bromobenzyl cyanide). The latter compound is too toxic to be used in riot control and is therefore obsolete.

## Signs and symptoms

After exposure to CS or CN, a strong burning sensation is felt in the eyes. Conjunctivitis, erythema and spasms of the eyelids occur, as well as violent lacrimation and photophobia. High concentrations of CN may cause temporary blindness. After exposure to CS, a burning sensation and pain is felt in the nose and throat, extending to the trachea and bronchi, followed by sneezing, rhinorrhea and possibly a feeling of suffocation. Nausea, headache and coughing may occur. CN affects primarily the eyes, whereas irritation of the upper airways occurs only after exposure to high concentrations of this lacrimator. High concentrations of inhaled lacrimator may cause lung edema. A transient burning sensation is felt on the skin, particularly on moist skin areas, with possible reappearance upon washing the affected area some hours later. Erythema and vesication may occur upon prolonged exposure to high concentrations of CS and CN.

## <u>Treatment</u>

Usually the symptoms will disappear when the patient is brought into fresh air. Preferably contaminated clothing is removed for cleaning. In addition, the lacrimatory effect on the eyes provides for considerable decontamination. Washing the eyes, mouth and skin with water may hasten the resolution of symptoms. Rubbing the eyes is dissuaded, in order to avoid mechanical injury. Casualties suffering from blindness as a result of exposure to CN vapor should be reassured, since this effect is only temporary. Droplets or solid lacrimator may cause corrosive burns and should be removed promptly by washing extensively with water.

Respiratory effects do not usually require treatment other than fresh air, unless lung edema develops. In that case, treatment is as described for lung damaging agents.

The transient effect on the skin does not require treatment. Inflammation of the skin, as well as itching, is treated with corticosteroid cream or calamine lotion. Secondary infection should be treated with the appropriate antibiotics. Blisters should be treated as are other second degree burns.

## **Prognosis**

Lacrimator patients usually recover without residual effects.

## 7.7.2. Vomiting agents

An example of this class is Adamsite (diphenylaminearsine chloride, DM).

## Signs and symptoms

After exposure, a quiescent period of several minutes may occur before symptoms manifest. A burning sensation in the eyes, nose and throat is felt, followed by rhinorrhea, lacrimation, excessive salivation, sneezing, severe frontal headache, nausea and vomiting. Prolonged exposure may lead to pain in the chest and asthma-like symptoms. The intensity of the effects is maximal 5 to 10 minutes after exposure. The symptoms disappear within 2 hours.

Exposure of the skin to high concentrations of Adamsite, which is unlikely in the open field, will cause erythema, itching and vesicle formation. Severe exposure may lead to neurological effects, such as unsteadiness and sensory effects in the legs. Loss of consciousness may occur.

## <u>Treatment</u>

Exposure to Adamsite is not as threatening as the symptoms suggest. The patient should be brought into fresh air. The eyes and skin may be washed with water. The mouth can be rinsed with water. Care must be taken not to swallow contaminated water. The effect on the lung function after severe intoxication should be treated as described for lung damaging agents. The frontal headache is treated with an analgesic.

## Prognosis

The patient usually recovers within 2 hours, without residual effects.

## **7.8.** Conclusions for CW agents

Possibilities for causal treatment of intoxications with CW agents are still quite limited. Some agents act so rapidly that after exposure to high doses timely intervention is hardly feasible. Furthermore, for some agents, adequate treatment is hampered by the absence of causal antidotes. Consequently, research on improving medical countermeasures, in the frame work of civilian casualties, needs to continued, in particular since emerging technologies may offer new opportunities. For the military environment it is unlikely that a well protected and well trained army will face an appreciable number of casualties when the likely quantities of agent in rogue states is on the order of 100 tons. For the military environment, money is better spent on medical counter measures for biological attacks.

In recent years in vitro diagnostic methods have been developed for various CW agents to establish the exposure in qualitative and quantitative terms. Such information can be used to design an optimum treatment regimen for CW patients and may be of help when judging the prognosis of such patients.

## 7.9. Viruses

7.9.1. Crimean-Congo hemorrhagic fever, Ebola viruses, Lassa fever virus, Marburg virus, South American hemorrhagic fever viruses (Junín, Machupo, Sabia, Flexal, Guanarito)

## Signs and symptoms

Although these viruses are quite different and from different sources, they all have one symptom in common. They all lead to hemorrhagic symptoms. The severity of the illness is also quite different

## Treatment

They have also in common that there is no specific therapy available. Neither are any vaccines available for the disease. The treatment is therefore mainly supportive.

## **Prognosis**

Fatality rates vary among the different types of viruses, ranging between 15 and 25% although information on some types (Ebola and Marburg) is lacking.

## 7.9.2. Rift Valley fever virus

#### Signs and symptoms

After an incubation period of 2-5 days, an illness period of 2-5 days develops with nonspecific signs of fever, conjunctival infection, and abdominal tenderness. A small fraction of victims will show signs of hemorrhagic fever. A similar fraction develops eye defects.

#### Treatment

There only are investigational drugs and vaccines available.

#### Prognosis

For the small fraction of more severe cases, the mortality is about 50%. The mortality is small for less severe cases.

#### 7.9.3. Variola major virus (smallpox virus)

#### Signs and symptoms

After an incubation period of about two weeks, a disease period of three weeks follows. The disease is highly contagious from patient to patient. High fever and typical skin eruptions (rash-lesions-macules-papules-vesicles-pustular lesions-scabs) are clear symptoms. They dissipate after some weeks and leave pockmark scarring. Other symptoms include malaise, rigors, vomiting, head and back aches. A fraction of patients develop delirium.

## **Treatment**

Therapy is supportive as there is no specific therapy or antidote available. The disease is highly contagious, and quarantine of potentially exposed persons is required. A vaccine is available and special vaccines are available for people with increased risk of vaccine complications.

#### Prognosis

Mortality varies between 1-30%. The disease has been eradicated by an intensive program of the WHO. Routine vaccination was abandoned in 1981.

7.9.4. Venezuelan equine encephalitis virus, Eastern equine encephalitis virus, Tick-borne encephalitis complex viruses.

#### Signs and symptoms

The incubation period is from 1-5 days followed a sudden outbreak of symptoms. Headache and spiking fevers are among the symptoms. Often rigors, general malaise, lumbrosacral and lower extremity myalgia, and sensitivity to light occur.

## **Treatment**

There is no specific therapy or antidote, and treatment is primarily supportive. There is an experimental vaccine available.

## Prognosis

Mortality of the encephalitis type viruses is low.

## 7.9.5. Yellow fever virus

#### Signs and symptoms;

After an incubation period of 3-6 days, the patient suddenly develops fever, chills, headache, muscle pain, and prostration. The virus grows in the lymphatic system and attacks internal organs and can cause skin eruptions and bleeding. Liver damage, gastrointestinal complications and hemorrhaging of the stomach can occur in severe cases. The illness lasts for about 2 weeks.

## Treatment;

There is no specific antidote or therapy available. A live attenuated vaccine is available.

## Prognosis;

Within two weeks from the onset of symptoms, the patients either recover or die. Mortality is high.

## 7.10. Bacteria

## 7.10.1. Bacillus anthracis

## Signs and symptoms

Depending on the route of entry, anthrax causes infections in the skin, lungs or gastrointestinal tract. Cutaneous anthrax can occur on the hands and forearms of persons working with livestock or infected products (wool). Pulmonary anthrax results from the inhalation of spores. After an incubation period of 1-7 days, patients suddenly develop flu-like symptoms. After a few days, patients develop more serious effects such as a respiratory tract syndrome, difficulty breathing, tachycardia, cyanosis and terminal shock. Usually the patient dies within a day after the onset of the severe effects.

## **Treatment**

Cutaneous anthrax can be treated with antibiotics and is cured relatively easily. There is a vaccine available, but in order to be effective, six shots are required with a yearly booster. This vaccine works against cutaneous anthrax and is believed to work against the other forms as well. For personnel not vaccinated, penicillin is the antibiotic of choice, however, some strains can be resistant, and other antibiotics should be used. This treatment is known to work for cutaneous anthrax and is believed to work also for the other forms. In the very early stages of disease development, treatment with antiserum might be useful for the pulmonary and intestinal forms of anthrax.

## Prognosis 1997

Cutaneous anthrax is cured relatively easily, but when not treated, it develops into a systemic form. More than two-thirds of victims in the anthrax letter incidents developed cutaneous anthrax, and all survived. When not treated, pulmonary anthrax has a lethality of more than 90%. Even in cases that are treated, the mortality is still significant. More than 50% of the patients who developed pulmonary anthrax in the anthrax letter incident died.

## 7.10.2. Brucella abortus, Brucella melitensis, Brucella Suis

## Signs and Symptoms

Route of entry is through inhalation or ingestion. After a widely varying incubation period of typically 1 to 3 weeks, but up to several months, intermittent fevers, sweating, chills, malaise, head and body aches and anorexia develop. The time required to confirm the diagnosis is quite long and may be more than a week.

## **Treatment**

Treatment with a combination of antibiotics shortens the disease period. Unfortunately, some cases are resistant and the relapse rate is high.

## **Prognosis**

When properly diagnosed and treated, the fatality rate is low, around 2%. For some strains, mortality may be up to 12%.

## 7.10.3. Burkholderia (Pseudomonas) mallei Glanders

## Signs and symptoms

Glanders is spread as an aerosol by infected animals and enters through the nose and mouth. It therefore affects the nasal area and respiratory tract leading to localized ulcerating and draining

lesions. Inhalation glanders can result after 10-14 days of severe pneumonia. The bacterium is highly infective, and the disease is contagious. It can also enter through broken skin which leads to draining pus-forming lesions after an incubation period of 3-5 days. After invasion of the bloodstream, the disease spreads through the body with a diffuse pustular rash and circulatory failure.

#### **Treatment**

Only traditional antibiotics, possibly requiring a combination of antibiotics, have an effect. A vaccine is not available. Patient isolation is required because the symptoms show some resemblance to plague (pneumonic disease) or smallpox (pustular rash). The disease is also highly contagious.

#### Prognosis

When the patient recovers, immunity is not acquired and a second infection is possible. Invasion of the blood stream is usually fatal.

## 7.10.4. Burkholderia (Pseudomonas) pseudomallei Melioidosis

#### Signs and symptoms

The signs and symptoms are very similar to those of glanders. Depending on the route of entry, the skin or the respiratory system is attacked. Chronic infections of many body parts are possible. The appearance of Melioidosis differs from glanders. It more closely resembles diseases such as tuberculosis (pneumonic type) or spotted typhoid fever (cutaneous type).

#### **Treatment**

There is no known treatment for the disease, but combined antibiotic therapy sometimes has an effect. The disease is contagious, and the differential diagnosis with other contagious diseases, such as tuberculosis, is difficult. Therefore, isolation of the patient is required.

#### Prognosis

Melioidosis is almost always fatal if it progresses and invades the bloodstream.

## 7.10.5. Francisella tularensis

#### Signs and symptoms

The infection can be acquired through insect bites or from contact with blood or tissue fluids. Ingestion of contaminated food or inhalation of aerosols containing the organism is also a possible route of entry. There are several different strains, and the virulence of the strains differs widely. The symptoms depend on how the infection is acquired. After an incubation period of 2-10 days, the general effects are fever, chills, headache and malaise. Skin lesions are normally accompanied by enlarged lymph glands. Typhoid type tularemia can result from all routes of entry and causes fever, prostration, and weight loss. Swelling of the lymph glands does not occur. The diagnosis is difficult because the signs can vary and are non-specific.

#### **Treatment**

Antibiotic treatment, in particular streptomycin or gentamicin, is effective. An experimental vaccine is available and has been shown effective against pulmonary tularemia.

#### Prognosis

Due to the wide variety in strains and the routes of entry, prognosis is difficult. Some strains which lead to pulmonary tularemia have mortality above 50% when untreated.

## 7.10.6. Yersinia pestis

#### Signs and symptoms

Transmission to humans is by the bite of infected fleas (bubonic plaque) or from human to human. In the bubonic form, the bacterium spreads through the lymphatic system resulting in enlarged lymph

nodes in the groin. After invasion of the bloodstream, a general infection is produced. With septicemia, clotting of blood vessels results in gangrene in fingers and toes. The disease is accompanied by high fever, weakness and painful buboes. Pneumonic plaque results in a rapidly progressive hemorrhagic pneumonia

## **Treatment**

Directly after exposure, doxycycline or ciprofloxacin may be useful for prophylaxis. When symptoms become evident, other antibiotics like streptomycin are favored. An attenuated bacterial vaccine is available and works against bubonic plaque but not against bacteria that are inhaled.

## Prognosis

Untreated bubonic plaque has 75% mortality. Pneumonic or septicemic plaque is usually fatal when treatment is delayed more than 24 hours after onset of symptoms.

## 7.11. Rickettsiae

## 7.11.1. Coxiella burnetii; Q fever

## Signs and symptoms

The disease is contracted by inhaling dust contaminated with the organism. One inhaled organism is sufficient to cause infection. After 10-14 days, victims develop chills, headache, fever, chest pain, weakness, perspiration and loss of appetite. The disease presents as a fever of unknown origin or an atypical pneumonia. The latter shows some resemblance with legionellosis (Legionnaire's disease). If the disease progresses to severe pneumonia, it resembles tularemia and plaque.

## **Treatment**

Treatment is with antibiotics for about one week. Vaccination with one dose of attenuated organisms provides protection for several years. Vaccination in persons who have acquired immunity can cause cutaneous reactions including necrosis.

## Prognosis

The disease is lethal in rare cases and not very contagious.

## 7.11.2. Rickettsiae Prowazekii

## Signs and symptoms

After an incubation period of 1-2 weeks, victims develop what is called epidemic typhus with a high sustained fever, general pains, a skin rash and severe headache.

## **Treatment**

Treatment is with antibiotics, while secondary infections must be prevented. Supportive treatment helps shorten the illness period. Vaccines provide good protection although for uncertain duration, therefore the vaccination should be repeated every 4 months when the risk of infection is high. Prognosis

# Prognosis

The FOA briefing book on biological weapons mentions a high mortality. Other sources mention a lower mortality rate of less than 20% (Janes CB Defense Guidebook). The mortality rate can be lowered by vaccination. In addition, vaccination changes the course of the disease and reduces the risk of infection.

## 7.11.3. Rickettsiae rickettsi Rocky Mountain spotted fever

## Signs and symptoms

Symptoms develop abruptly after an incubation period of 3-9 days. Typical symptoms are photophobia and further include fever, joint and muscle pain, and head ache. About half a week after the first signs, a skin rash develops at the ankles and wrists which rapidly spread over the body

## <u>Treatment</u>

Antibiotics are effective in shortening the illness period and reducing the mortality rate. There is no vaccine available.

## Prognosis 1997

Fatality rate of the untreated Rocky Mountain spotted fever may be as high as 25%, though other strains are less lethal.

## 7.12. Toxins

## 7.12.1. Botulinum toxins

## Signs and symptoms

After an incubation period of 1-3 days, victims become ill with disturbed vision, stomach pains, diarrhea, giddiness and muscular weakness. Flaccid paralysis follows as a result of motor nerve terminal blockade at the myoneural junction. The paralysis starts at the face (eyes). It progresses symmetrically downward in the throat, chest, stomach and extremities. When the diaphragm and\_chest muscles become fully paralyzed, respiration is inhibited, and the victim suffocates.

## Treatment

An antitoxin is available, but it should be given early in the development of the disease in order to be effective. Further treatment is supportive and includes assisted breathing. A vaccine against type A, B, C, D and E is available (limited distribution) and has been used to protect occupationally at risk workers.

## Prognosis 1997

The treatment does not reverse existing paralysis. When not treated, the intoxication is lethal.

## 7.12.2. Ricin

## Signs and symptoms

Ricin blocks the production of proteins by the cells, and it attacks various tissues in the human body. As a result, symptoms are dependent on the route of entry of the toxin, either inhaled, ingested or injected. When inhaled the incubation period is a few hours and the symptoms include cough, chest tightness, difficulty breathing, nausea and muscle pain. This becomes more severe with inflammation of the lungs, cyanosis and death within 36-48 hours.

## **Treatment**

There is no therapy and the only treatment is supportive. For inhaled ricin, the lungs and circulatory system might be supported by providing oxygen. Ingested ricin might be absorbed by active carbon. Ingested ricin leads to fluid loss and fluids should be replaced. There is no treatment available for ricin injection other than supportive measures to treat organ failure. There is no vaccine.

## **Prognosis**

Mortality rate is high. The LD<sub>50</sub> is approximately 1 mg.

## 7.12.3 Saxitoxin

## Signs and symptoms

Typical for saxitoxin poisoning is the rapid onset of effects, which is only delayed in cases where the ingested dosage was very small. The initial symptoms are numbness or tingling of the lips tongue and finger tips, followed by the neck and extremities. Mainly nerves are attacked affecting muscle coordination, and incoherence is common. The attack of nerves in the eyes and mouth area leads to uncontrolled ocular movements and difficulty in speech and swallowing.

## Treatment

Vomiting should be induced and general supportive care provided. Supported breathing may be required.

## Prognosis

The mortality rate is high depending on the dose ingested and the time until the vomiting response. The  $LD_{50}$ to man is about 1 mg.

## 7.12.4. Staphylococcal enterotoxin B, (SEB)

#### Signs and symptoms

Infected persons fall ill after a few hours with typical food poisoning effects such as diarrhea, vomiting and stomach cramps. Inhalation of SEB may lead to different effects. The incubation period is 1-6 hours after which high fever (39-43°C), chills, headache and myalgia develops. There might be non-productive coughs which persist together with the fever for one to four weeks. When the infective dosage is large, chest pain and shortness of breath may also occur.

## Treatment

Food poisoning victims frequently recover without any treatment within 24 hours. For those infected through the inhalation route, pain relievers and cough suppressant help the recovery.

#### Prognosis

Mortality of both forms is very low. Victims of food poisoning usually recover in 24 hours. Those exposed through the respiratory system take up to 4 weeks to recover.

#### 7.13 Conclusions for Biological Agents

In many cases, there is no therapy or prophylaxis. Because detection is difficult, exposure may pass unnoticed. The medical community will most likely be the first to recognize exposures as victims present with illness. New options for prophylaxis and treatment are urgently required.

# 8. Decontamination

# 8.1. Introduction

Decontamination has always been the most controversial issue in the defense against chemical warfare agents. The difference between chemical and biological decontamination is striking as stated in a WHO review:"Decontamination is not as critical for biological agents as it is for chemical ones, since biological agents are non-volatile, difficult to re-aerosolize and leave little residue on skin or surfaces". Several forms of decontamination have been considered, such as personnel, equipment and terrain. Decontamination levels include expedient, thorough and complete. In WW I, decontamination activities were restricted to cleaning up puddles of liquid from ruptured (unexploded) shells and skin decontamination after the introduction of mustard gas in 1917. During WW II, there was little attention given to decontamination. Over the course of the cold war period, many countries became active in developing decontamination procedures primarily for the individual soldier and military equipment. However, it appeared that accomplishing thorough decontamination to the degree that there was no longer a vapor hazard or more importantly a contact hazard was very difficult to achieve. Agent is absorbed by many of the modern construction materials such as rubbers, plastics and paints. Agent absorption by "older" materials such as wood and leather is also significant. Absorption of agent can largely be prevented if decontamination procedures are started within a few minutes after contamination. From field trials it became clear that the minimum time for larger equipment such as tanks and vehicles to reach a decontamination station was on the order of several hours. Over this period, weathering, the natural form of decontamination has transpired, and the absorption process had finished making most decontamination actions redundant. Additionally, to decontaminate contaminated terrain to eliminate hazard appeared difficult to accomplish. It should be remembered that initial decontamination concepts were developed during a time that repeated chemical attacks were expected, several per week of war, and that the encumbrance of personnel protective equipment and degradation of performance of personnel was considered a serious issue. Decontamination was required to enable soldiers to operate without the hindrance of the mask and protective clothing.

The difficulties encountered in reducing contamination quickly to non-hazardous levels induced another concept: "Contamination Control" and "Fight Dirty". The latter concept aimed at fighting and winning the battle first before considering decontamination. In many cases this resulted in disposal of the contaminated items, to include protective clothing. The goal of "Contamination Control" was to keep contamination under control in those cases were it matters. It is obvious that both the incomplete decontamination and the contamination control principle involved some risk. This was thought to be acceptable when engaged in warfare.

As indicated in Chapter 3, in the 21st century there are several major changes relevant for decontamination concepts. Those changes are:

- Low (zero) casualty acceptance
- Chemical attacks will come as a surprise
- Decontamination of individuals or casualties is required
- 100% clean guarantee after decontamination is required
- Chemical attacks are rare incidents
- Chemical attacks are less massive

- Disposal of contaminated items is probably more cost effective than decontamination.
- Chemical attacks by terrorists have to be considered with a different attitude toward decontamination.

Most military still maintain the previous century's view on decontamination and "Contamination Control". Relevant in this context are the definitions used in decontamination, as are stated below. It can be expected that with the decreasing probability of a chemical incident in the future that there will be less emphasis on the military aspects of decontamination. Contaminated equipment will most likely be abandoned.

In the case of CB incidents in a civilian environment, clean up or decontamination is mandatory.

The military doctrine for biological decontamination is not as well established. Nevertheless, this doctrine or at least the technical actions relevant for biological decontamination are very similar to and shall be executed by the same systems and equipment as chemical decontamination.

# 8.1.1. Definitions

**Contamination Avoidance:** Refers to any actions taken in order to render ineffective the attempt to contaminate materiel; includes design as well as mission-paralleling actions, e.g. covering materiel with a protective tarpaulin.

**Contamination Control:** Refers to any measure taken to assure that existing contamination is eliminated or contained at its actual location and not transferred to other locations. This does not include control in the sense of detection.

**Immediate Decontamination**: Decontamination carried out by individuals upon becoming contaminated and may include decontamination of personal clothing and/or equipment in addition to skin. The goal is to save lives, minimize casualties and limit the spread of contamination. Although somewhat less effective, decontamination of skin some 30 minutes after contamination still might reduce the severity of the effects. Reactive Skin Decontamination Lotion, with a capability to extract agent that has penetrated into the skin, must be used.

**Operational Decontamination**: Decontamination carried out by an individual and/or unit and is restricted to specific parts of operationally essential equipment, materiel and/or working areas. The aim of operational decontamination is to sustain operations by reducing the contact hazard and limiting the spread of contamination.

**Thorough Decontamination**: Extensive decontamination of personnel, equipment, materiel, and/or working areas, in order to permit the total removal of individual protective equipment and to maintain operations with minimum degradation. The aim of thorough decontamination is to eliminate the need for individual protective equipment so that the soldier can continue the mission while safely handling the materiel. The lessons from recent conflicts, however, give proof that the knowledge about the risks of mission-acquired health damages among the military community has greatly improved. Due to this fact, the requirement is foreseeable for thorough decontamination procedures to approach or meet the civilian workplace standards.

**Repatriation/Recovery Decontamination**: Decontamination to nationally established standards so that the equipment can be transported through third party states and/or returned to the country of origin. This level must consider the standards of the country or countries through which the equipment will be transported on its way to its final destination as well as the civilian standards of the country of origin, since this equipment may well be used in case of a civil emergency or a terrorist attack.

# 8.2. Decontamination, Basic Principles.

# 8.2.1. "Aging" or "weathering"

"Aging" or "weathering" occurs through the influence of environmental factors, such as wind, temperature, humidity, UV radiation, etc. It usually takes some time to be effective. In some cases of very persistent biological warfare agents, it might take years. It is therefore only acceptable for areas/materiel that is not needed in the near future.

# 8.2.2. "Physical decontamination"

Physical decontamination consists of removing/encapsulating contaminants (destruction or detoxification is not necessarily achieved). Subsequent treatment of the contained agent is required to achieve complete decontamination. For this reason, physical decontamination may be considered as a partial method, although the action of removing contaminants can still achieve the main goal of contamination control.

Examples of physical decontamination include:

- a) Rinsing with water
- b) Extraction with organic solvents
- c) Accelerated evaporation by heating (including using hot liquid or gaseous media, e.g. steam)
- d) Adsorption and removal with solid adsorbents e.g. fuller's earth
- e) Removal of protective layers applied prior to contamination
- f) Burying or sealing contamination, e.g. using blankets containing activated carbon

Example "b". Chemical agents tend to show greater affinity to organic compounds and thus rinsing with organic solvents normally results in improved physical removal. In addition, organic solvents also allow the extraction of absorbed agents from porous materials but may, as a result, damage certain substrates and/or coatings.

Example "c". Thermal desorption of agents can, among others, be achieved by the use of heated air which results in evaporation of the contaminant. With this method, the toxic agent is released into the atmosphere which may present an increased vapor hazard.

Example "d". Solid adsorbent decontaminants are very useful in removing contaminants from surfaces. Activated carbon, certain polymer ion-exchangers and fuller's earth are typical examples of solids that adsorb agents and retain them, allowing for safe removal and subsequent disposal. The usefulness of solid compounds for the decontamination of large equipment or vehicles is limited due to the problems of application over large surface



Most decontamination is accomplished by brooms and water sprays. Although more advanced equipment is available, it is often difficult to get it to the place were it is needed in a timely manner. Adding enzymes might work if time is no constraint and the agent is known. Enzymes do not work for N.

areas. In addition, decontamination with solid absorbents may produce toxic dusts requiring further treatment and/or continued wearing of respiratory protection. A typical example of a solid adsorbent decontaminant combined with an organic solvent is the stain remover with the brand name K2r.
Examples "e" and "f". Coatings intended to seal or retain contamination can also be used to ease the burden of decontamination. Although covering or burying contaminated items to protect personnel is not a method of decontamination, this method may still meet operational requirements. Usage of contamination control procedures such as adsorbent layers and disposable covers that are removed after initial contamination can also reduce the subsequent decontamination burden.

Example "f". A special case of sealing contamination is in the case of covering contaminated casualties with activated carbon-containing blankets. In this way, the vapor hazard for the environment is reduced, and the patient becomes decontaminated although in a passive way

## 8.2.3. "Chemical decontamination"

Chemical decontamination methods rely on chemical reactions, which transform toxic molecules into less toxic or non-toxic compounds. Due to the specific nature of most chemical agents, hydrolysis, oxidation and nucleophilic displacement are the principal reaction mechanisms that allow efficient decontamination. H and V type chemical agents have a sulfur atom that is quite susceptible to oxidation and nucleophilic displacement, whereas both G and V type agents are sensitive to hydrolysis at the phosphorus atom.

Chemical decontamination methods may belong to one or any combination of three processes:

Electrophilic (oxidation, chlorination)

Nucleophilic (hydrolysis or other nucleophilic attack, e.g. with oximate) Complete destruction (full oxidation, thermal degradation)

8.2.4. "Biochemical decontamination"

Biochemical decontamination relies either on agent scavengers or on enzymes that can catalyze specific neutralization reactions. The main advantage over chemical reactions is that enzymes are selective and also exhibit catalytic effects (i.e. a single enzyme can perform the same decontamination reaction many times), whereas chemical reactants are normally consumed during each reaction. The main disadvantage, on the other hand, is the high specificity of the decontamination, which leads to different decontamination formulations for each group of agents. Moreover, agents containing toxic elements (e.g. arsenic) cannot be decontaminated with this method. Some agents might not be amenable to destruction by the highly specific biochemical methods. Both chemical decontamination methods and biochemical methods rely on a suitable medium (e.g. solvents, foams or emulsions with appropriate additives) to optimize solubility in the decontamination solution, to extract the agent from the substrates, and to retain the warfare agents in the decontaminant. An elegant application of the enzyme technology is to use the enzymatic reaction for indication of the liquid contamination. In that case, a mixture of enzymes is sprayed onto a possibly contaminated surface. The reaction that follows, together with a color indicator, will show if the substrate is seriously contaminated and where the contamination is.



Although promoted as a general purpose decontaminant, it is highly questionable whether it is generally applicable. A complex mixture of enzymes is required to work against all known CW and BW. If enzymes will work for an unknown agent, one that is not on the general lists is an open question. The enzymes will do a very nice job when a known single agent is involved. Choosing the appropriate surface tension of the medium, the enzymes might even be capable of destroying agent that resides in cracks and crevices. In the future, decontamination of materiel will become less important due to the very much reduced number of CW incidents.

## **8.3.** Contamination Avoidance

One or more of the following methods may result in contamination avoidance:

- a. Well-designed construction
- b. Appropriate choice of materials (hardening)
- c. Tactical means, such as mobility and reconnaissance
- d. Stand-off detection

In view of the fact that chemical or biological attacks will be an exception rather than the rule, it might be not cost-effective to spend attention and funding to develop contamination avoidant constructions or procure more expensive materials. Mobility, reconnaissance and detection methods also serve other purposes and can likely be easily adapted to contribute to contamination avoidance.

## 8.4. Decontamination, Procedures and Methods

Decontamination procedures today are both labor and time intensive. There are aggressive chemicals available e.g. NaOH in organic solvents that can destroy almost all agents rather quickly. At the same time, however, the chemicals are corrosive and destroy materials. An alternative to compounds with a general effect are those compounds acting against a limited group of agents. Typically these compounds act more swiftly against the chemical agents that they were designed to neutralize and have a milder effect on the contaminated materials. An example is chloramine solutions often used to decontaminate individuals and their personnel equipment. Chloramine works very well against mustard and VX but not against nerve agents of the G-type. Troops landing in Normandy in 1944 were wearing underwear impregnated with the chloramine type of decontaminant. They were well protected against mustard but lacked skin protection from compounds including Tabun, Sarin and Soman. A water solution of soda would have rendered those compounds harmless very quickly.

## 8.4.1. Decontamination of personnel

Decontamination of the individual is the most important aspect of the decontamination arena. As soon as a contamination of the skin is suspected, decontamination should commence. The main reason is that agents, especially liquid agents, rapidly penetrate the skin. Once absorbed

into the skin, it becomes much more difficult to render the compounds harmless. Time is of such great importance that the means used to decontaminate is not that critical. If applied within 1 minute after contamination, the efficiency of water and soap, absorbing powders such as flour and dirt, specially designed decontaminants or barrier creams are about equal. If the decontamination is delayed, it becomes important to use decontaminants that are capable of extracting agent out of the skin, e.g. RSDL which was developed in Canada.



The skin decontaminant of choice is the product from Canada RSDL, Reactive Skin Decontamination Lotion. It is the only product capable of extracting agent out of the skin.

It is still effective 30 min. after contamination

Decontamination procedures that are employed mainly consist of external scrubbing and showering operations, and/or the application of specific antidotes. Reactive barrier creams are appropriate in some cases. Decontamination of wounds, however, may require more delicate methods and can better be left to medical care.

## 8.4.2. Decontamination of equipment

Classic decontaminants typically contain a large excess of very aggressive chemicals, e.g. C8/A4 emulsion (German Emulsion), DS2, STB (super tropical bleach), HTH (high-test hypochlorite), and sodium carbonate. Strategies for future decontamination processes are based upon eliminating/minimizing the corrosive and/or components which are hazardous to the user or the environment.

#### 8.4.2.1. Large equipment

The procedures consist of spraying/rinsing the materiel with reactive liquids, either inorganic, organic or mixtures thereof, and the reactive component is an oxidant or nucleophilic solution, or in case of BWA, a disinfectant.

#### 8.4.2.2. Clothing

The procedures, if any are in use, are based upon washing or dry cleaning the contaminated clothing or upon applying diesel engine exhaust or other hot gases. Simultaneous application of heat and water vapor might accelerate hydrolysis. In the future with a much lower frequency of chemical attacks, it might be much more cost effective to replace contaminated clothing than to decontaminate it. Very light or non-contaminated clothing should be washed and made ready for repeated use. However, the protective properties of the clothes should not be compromised by the washing process.

#### 8.4.2.3. Sensitive equipment

At present, there are few procedures for the decontamination of sensitive equipment. The likelihood that this type of equipment will become contaminated is extremely small. It is most likely that the equipment will be replaced when contaminated.

#### 8.4.2.4. Decontamination of equipment of high value

Even if a nation decides that most of the equipment subject to contamination is expendable and shall not be decontaminated but replaced, there exist a number of mission-essential nonexpendable items (e.g. tanks, helicopters, C3I units, mobile air traffic control centers) which will require decontamination due to their high cost. It is extremely difficult to guarantee that the equipment is free of contamination once decontamination procedures have been completed. This thorough decontamination is also very costly. The decontamination of postal offices in the US after the Anthrax letters incident, cost twice the value of the total facility.

#### 8.4.2.5. Residual contamination

When a very low level of residual contamination is required, e.g. as in the case of repatriation, time is no longer a critical parameter. The diversity of materials that are part of the system may require the application of a multiple available decontamination techniques and possible disassembly of the system. This applies if there is no other technique available to address all possible contaminants, some of which may have migrated into the material or become trapped in interior compartments.

## **8.5.** Decontamination of Water

There are currently three processes used for military/mobile water purification plants. These are:

Destruction of agents by oxidation using chlorine, followed by treatment with activated carbon and filtration,

Distillation followed by charcoal filtration, and

One or more step membrane processes such as micro/ultra-filtration, followed by reverse osmosis.

While the first technology is long standing, the second is characterized by poor energy efficiency. Both are falling out of favor in preference to the last approach.

#### 8.6. Decontamination of Terrain

At present, only mission-essential terrain is decontaminated. Procedures used consist of spraying the terrain with chemical decontaminants such as aqueous solutions of calcium hypochlorite. More expedient procedures use bulldozers to remove or cover the top soil; however, CBW agents covered by soil are more protected from the effects of weathering.

With respect to BW agents, the decontamination of terrain can be extremely difficult. There are strong protective factors, such as encapsulation and the natural stability of some BW agents to persist against environmental factors, which normally prevent the complete inactivation of the BWA.

#### **8.7.** What is clean enough?

#### 8.7.1. Different perspectives

The question posed in the title of this paragraph is of interest for anybody involved in decontamination. An attempt will be made to formulate an answer to this question from different perspectives. Technical, military, medical, first responders and legal perspectives will be dealt with for several different situations in which decontamination in some form might be applied. The situations to be considered are for an armored vehicle/tank and protective clothing that both have gone through a contamination/decontamination cycle. Two forms of hazard will be reviewed, a respiratory hazard that might result from emanating vapors and contact hazards due to residual contamination.

#### 8.7.2. What is clean enough?

Unfortunately there are several definitions of the concept "clean". One definition is that there are absolutely no more contaminants, microorganism, molecules or particles in or on the substrate that was contaminated. In other terms the words "absolutely no more" are replaced by not "detectable" or not "effective". In the chemical/biological arena, clean is often defined as "No longer presents a hazard to the user". This definition is not accurate because the phrase "hazard for the user" is on a sliding scale. Due to lack of any better terminology, this definition will be used here.

"No longer presents a hazard to the user" is clearly a step further than an often previously used expression, "Does not seriously degrade the performance of the military".

The main reason for these rather ambiguous definitions is that "degradation in performance", "hazard" and "effect" or detectable levels are related to the toxicology of chemical agents and to the pathogenic action of biological agents. Biological agents are respiratory hazards 99.99% of the time, and after decontamination, the only effect to be considered is resuspension of the residual microorganism into the air. (There is only one situation known in which biological agents form a skin hazard, namely when wool sorters handle contaminated wool of sheep infected with anthrax, known as wool sorter's disease). Re-suspension of

biological agents into the air occurs in specific situations such as undressing, or driving a vehicle over a contaminated road or terrain. In those situations, respiratory protection is mandatory. The discussions will therefore be restricted to residual contaminations from chemical agents.

The question "What is clean enough?" can be asked for every contamination/decontamination cycle. The respiratory hazard from vapors emanating from a decontaminated armored vehicle can be derived from model calculations. The contact hazard can be derived from a similar model, but in some past cases, contact hazard was based on incorrect toxicological data which at the time were thought to be correct.

In chapter 4 more realistic toxicological data are given and will be used to derive the permissible residual level after protective clothing has gone through a contamination/ decontamination cycle. The technical answer to the question might thus be formulated, but a follow-on question is, "Can the operational military and military medical communities live with that answer?" It is even more difficult to assess the answer to the question from a legal or first responder perspective. Nevertheless, a review of available information will be accomplished to develop an answer for these non-military perspectives.

Because the toxicology of chemical agents plays such a vital role in answering the question, "What is clean enough?" the toxicity of CW agents toward humans described in Chapter 4 will be briefly reviewed.

## 8.7.3. Toxic effect levels of selected CW agents

The two types of agents of most interest for setting decontamination levels are nerve agents and blister agents. The toxicity of those agents will be briefly reviewed. All data are given as estimates and apply to a very limited range of exposure times and for young healthy males. Among young healthy males, variations in sensitivity exist toward the poison. Some individuals are more or much more sensitive than others. Many of the estimates use animal model studies as an input. The poisons are species specific, which add to the uncertainty in the numbers presented. There are many more caveats involved, and in using the numbers it is wise to involve an expert toxicologist. The table below was presented by the NATO group of experts on toxicity with some additions and comments from the Long Term Scientific Study LTSS.

For contact hazards, it is important to note that the permeability of the skin for the agents of concern vary widely over the human body, usually by two orders of magnitude. The groin and axillae have very thin skin and are highly permeable. Feet and hands have a relative thick skin and are relatively impermeable.

There appears to be a printing error in the NATO table for toxic effects of mustard agent, where 200 mg per man exposed on one side of the body (~1 m<sup>2</sup> skin) would correspond to more than 20  $\mu$ g/cm<sup>2</sup>. The values severe effects due to Lewisite are very much lower. There is no discrepancy in the vapor exposure data. It is generally understood that H and L are similarly potent skin agents. If that is accepted, a value on the order of 1  $\mu$ g/cm<sup>2</sup> for clinically noticeable mustard agent effects seems more appropriate. A value of 1-2  $\mu$ g/cm<sup>2</sup> for mustard agent agrees better with a WWII study by Nagy et.al. published in the *Journal of the American Chemical Society* in 1946. They found exposure to vapor resulting in the absorption of 3-6

 $\mu$ g/cm<sup>2</sup> gave severe erythema and sometimes vesication in the skin of trial subjects. The value of 1-2  $\mu$ g/cm<sup>2</sup> can be derived as well from field trials in humans by Dawson and Gilchrist. As cell, similar values are derived from studies using polyethylene films to model skin exposure.

Table 8.1. Effect levels for negligible effects due to inhalation (LTSS) or percutaneous exposure (NATO GOE and LTSS) Inhalation 10 min. exposure, percutaneous 30 min. exposure.

|                     | Inhalation                             | percutar                               | ieous                                       |
|---------------------|--|--|---|
| Agent               | Vapor (mg $\cdot$ min/m <sup>3</sup> ) | Vapor (mg $\cdot$ min/m <sup>3</sup> ) | Liquid (mg per man)                         |
| Tabun GA            | 0.5                                    | 3000                                   | No data                                     |
| Sarin GB            | 0.5                                    | 2000                                   | 300   |
| Cyclohexyl Sarin GF | 0.3                                    | 350                                    | 15  |
| Soman GD            | 0.3                                    | 350                                    | No data                                     |
| VX                  | 0.1                                    | 30                                     | 1.75(0.05 <sup>a</sup> )                    |
| Sulfur Mustard H    | 25                                     | 50 (50, 25) <sup>b</sup>               | $200^{\rm c} (1  \mu {\rm g/cm}^2)^{\rm c}$ |
|                     |  | Severe <200-500                        | Severe 600-800                              |
| Lewisite L          | No data, probably same as H: 25        | (No data)                              | (No data)                                   |
|                     | Sume us 11, 20.                        | Severe 400-500                         | Severe 5 $\mu$ g/cm <sup>2</sup>            |

<sup>a</sup> When applied on the skin of the cheek

<sup>b</sup> Values in between brackets are from LTSS, 50 for moderate temperatures and 25 for high temperatures

<sup>c</sup> H is locally acting. A value per cm<sup>2</sup> skin is more meaning full, 200 is probably a printing error.

## 8.7.4. Technical perspective, respiratory and eye exposure

Grabowski et al. (Wehr Wissenschaftliche Dienststelle der Bundeswehr, Munsterlager, Germany) described the hazard to a man standing at short distance downwind from a decontaminated tank in a wind of 1 m/sec and receiving the full dosage assuming that all residual agents were desorbed in the period that the man was present. The technicians have established about  $0.1 \text{ g/m}^2$  of mustard (H) remains as residual contamination after a standard decontamination. For nerve agents, the residual contamination is 10 times smaller.

Under normal environmental conditions, it takes several hours before residual H has fully evaporated. For the more volatile nerve agents like GB and GD, it takes approximately one hour. For the less volatile GF, it takes about the same time as mustard. The less volatile VX takes days to evaporate. In the model used by Grabowski, it was assumed that the agent was released in 15 minutes and that the man remained for this time downwind from the tank. It is important to note that the dosage is independent of the time it takes to release the agent as long as the man stays in the downwind area for the entire exposure time. The dosage received and the critical dosage as follows from the toxicity table is given in the table below:

| Agent Class | Maximum exposure                 | Exposure received after |
|-------------|----------------------------------|-------------------------|
|             | mg·min/m <sup>°</sup> , eyes and | decontamination,        |
|             | respiratory                      | mg·min/m <sup>°</sup>   |
| Н           | 25                               | 16.5                    |
| G           | 0.4                              | 1.65                    |
| VX          | 0.1                              | 1.65                    |

Table 8.2. Vapor hazard just downwind from a contaminated armored vehicle

In the extreme case as depicted above for a mustard contamination/decontamination cycle, the exposure stays below the clinically noticeable effect level. For G and VX, the exposure level is above the clinically noticeable effect level. It must be noted, however, that it will take days before the VX has emanated from the surface of the tank. Therefore, in general one could say that the respiratory hazards after decontamination are minimal.

In the previous century, one of the situations to be considered was a tank in typical nighttime conditions. (Night hide, a night hide is a more or less enclosed space with relatively low wind speeds). In such conditions wind speed is very low. Concentration is inversely proportional to the wind speed. A ten-fold lower wind speed would therefore result in a ten-fold higher exposure for the man in the vicinity of the tank. The relation breaks down as wind speed approaches zero. In that case, the exposure can be calculated from the solution of the differential equation described by Hembold (R.L. Helmbold, A General Mathematical Treatment of Hazards of NBC Collective Protection Systems with Application to Particular Cases. Naval Air Development Center, Warminster, PA, Report No NADC-82255-20).

The solution for this case is quite simple - the amount of agent brought into the night hide in mg divided by the ventilation flow rate in  $m^3/min$ . With a ventilation of 60  $m^3/min$ , the exposure dosage for H and nerve agents would be 165 and 16.5, respectively. With these dosages, one becomes clearly in the hazard zone. The solution is quite simple: Do not place vehicles after a contamination/decontamination cycle in an enclosed space. Even simpler is never stay in the vicinity of such a vehicle. Technically speaking, only in extreme cases will there be a slight hazard from emanating residual contamination. With adequate procedures these hazards can be reduced to negligible.

## 8.7.5. Technical perspective, contact hazard

The data from the toxicity table indicate that the contact hazard to a man exposed to  $1 \text{ mg/m}^2$  VX would result in clinically noticeable effects. For the locally acting H, it would be  $1 \mu \text{g/cm}^2$ . Based on a contact area of two hands (200 cm<sup>2</sup>) and a residual contamination of 10 mg/m<sup>2</sup> or  $1\mu \text{g/cm}^2$ , the total amount of agent that could act upon a person would be 200 µg, which is considerably below the 1.75 mg given in the NATO table. It is noteworthy that VX penetrates the cheeks much more rapidly, so contact of this body part with contaminated/decontaminated equipment should be particularly avoided. (Do not hug or kiss an armored vehicle that went through a contamination-decontamination cycle.)

Mustard agent with a residual contamination level of 10  $\mu$ g/cm<sup>2</sup> would definitely result in effects for sensitive skin but less likely for thick skin, such as for the palms. Therefore in this case, the hazards are minimal for the ordinary soldier. As well, it is unlikely that the entire agent will be released during the 15 minutes contact time.

It should be noted that many FINABEL studies (Joint investigations of France, Italy, Netherlands, Allemande, Belgium, England and Luxembourg) have led to conflicting data regarding the contact hazard. Of particular interest is a study from the Wehr Wissenschafliche Dienstelle in Munster, Germany, regarding the contact hazard from painted metal surfaces? It seems that polyurethane painted surfaces give rise to much lower hazard dosages than alkyd (oil-based) painted surfaces. These and other questions from the FINABEL studies have not been resolved.

Another contact hazard issue is the wearing of protective clothing that has gone through a contamination/decontamination cycle. Residual VX and G agents could be transported to the skin by perspiration and rain. Fuel spills are of particular concerns. In case of residual H contamination, this could lead to hazardous situations because fuel spills might easily involve sensitive skin areas like the crotch. Residual agent concentrations should be below  $1 \mu g/cm^2$ . A drop of 25 mg on 1 cm<sup>2</sup> must be reduced by a factor of 25,000. Larger drops would require an even higher decontamination reduction factor. The decontamination procedure for protective clothing must be 100 times better than that for an armored vehicle. It is questionable whether this can be achieved with active carbon-based protective clothing. The carbon is known to hold the agent strongly but it can be released by a fuel spill. To prevent such problems, it would be better to replace the suits with new ones. This becomes the more attractive option in the future as the frequency of chemical attacks and the mass involved will be reduced.

## 8.7.6. Technical perspective, biological hazard

At present, it is only possible to define a general goal for biological decontamination. Decontamination equipment should reduce the level of contamination from any given hazard to a level below that which will result in an infectious dose for that hazard, with an appropriate safety margin. The target level for decontamination will vary with each hazard and can only be defined after the hazard has been identified. The use of appropriate donning and doffing procedures for IPE are of special concern in the case of BWA contamination. A very serious respiratory hazard is created by the re-aerosolisation of microorganisms that have settled onto the clothing and become airborne again during donning and doffing of contaminated clothing.

## 8.7.7. Medical perspective

For the technical perspective, usually the clinically noticeable effect level for population 50<sup>th</sup> percentile is used. One of the fundamental aspects of the medical profession is the diversity among humans in their sensitivity toward diseases. Consequently, the emphasis is on the more highly sensitive individuals in a population. In terms of the dose-effect relationship, the 5<sup>th</sup> or even a lower percentile will be taken for the acceptable exposure level that will lead to clinically noticeable effects. The slopes of the probit curves for nearly all CW agents are rather steep. The dosages that would produce clinically noticeable effects in 5% of the population are therefore less than one order of magnitude below the 50<sup>th</sup> percentile. However, uncertainty in the dose-effect relationship increases at the extreme sides of the distribution,

and therefore in the medical perspective, the general trend is to set the clinically noticeable effect dosage at a level ten times below the  $50^{\text{th}}$  percentile level. The one order of magnitude rule might hold for most chemical agents, but for biological agents the slope of the probit curve on dose-effect relationships is usually far less steep. The 5% level might be two or three orders of magnitude below the 50% level. The slope of the probit curve for anthrax shows such behavior.

Another important consideration for the medical profession is whether or not there exists a therapy to cure the effects. Unfortunately there is hardly any therapy for mustard agent effects and for some of the nerve agents, e.g. the efficacy of GD therapy is questionable.

Based on the medical perspective compared to the technical perspective, "No longer a hazard for the user" is set at about a ten-fold lower for chemicals and more than 100-fold lower for biological agents.

#### 8.7.8. *Military perspective*

In broad terms, the military considers two types of operations: War and "Operations Other Than War" (OOTW) either involving the homeland or outside the area of direct interest.

In war, a balance exists between accomplishing a mission and the risk of losses in casualties and equipment. In assessing the risks of a chemical attack, followed by decontamination and the possible residual contamination, the military commander will follow the advice of his NBC officer and the medical staff. Most likely the NBC officer will base his advice on the technical perspective. In short, during war it is acceptable to take a small but calculated risk. "No longer is a hazard to the user" is a value derived from the technical perspective.

In OOTW, the rules are different. It is in general somewhat dependent on the type of operations, e.g. Afghanistan, Somalia, Georgia, Republic of Congo, Darfur, etc., but the risk acceptance has shifted to none or a very few casualties. In fact, in most peace keeping operations, the casualty acceptance is very low. In assessing the consequences of a chemical attack, the commander will be more geared toward the medical perspective than the technical perspective. Most of the troops taking part in OOTW missions do not have decontamination equipment available, which makes decontamination irrelevant.

When operating in support of first responders in the homeland, the military must follow the rules of the first responders despite the fact that the passive chemical defense practiced by the military is in many respects higher quality than that of the first responders.

## 8.7.9. First responder perspective

First responders operate under a zero-risk principal. Prevention of exposure of first responders to hazardous situations is of prime importance. This does not mean that there will never be casualties. Human error and unexpected situations (9/11 attacks) might cause severe casualties among first responders, but the casualty acceptance is very low.

As soon as CBRN terrorism against civilians was recognized as a possibility, the first responder community adopted the protective posture and procedures developed for accidents with hazardous industrial chemicals. It is important to note that in these types of accidents one of the prime hazards is contact with splashes of liquid. Another aspect is that high

concentrations of unknown toxic agents might be encountered. These two factors are the main reasons for choosing level A or B impermeable protective clothing combined with an independent breathing apparatus. In most cases, decontamination is carried out by using large quantities of water (sometimes warm or with soap). Clean is mostly defined as no longer detectable.

The complicated protective equipment is easy to decontaminate by showering, which is also effective for biological agent decontamination. Disadvantages include the high cost of the equipment, its limited availability, and the time required to don and doff. The operational time is limited by the limits in air supply and heat stress limits. With speed being the determining factor for successful rescue of chemical agent casualties, the time required to don complicated protective equipment is a severe detriment.

Second-line first responders are mostly equipped with normal (military type) gasmasks and plastic impermeable clothing. These types of clothing can be easily donned and doffed, and they are easily decontaminated from the outside by hose spraying. However, due to the bellows effect inherent to these suits, they offer a very poor protection against agent vapor and there will be a considerable ingress of biological aerosols that are not accessible for the decontamination and which can become airborne again during undressing.

There are unique factors associated with terrorist CBRN incidents. First, the quantity of agent involved is three orders of magnitude below the level that the military might face. Second, due to first responders arrive at the scene some time after release, the contamination levels that first responders will face is further decreased. If 1 kg of mustard agent is spread over an area of 100 by 100 m, the first responders would call it a hot zone, while the military would consider it a decontaminated surface. The residual amounts of Sarin in the trains and stations of the Tokyo Metro subway were in military terms negligible, however, both the trains and the stations were hosed down with water and steam, and the trains in the center of the attack were destroyed. The post office involved in the anthrax letter incident was contaminated with a maximum of 5 grams of anthrax spores. The decontamination operation was expensive costing 25 million dollars for a building worth half the price.

In short, first responders want a high degree of protection and a rigorous decontamination often for situations that the military regards as "decontaminated". For most situations encountered so far, "clean enough" was defined as not detectable or rigorously hosed down with water, steam and soap. Technical proof of the procedures employed was seldom provided.

It might therefore be wise for the first responder community to reconsider the passive chemical defense posture as a whole, in particular regarding individual protection. The availability of individual protective equipment varies widely, ranging from highly overdone to inadequate or at least far below military standards. For decontamination, first responders often use procedures without documented proof of efficacy. For example, it makes sense to decontaminate until agents are no longer detectable, but the absorption of chemical agents into substrates and subsequent emanation (e.g. compounds from plywood) has taught that it takes in-depth studies to define for the civilian sector which procedures lead to "clean enough".

#### 8.7.10. Legal perspective

After WWI, soldiers tried to receive compensation for being gassed. Some experienced significant morbidity, but in nearly all cases, claims were rejected because chemical warfare agents (CWA) were thought not to be disabling. In fact, in the scientific world, some people favored CWA because it was more humane than conventional weapons with a low crippling and death rate and "only temporarily disabling". As a result, those who were highly exposed to mustards and years later became blind had great difficulties being recognized as disabled veterans.

Between the two World Wars, several human volunteers were exposed to CW but again compensation was hard to get. During test production of mustard gas in the Netherlands in 1938, a few men working on sewage lines were accidentally exposed. This incident raised questions in Parliament, but after a short sick leave, the men returned to work.

Directly after WWII, workers from nerve agent production plants in Germany fought for compensation because of disabling effects due to accidental exposure during leakages in the production facility. Those cases making it to court were all rejected. The idea was that CWA would either kill or only temporarily disable. So those who survived had no right to compensation.

Many who had carried out human experiments with CWA in Japan or Nazi Germany escaped prosecution by providing the Allied forces with information. Examples are described in "War of Nerves" by Jonathan Tucker. It is only in the last decades that Japan has brought to trial some of the personnel working in Unit 731. Others have come forward as very old men to confess war crimes.

During and shortly after WWII, many human volunteer tests were conducted in order to establish incapacitating doses and to derive artillery firing tables. Some of these experiments resulted in fatalities, either directly as a cause of the experiment, e.g. in Porton Down, England, or later due to cancer believed to be late effects of mustard exposure (Australia). Both cases went to court, and in the case of the tragic death of the Air Force man in the UK, it took 50 years before the family received apologies and compensation from the government. The widow of the victim (he was the cameraman during the trials), in Australia, found the films that were made of men exposed to mustard gas and then crossing an obstacle course made of sharp bamboo sticks. Men badly blistered continued to cross the obstacle course three weeks after exposure. The dosage received was regarded as "clinically disabling". These trials are partly the basis for the common 500 mg·min/m<sup>3</sup> criterion used for vapor exposures in moderate climates. For warmer and more humid climates, where mustard agent was more effective, the values were set at approximately half this value.

In the US, it took two or three decades before the individual who carried out human volunteer CWA testing stood trial for these experiments. Although he did the experiments under contract with CRDEC and thus the Pentagon, he alone was held responsible and convicted. Because the US government was afraid of more claims from human volunteers, they refused to open up the files or make a clear statement about the toxicity of H and G and V agents based on the human volunteer experiments, or the data received from WWII German experiments.

During the Vietnam War, Agent Orange, now considered an official CWA, was frequently used to defoliate the forest occupied by the opposing forces without many precautions. Many of the US military who worked with the agent developed more or less serious effects. They sued the government and the manufacturer, and the servicemen won the case and were compensated.

In the Iran-Iraq War, tens of thousands of Iranian soldiers were attacked by nerve agent and mustard gas. The casualties that survived the onslaught received medical attention and were followed for years after the war. Still in the 21<sup>st</sup> century, Iran is proud to take care of their casualties and treats them as disabled war veterans.

Shortly after the Persian Gulf War, it appeared that about 10% of the US and UK soldiers that took part in the operations showed more or less severe after effects, later termed the Gulf War Syndrome. Some soldiers have attributed the syndrome to accidental exposure to very low concentrations of agent. (The low concentrations would have been generated due to destruction of production and storage sites of chemical warfare agent.) It is unknown whether the effects are a result of low-level exposures, and studies have been initiated to determine if the exposure alone or in combination with other medical treatment could cause of the syndrome. Nevertheless, the victims are under constant surveillance and are regarded as at least partly disabled veterans.

For the military and civilian populations, there has been a tremendous change in attitude toward chemical warfare agents during the past 100 years. Chemicals that were first seen as a blessing are now seen as a hazard. In many cases, victims of accidental exposure, especially toward toxic industrial compounds, have received compensation in nearly all incidents. The legal situation has changed from "The chemical agent is innocent until proven guilty" to "Guilty until proven innocent".

Science now faces two problems. First delivering proof that there was no exposure. This might be difficult if not impossible. The exposure during the Gulf War, if there was any, was minimal. Nevertheless the claim was made that this minimal exposure caused effects. An example of an extraordinary low exposure that still caused effects occurred during the anthrax letter incidents in the US. An elderly lady living at an appreciable distance from the contaminated post office (> 100 km) died of pulmonary anthrax exposure. It was established that she had not received any mail from the contaminated post office or that her mail was contaminated in any way. Airborne transmission of the spores was the only plausible cause and could not be more than one or a few spores, over 4 orders of magnitude below the LD<sub>50</sub>.

The second problem is the difficulty proving that low exposures will not induce any adverse effects. Many animal and even human volunteer experiments are required before one can prove that this is the case. With the current objections against animal experimentation, not to mention the human experiments, it becomes nearly impossible for the scientific community to prove beyond any doubt that the chemical or biological agent is not to blame. That being the case, the legal perspective defines clean as "No trace of contamination left."

## 8.8. CB Terrorism and Decontamination

The response to the asymmetric CB attack on military installations would be the same response as in war. The same technological responses should be adequate for the forces

involved. Standards of decontamination for the general population, however, may be more stringent. In this case, existing methods of decontamination may not be adequate or multiple (repetitive) decontamination operations may need to be performed. In the case of a CB attack against a civilian target, not directly involving military forces, any response would have to be at the request of the civilian authorities of the respective nation. Here again, technologies used for military response are still applicable, but standards for decontamination will be higher. It is unlikely that the military forces would have access to sufficient decontamination materials to handle a large civilian incident.

#### 8.9 Decontamination of Biological Agents, Disinfecting

The first problem to be solved in the decontamination of biological agents is to determine the location of the source. It is highly likely that a contaminated zone might only be identified when the outbreak of disease has become apparent. For instance, the sites to be decontaminated in the anthrax letter incidents in 2001 in the US were identified days after the first victims were identified. At the point of release, larger particles might be deposited and area decontamination or decontamination of personnel might be appropriate. However, even if the hot zone is identified, it is an enormous task to decide where in this zone decontamination should take place. After these decisions have been made, all-purpose decontaminants having 0.05-0.5% chlorine should be applied, but other disinfectants might be applied as well. Ultimately, it is possible that the total costs of identifying the hot zone and cleaning this zone is double the cost of the facility. Thoroughly destroying the facility might be a more cost-effective option, particularly so because it will be extremely difficult to guarantee that the facility is clean and can be used safely.

In order to prevent the spread of infectious diseases, many procedures have been developed to render microorganism harmless. The most effective way is by sterilization (destruction of the reproduction cycle), but this can often not be applied due to the aggressive methods required which would harm people and ruin equipment. It is also possible to use methods to reduce the number of pathogenic microorganisms to such a degree that they no longer present a hazard. This is called disinfection. Decontamination of biological agents means removal to such an extent that new infections are prevented. Decontamination through removal of agent is mostly achieved by mechanical methods such as cleaning with water and soap. A simple example of mechanical removal is filtration of water in order to make it potable.

All microorganisms are sensitive to heat and radiation to a certain degree. Heat treatment alone requires high temperatures >150 °C and long contact times (2 hours). Steam treatment is much more efficient and 15 minutes in steam of  $120^{\circ}$ C renders most pathogenic organisms harmless. However, such a procedure requires special equipment (autoclave) to achieve  $120^{\circ}$ C and two atmospheres of pressure. Some microorganisms, like spore-forming fungi and heat resistant viruses, will not be destroyed by this procedure.

Some bacteria are destroyed at an appreciable rate by the UV radiation in sunlight. Up to an 8 log decrease in viable bacteria is achievable in 2.5 hours of sunlight, whereas indoors it requires 10 hours to reduce the concentration of viable bacteria by one order of magnitude. Some spore-forming organisms, e.g. anthrax, are resistant to UV radiation.

The most frequently used methods of biological decontamination are based on chemical interactions with the microorganisms. There is a large arsenal of compounds that are active in

rendering pathogenic microorganisms harmless. They come in the form of liquid or solutions, gases and aerosols.

A well-known decontamination liquid is alcohol, and although most people would prefer to drink it, strong liquors can be used for decontamination of the skin in emergency cases. Most liquid decontaminants are based on solutions, e.g.:

| Phenol            | a few%   | not effective against spores and not very effective against |
|-------------------|----------|---|
| viruses           |          |   |
| Quaternary ammoni | ium 1%   | ibid  |
| Chlorohexidin     | <0.5%    | ibid  |
| Chlorine          | up to 5% | does not kill all types of spores                           |
| Iodine            | up to 2% | ibid  |
| Formaldehyde      | 3-8%     | effective against all!                                      |
| Glutaraldehyde    | 1-2%     | ibid  |

Some disinfectants can be used as gas or aerosol and are quite effective because they can reach many places.

| Ethylene oxide    | 400-1000 | g/m <sup>3</sup> effective against all |
|-------------------|----------|--|
| Betapropiolactone | 2-10     | g/m <sup>3</sup> ibid                  |
| Formaldehyde      | 3-10     | g/m <sup>3</sup> ibid                  |
| Glutaraldehyde    | 3-5      | g/m <sup>3</sup> ibid                  |

Contact times range from a few minutes for bacteria to several hours for the more difficult to kill spores. It should be noted that many of the disinfectants also react with organic residues, and therefore the objects to be cleaned must first be cleaned with water and soap.

Although effective disinfectants some of the compounds used are known or suspected carcinogenic e.g. Phenol and Ethylene oxide, consequently the necessary protective measures should be taken when these compounds are applied.

## Annex A. Chemical Warfare Agents, History, Use and Properties, Biological Warfare Agents

This Annex provides detailed information on the chemical and biological agents of concern, including relevant physical and chemical characteristics.

# A.1. Advantages and Disadvantages of the Use of Chemical Warfare Agents in a Military Conflict

## A.1.1. Advantages

Chemical weapons are anti-personnel weapons and do not induce material damage. This may provide an advantage, e.g. when infrastructure elements, such as roads, harbors and airports have to be taken intact. Accurate target acquisition is not necessary since chemical weapons may have an effect on a large area. They may harm an opponent who is operating largely dispersed or having some protection against explosive munitions, e.g. in trench lines or other fortifications.

Chemical warfare (CW) agents may differ in persistency and, accordingly, may remain harmful over different periods of time after dissemination. This offers the possibility to use more or less persistent agents depending on the intended tactical scenario. As an example, soil contaminated with a persistent agent can remain potentially harmful for days to troops attempting to cross it. In this way, an attacking force could screen its flanks whereas a defending force could hamper an enemy advance.

Finally, CW can be used as an element of surprise that will confuse and demoralize an enemy ("silent death"). Even the mere threat of CW will act as a disabling measure that reduces the fighting efficiency of an opposing force. Recipients of a potential chemical attack will be forced to take defensive measures and even the most efficient protective outfit (mask gloves, boots and protective clothing) is uncomfortable to wear after a short period of time. Additionally, a gas mask decreases combat efficiency by reducing vision, hindering communication, and even basic self-maintenance activities such as eating and drinking.

## A.1.2. Disadvantages

Chemical weapons require special and careful handling and storage if they are not to be a hazard to the users themselves. Many CW agents are corrosive and inclined to leak from their containers in due time.

Responsive dismantling and destruction of outdated stocks are very expensive due to the extensive measures needed to preclude contamination of the decommissioning work force and the environment. Both the storage hazard and the high costs of dismantling led to the development of binary chemical munitions.

CW agents generally cause casualties at a certain time period after exposure, except for the immediate action of some irritating agents and nerve agents. Specific information on onset times is given in the descriptions of the various agents. These delayed effects of CW agents may hamper the planning of their offensive use.

The effect of CW agents is unpredictable to a certain extent. The behavior of gas or aerosol agents cannot be controlled but depend on meteorological conditions (wind direction and

velocity, turbulence, temperature, air humidity) at the target area. This implies that an attack on military targets may also affect the unprotected civilian population especially when the actions are carried out in densely populated areas. The ultimate effect will be largely determined by the degree to which an opposing force is able to use protective measures.

[As this section of the document was being written, a clear example of the unpredictability of the cloud behavior was observed. A chimney generating black smoke under inversion conditions and low wind speeds appeared as a 1 km long black cloud in an easterly direction. The cloud probably was formed during the early morning hours. At the time of observation (around ten o'clock in the morning), some of the hot gases from the chimney penetrated the inversion layer and formed a cloud above the layer slowly moving in westerly direction. The main part of the cloud stayed below the ceiling and moved in easterly direction.]

Finally, the use of chemical weapons is prohibited by the Geneva Protocol of 1925. Most countries have ratified the protocol, although many countries restricted their ban to a non-first-use declaration. History has shown, however, that these politically restraining factors were insufficient to preclude chemical warfare on many occasions. The Chemical Weapon Convention (CWC) totally prohibits the use, production, and stockpiling of chemical warfare agents fall under a strict verification regime.

## A.2. Characteristics of Chemical Weapons

## A.2.1. Dispersion and Weapons

The first agents used such as chlorine and phosgene, are gases at ambient temperature and normal pressure. For this reason, chemical warfare agents are often called war gases. The original delivery system was a gas cylinder, which is still a viable system.

Most of the current CW agents, however, are liquids and solids. Technology has been developed to disperse liquids and solids as vapor, droplet (liquids), or aerosol form efficiently (Figure 1). Due to these modern developments, almost every weapon system can be equipped with chemical munitions. Chemical warfare agents may be delivered by aircraft wing mounted tanks or containers, from bombs, from artillery shells or by rockets. The quantity of chemical warfare agent may be less than a kilogram or up to several hundred kilograms depending on the weapon system used but every chemical weapon leaves a characteristic foot print. If no foot print can be detected in a incident it is highly unlikely that a chemical or biological weapon has been used.

## A.2.2. Binary Weapon System

A binary weapon system contains two relatively harmless starting compounds placed separately in the warhead. The starting compounds are automatically mixed after the binary weapon has been released from its delivery system. A highly toxic CW agent is then rapidly formed and subsequently dispersed. The US developed binary weapons for two nerve agents, Sarin and VX. The USSR developed binary munitions as well, such as the USSR form of VX and "Novichoks". The starting compounds and the associated chemical are described in the sections pertaining to corresponding nerve agents.



Figure 1. Dispersion of a chemical warfare agent after explosion of a warhead containing liquid agent.

This weapon system has the advantage of simplifying the storage and handling of the extremely toxic agents as well as the destruction of outdated stocks. Moreover, binary techniques make it possible to use unstable or non-storable chemical warfare agents. A disadvantage is the added complexity in comparison with conventional chemical munitions and therefore a greater risk of malfunction. Additionally, the binary process yields by-products, which become ballast and reduce the payload of the weapon system even in the case of a complete conversion of the starting compounds into the agent.

## A.2.3. Persistency

The wide range of volatilities and stabilities of chemical warfare agents toward moisture makes it possible to choose the duration of contamination of an area, i.e. the persistence of the agent after dissemination has taken place. Persistency may range from a few minutes, as for phosgene and hydrogen cyanide, up to several days or even weeks, as for mustard gas. Specific data on persistency are given in the description of the various agents.

Each tactical scenario requires agents with a specific persistency. For example, a nonpersistent agent may be employed when the agent delivering forces are intending to operate within the hostile zone immediately after the dissemination. The use of a persistent agent is required for denying an area to the opposing forces for prolonged periods. This suggests that it is conceivable to tailor a CW agent for a specific purpose. Persistency, however, depends considerably on a number of conditions such as meteorological conditions (wind, temperature and humidity) and the type of contaminated surface. The data collected in Table A.2.1 give an impression of the dependence of persistence on meteorological conditions for some nerve agents and mustard gas.

| Chemical<br>Agent | Sunny, little wind 15°C               | Windy, rain<br>10°C                  | Sunny, snow<br>-10°C |  |
|-------------------|---------------------------------------|--------------------------------------|----------------------|--|
| Sarin             | <sup>1</sup> / <sub>4</sub> - 4 hours | <sup>1</sup> / <sub>4</sub> - 1 hour | 1 - 2 days           |  |
| Tabun             | 1-4 days                              | $\frac{1}{2}$ - 6 hours              | 1 - 14 days          |  |
| Soman             | 2.5 – 5 days                          | 3 - 12 hours                         | 1 - 6 weeks          |  |
| Mustard gas       | 2 - 7 days                            | $\frac{1}{2}$ - 2 days               | 2 - 8 weeks          |  |
| VX                | 3 - 21 days                           | 1 - 12 hours                         | 1 - 16 weeks         |  |

Table A.2.1 Persistency of four nerve agents and mustard gas at various meteorological conditions

#### A.2.4. Thickeners and Mixtures

Physical properties of CW agents have been modified by the addition of thickeners, i.e. resinous polymers, or by mixing with a second CW agent. In comparison with neat agent, the drops produced from the more viscous thickened agents are approximately ten times larger. The larger drops have a higher free fall velocity and are less affected by wind and diffusion. Therefore, thickening facilitates the controlled dispersion of a CW agent to restrict the contamination to a desired area. Dispersion can be carried out at a greater altitude without major secondary contamination due to drifting of agent clouds. The adhesive capacity of the CW agent will also be improved by thickening, which makes contaminated surfaces much more difficult to decontaminate. An obvious disadvantage of the addition of a thickener is the decrease in effective loading of a warhead with toxic agent.

A well-known chemical warfare agent mixture is the combined blistering agents mustard gas and Lewisite. Mustard gas has a relatively high melting point (14 °C), whereas Lewisite is very unstable under humid conditions. Adding Lewisite to mustard gas gives a low-freezing mixture with reasonable persistence for use in cold weather operations or as a high-altitude spray.

## A.3. Properties of Chemical Warfare Agents

#### A.3.1. Required and Desirable Properties

To be effective, a CW agent must produce damaging or lethal effects on individuals, animals or plants, or must at least disable individuals from the performance of their assigned tasks. Toxicity, therefore, is an important characteristic but certainly not the only requirement to be met by a CW agent. Others include:

- The agent must be producible on a large scale at a low cost.
- It must be stable in storage, at the conditions present during dissemination, e.g., high temperatures at explosive dispersion, as well as with respect to environmental conditions, particularly moisture.

- It should preferably have little or no corrosive action on munitions or containers during storage.
- Finally, detection and protection against the agent should be very difficult, and it should be difficult to decontaminate. Although many CW defense aspects are highly selective, such as detection and medical countermeasures, physical protection is based on the laws of physics and works under all circumstances and to a degree for all agents.

Because of these requirements, only a limited number of toxic chemicals have been produced for use as CW agents. Moreover, the process to be pursued for achievement of a military and political decision to produce a CW agent, as well as logistic considerations, have contributed to a limitation of the number of chemical warfare agents incorporated into military arsenals. The relevant compounds are mentioned in the schedules of the verification annex of the CWC.

#### A.3.2. Classification of Chemical Warfare Agents

Chemical warfare agents may be classified in many ways, such as their physical state (solid, liquid, gas) or their persistency (non-persistent, semi-persistent, and persistent). In this chapter, the common classification according to use and physiological effect will be followed:

Lethal agents choking blood poisoning nerve toxins Incapacitating agents physical incapacitants psychogenic incapacitants Irritating tear gases vomiting agents Herbicides

The main division is made based on the intended use of the agent. The lethal, incapacitating and irritating agents are subdivided according to their physiological effects. Toxins will be dealt with as a separate group of poisonous products from microorganisms, plants or animals. Two toxins are mentioned in the schedules of the verification annex of the CWC, however most people regard toxins as part of the Biological Weapon Convention, because they were originally from biological origin.

Irritating agents and herbicides are not listed in the schedules contained in the CWC, although riot control agents, which are mostly irritating agents, are mentioned in Article I, II and III. The situation is confusing in that in Article I tear gas is forbidden as a form of warfare. (Sometimes this is interpreted as: All other ways of using irritating compounds, e.g. riot control in a POW camp or during UN peacekeeping missions, are not prohibited by the CWC.) However, in Article II, it is stated that all agents which have an effect on the human physiology are regarded as chemical warfare agents and therefore forbidden, except for uses permitted by the CWC. Permitted uses are for riot control and law enforcement. (Sometimes this is interpreted as: Every use of irritating agents is forbidden except the forms permitted

under the CWC). So for riot control agents, Article I is interpreted as "permitted unless forbidden", and Article II and III are interpreted as "forbidden unless permitted" In between those interpretations, there is room for debatable applications of irritating agents.

## A.4. Properties of Specific Agents.

Where available the general information and physical and chemical properties are provided in the description of each individual CW agent. Other than a general statement, physiological and toxicological properties are provided separately in Chapter 4.

#### General information

In addition to the chemical name and structure, the generally used US code for the agent is given as well as its Chemical Abstracts Service registry number (CAS). Each compound mentioned in Chemical Abstracts is labeled with a unique number, its registry number. The use of the registry number makes literature searches on the compound in Chemical Abstracts much more convenient.

## **Physical and Chemical properties**

The physical state at ambient temperature, melting point, boiling point, density, stability in storage and persistency will be given, if available. Furthermore, two additional important properties of a CW agent will be considered: volatility and rate of hydrolysis.

Volatility is defined as the weight of vapor present in a unit volume of air (concentration) at a specific temperature, under equilibrium conditions between the vapor and it's liquid or solid. Volatility is generally expressed as milligrams of vapor per cubic meter. A property directly related to volatility is the pressure exerted by a vapor under equilibrium conditions, the vapor pressure, expressed in millimeters of mercury (1 mmHg = 133.322 Pascal). The relation between volatility and vapor pressure at a given temperature is roughly given by

Volatility (mg.m<sup>-3</sup>) = vapor pressure (mmHg) x molecular weight  
$$760x22.4 \times 10^{-6}$$

Hydrolysis of a CW agent, i.e. its reaction with water, mostly yields compounds of much lower toxicity. It should be kept in mind, however, that a toxic product might be formed, as in the case of agents containing arsenic, such as Lewisite, and the nerve agent VX. For a number of CW agents, the rate of hydrolysis is highly dependent on the pH. The rate of hydrolysis is very important in chemical operations: rapid hydrolysis results in low persistency. If quantitative data are given, they are presented as a half-life time, i.e., the time period in which half of the amount of the compound is hydrolyzed.

## Physiological and toxicological properties

A short description will be given of the symptoms induced by an agent and its mode of action, if known. Also, the rate of action, i.e., the rate at which symptoms become apparent after exposure, will be given. The rate of action may widely differ for various CW agents. Knowing this rate of action is important both for an aggressor and the defender. The aggressor

needs the information to plan his battle tactics and the defender with regard to diagnosis and the period of time in which medical countermeasures must be taken.

A simple distinction between toxic and nontoxic compounds cannot be made. An important characteristic of a CW agent is the dose at which it causes a physiological effect.

This dose may depend on the route by which the agent enters the body. The three natural routes are inhalation, absorption through the skin or eyes, and ingestion. Gases, as well as liquid and solid aerosols, can be taken up via the respiratory organs. Liquids, and in certain cases gases and aerosols, invade the body via the skin and eyes. Skin and eye effects, however, are usually local, although blistering agents can cause systemic intoxication. The ingestion route is less important from the point of view of chemical warfare.

#### A.4.1. Lethal Agents

Lethal agents are capable of producing incapacitation, serious injury or death when used in field concentrations.

#### A.4.1.1 Choking Agents

Choking agents injure an individual mainly in the respiratory tract. In severe intoxications, edema of the lung develops, i.e. an abnormal accumulation of liquid in the lung tissue, and blisters form in the respiratory tract which burst to release blood and liquid. The edema hinders the exchange of gas in the lungs. Finally, victims may be choked by the agent due to lack of oxygen.

#### (i) **Phosgene**

Chemical name: carbonyl dichloride. Code: CG, after first production site in France CAS registry number: 75-44-5.

Phosgene was used extensively in the First World War. More than 80% of the fatal casualties due to CW agents were caused by phosgene. It may be disseminated in very high vapor concentrations, and it is still attractive as a CW agent. Currently, it is manufactured worldwide on a large scale for industrial purposes.

Phosgene is listed as a Schedule 3 chemical in the Chemical Weapons Convention.

| Physical state (20 °C): | gas  |
|-------------------------|--|
| Odor:                   | freshly cut hay  |
| Melting point:          | -120 °C  |
| Boiling point:          | 7.6 °C   |
| Density:                | of the vapor, 3.4 (compared to air); of the liquid, $1.37 \text{ g.ml}^{-1}$ (20 °C) |
| Volatility:             | 2,200,000 mg.m <sup>-3</sup> (-10 °C), 4,300,000 mg.m <sup>-3</sup> (7.6 °C)         |
| Vapor pressure:         | 365 mmHg (-10 °C), 1173 mmHg (20 °C)   |
| Rate of hydrolysis:     | rapidly hydrolyzed under field conditions, by rain and on leafy surfaces             |

Stability on storage:stable in steel containers if it is dry; corrosive when it is moistPersistency:short

#### (ii) **Diphosgene**

Chemical name: trichloromethyl chloroformate. Code: DP. <u>DiP</u>hos CAS registry number: 503-38-8.

Diphosgene has a much higher boiling point than phosgene. It is slightly lacrimatory and has therefore less surprise value than phosgene.

The compound is not incorporated into one of the Schedules of Chemicals contained in the Chemical Weapons Convention.

| Physical state (20 °C): | liquid  |
|-------------------------|---|
| Odor:                   | freshly cut hay   |
| Melting point:          | -57 °C  |
| Boiling point:          | 127-128 °C  |
| Density:                | of the liquid, 1.65 g.ml <sup>-1</sup> (20 °C)                          |
| Volatility:             | 45,000 mg.m <sup>-3</sup> (20 °C)                                       |
| Vapor pressure:         | 4.2 mmHg (20 °C)  |
| Rate of hydrolysis:     | slow at ordinary temperatures   |
| Stability on storage:   | unstable; conversion to phosgene. The conversion is catalyzed by metals |

#### (iii) **PFIB**

 $(CF_3)_2C=CF_2$ 

Chemical name: 1, 1, 3, 3, 3-pentafluoro-2-(trifluoromethyl)-prop-1-ENE CAS registry number: 382-21-8.

The gas PFIB produces pulmonary edema. It is generated when the plastic polytetrafluoroethene is pyrolized. PFIB is listed as a Schedule 2 chemical in the Chemical Weapons Convention.

| Physical state (20 °C): | colorless gas                          |
|-------------------------|--|
| Odor:                   | none                                   |
| Melting point:          | -130 °C                                |
| Boiling point:          | 5-6 °C                                 |
| Density:                | of the vapor, 6.95 (compared with air) |
| Volatility:             | 9,000,000 mg.m <sup>-3</sup> (5 °C)    |
| Vapor pressure:         | 1260 mmHg (20 °C)                      |
| Rate of hydrolysis:     | very slow                              |
| Persistency:            | short                                  |

#### (iv) Chloropicrin

 $Cl_3CNO_2$ 

Chemical name: trichloronitromethane. Code: PS, <u>Port S</u>unlight, first production site in UK CAS registry number: 76-06-2.

Chloropicrin was used in the First World War. The compound is used in the chemical industry and is used as a pesticide. It has a relatively low melting point (-69  $^{\circ}$ C). It can be mixed readily with other chemical agents, such as mustard gas and nitrogen mustard. Adding chloropicrin to these agents lowers the melting point to provide a low-freezing mixture for cold weather operations. Chloropicrin is listed as a Schedule 3 chemical in the Chemical Weapons Convention.

| Physical state (20 °C): | colorless oily liquid  |
|-------------------------|--|
| Odor:                   | characteristic pungent smell   |
| Melting point:          | -69 °C   |
| Boiling point:          | 112-113 °C   |
| Density:                | of the vapor, 5.7 (compared to air); of the liquid, $1.66 \text{ g.ml}^{-1}$ (20 °C) |
| Volatility:             | 184,000 mg.m <sup>-3</sup>   |
| Vapor pressure:         | 17 mmHg (20 °C)  |
| Rate of hydrolysis:     | not measurable   |
| Persistency:            | short  |

A.4.1.2. Blood Poisoning Agents

The blood poisoning agents, hydrogen cyanide and cyanogen chloride, interfere with the cell respiration (the utilization of oxygen by the cells) due to interaction with the enzyme cytochrome oxidase. Symptoms may set in extremely rapidly. Their effects depend strongly on the concentration to which a casualty was exposed since the agents are relatively rapidly detoxified in the body. The agents are considered to be of relatively low relevance to CW because of their high volatility in combination with a moderate toxicity. However, hydrogen cyanide is used on a large scale in the chemical industry. Moreover, protection of the respiratory tract from the agents is difficult, particularly in the case of cyanogen chloride. Hydrogen cyanide and cyanogen chloride are listed as Schedule 3 chemicals in the Chemical Weapons Convention.

#### (i) Hydrogen Cyanide

HCN

Chemical name: hydrogen cyanide. Code: AC, from the French <u>A</u>cide <u>Cyanique</u> CAS registry number: 74-90-8.

Physical state (20 °C):liquidOdor:bitter almondsMelting point:- 13 °C

| Boiling point:        | 26 °C   |
|-----------------------|---|
| Density:              | of the vapor, 0.93 (compared to air); of the liquid, 0.687 g.ml <sup><math>-1</math></sup>  |
| Volatility:           | $1,080,000 \text{ mg.m}^{-3} (25 \text{ °C})$   |
| Vapor pressure:       | 742 mmHg (25 °C)  |
| Rate of hydrolysis:   | low under field conditions.   |
| Stability on storage: | forms explosive polymers; stabilization by addition of phosphoric acid<br>or sulfur dioxide |
| Persistency:          | short   |

#### (Ii) Cyanogen Chloride

CNCl

Chemical name: cyanogen chloride. Code: CK CAS registry number: 506-77-4.

Cyanogen chloride is more volatile than hydrogen cyanide and, unlike the latter agent, can produce casualties in sub lethal doses. In addition to the effects caused by hydrogen chloride, Cyanogen chloride has a powerful lacrimatory action and a choking effect. The agent penetrates the filter elements of gas masks more readily than most other agents.

| Physical state (20 °C): | gas  |
|-------------------------|--|
| Odor:                   | pungent smell masked by its lacrimatory action                                       |
| Melting point:          | -7 °C  |
| Boiling point:          | 12.8 °C  |
| Density:                | of the vapor, 2.1 (compared to air); of the liquid, $1.18 \text{ g.ml}^{-1}$ (20 °C) |
| Volatility:             | 3,300,000 mg.m <sup>-3</sup> (20 °C)   |
| Vapor pressure:         | 1000 mmHg (25 °C)  |
| Rate of hydrolysis:     | very low   |
| Stability on storage:   | tends to undergo condensation and polymerization                                     |
| Persistency:            | short  |

A.4.1.3. Nerve Agents

Nerve agents belong chemically to the group organophosphorus compounds or organophosphates.

The biologically active organophosphates can be denoted by the general structure  $R_1(R_2)P(O,S)X$ , where  $R_1$  and  $R_2$  are either alkoxy, aryloxy, alkylmercapto, (alkyl)amino, alkyl or aryl groups, and X is a residue easily liberated upon a nucleophilic attack on phosphorus, such as fluoride, chloride, cyanide, phosphate, phenolate or thiolate. Nerve agents contain a P=O moiety. In insecticides this is often replaced by P=S and X consists of a less reactive group than in the case of a nerve agent. Examples of organophosphorus insecticides are malathion, parathion and dichlorphos.

The first nerve agent compound later referred to as a nerve agent, Tabun, was prepared by Schrader in 1936. As already mentioned, the nerve agents Tabun and Sarin were produced for employment as chemical weapons in Germany during the Second World War. These agents together with Soman, prepared in 1944, belong to the so-called G agents. A group of even

more toxic nerve agents, the V agents, was developed in the 1950s. These agents are more stable and less volatile than the G agents and are consequently more persistent.

The toxicity of nerve agents (and of the organophosphorus insecticides) is mainly due to their inhibitory effect on the enzyme acetylcholinesterase. This enzyme acts as a highly active catalyst for the hydrolysis of acetylcholine, a transmitter substance responsible for the transfer of nerve impulses across the cholinergic synaptic junctions. Inhibition of the enzyme causes accumulation of acetylcholine which leads to over-excitation or paralysis depending on the type of cholinergic junction involved. Over-excitation leads to the following symptoms: increased production of saliva, a running nose, difficult breathing due to secretion in the respiratory tract, increased perspiration, vomiting, diarrhea, involuntary discharge of urine and defecation, cramps and convulsions. The most critical effects are paralysis of the respiratory muscles in combination with the effects on the respiratory center in the central nervous system. Nerve agents also have effects on the central nervous system producing symptoms such as anxiety and stress. Death is generally caused by respiratory paralysis.

A typical symptom of intoxication by nerve agents in the form of gas or aerosol is miosis, the contraction of the pupil, which can be induced at very low doses. This results in impaired night vision. Nerve agents are much more toxic when absorbed by the eye than through the skin.

Respiratory lethal doses and liquid agent in the eye kill in 1 to 10 minutes. After skin contamination, symptoms do not generally appear for 30 minutes. Death may be delayed for 1 to 2 hours.

Four nerve agents have been weaponized: the G agents Tabun, Sarin and Soman, and the V agent VX. The nerve agents represent a chemical threat that is still not surpassed by any other group of CW agents. This is only partially due to their extremely high toxicity and ability to rapidly induce toxic effects. They are stable, easy to disseminate and relatively simple to produce from cheap starting materials. They may enter the body by inhalation as gases or aerosols and through the skin and the eyes as a liquid. They have various persistencies ranging from the low persistency of Sarin to the relatively persistent V agents.

The nerve agents are listed as Schedule 1 Chemicals in the Chemical Weapons Convention.

(i) Tabun

Chemical name: ethyl N,N-dimethylphosphoramidocyanidate. Code: GA. First produced German nerve agent, Tabun originates from Taboo, Tabu in German (Ref 18a chapter 3) CAS registry number: 77-81-6.

Tabun was produced on a large scale during the Second World War. It is the least toxic member of the four well-known nerve agents.

| Physical state (20 °C): | colorless to brownish liquid  |
|-------------------------|---|
| Odor:                   | faintly fruity  |
| Melting point:          | -50 °C  |
| Boiling point:          | 108 °C (12 mmHg)  |
| Density:                | of the vapor, 5.63 (compared with air); of the liquid, 1.073 $\text{g.ml}^{-1}$ (25 °C)                                   |
| Volatility:             | 610 mg.m <sup>-3</sup> (25 °C)  |
| Vapor pressure:         | 0.070 mmHg (25 °C)  |
| Rate of hydrolysis:     | half-life time in distilled water, 9 hours; autocatalytic hydrolysis below pH 4; rapid hydrolysis in strong acid and base |
| Stability on storage:   | stable at ordinary temperatures   |
| Persistency:            | see Table A.2.1; heavily splashed liquid persists 1-2 days under average weather conditions(ii)                           |

#### Sarin

Chemical name: isopropyl methylphosphonofluoridate.

Code: GB. Second produced German nerve agent, Sarin originates from <u>S</u>chrader, <u>A</u>mbros, <u>R</u>udiger and L<u>in</u>dner (ref 18a chapter 3) CAS registry number: 107-44-8.

Sarin has been heavily stockpiled. It is the most volatile nerve agent. A binary weapon system has been developed for Sarin. In this system, Sarin is formed from  $(CH_3)_2CHOH$  (code: IP) and  $CH_3P(O)F_2$  (code: DF). Hydrogen fluoride is formed as a by-product, which is highly

irritating to mucous membranes and can accelerate the hydrolysis of formed Sarin.

Physical state (20 °C): colorless liquid

| Odor:                 | none  |
|-----------------------|---|
| Melting point:        | -57 °C  |
| Boiling point:        | 151 °C (decomposition)  |
| Density:              | of the vapor, 4.86 (compared to air); of the liquid, $1.0887 \text{ g.ml}^{-1}$ (25 |
| Valatilitan           | $22,000 \text{ mg m}^{-3}(25,\%)$   |
| volatility:           | 22,000 mg.m (25 °C)   |
| Vapor pressure:       | 2.9 mmHg (25 °C)  |
| Rate of hydrolysis:   | half-life time of 175 h at pH 4-6.5; rapid hydrolysis at low pH, very               |
|                       | rapid in alkaline solutions   |
| Stability on storage: | fairly stable; slightly corrosive to steel  |
| Persistency:          | see Table A.2.1; evaporates at approximately the same rate as water                 |

(iii) Soman

Chemical name: 1,2,2-trimethylpropyl methylphosphonofluoridate. Code: GD. third produced German nerve agent, origin of the name Soman unknown. GC is missing from the series because either it could be confused with CG, the code for phosgene (ref. 18a chapter 3) or in medical community it stands for Gonorrhea Coccus. CAS registry number: 96-64-0.

Soman is notorious for its resistance to the standard medical treatment for nerve agent intoxication.

| Physical state (20 °C): | colorless liquid   |
|-------------------------|--|
| Odor:                   | fruity; odor of camphor when impure  |
| Melting point:          | -42 °C   |
| Boiling point:          | 85 °C at 15 mmHg   |
| Density:                | of the vapor, 6.33 (compared with air); of the liquid, 1.0222 g.ml <sup>-1</sup> (25 |
|                         | °C)  |
| Volatility:             | 3,900 mg.m <sup>-3</sup> (25 °C)   |
| Vapor pressure:         | 0.40 mmHg (25 °C)  |
| Rate of hydrolysis:     | rapid hydrolysis in acid and alkaline solutions                                      |
| Stability on storage:   | less stable than Tabun and Sarin; slightly corrosive                                 |
| Persistency: see Tabl   | le A.2.1   |

(iv) VX

 $\begin{array}{c} C_2H_5O \\ H_3C \end{array} \begin{array}{c} O \\ SCH_2CH_2N \\ CH(CH_3)_2 \\ CH(CH_3)_2 \end{array}$ 

Chemical name: S-2-diisopropylaminoethyl ethyl methylphosphonothioate. Code: VX. CAS registry number: 50782-69-9.

VX is the standard V agent. It is much less volatile than the G agents, but it is more toxic, particularly as a liquid on the skin. A binary weapon system has been developed for VX in which  $C_2H_5O(CH_3)POCH_2CH_2N(i-C_3H_7)_2$  (US code: QL) and  $CH_3(S)_nCH_3$  (US code: NM) are the starting compounds. The former compound is relatively toxic; the latter is an extremely foul-smelling liquid. Mixing of the starting compounds yields VX and sulfur-containing by-products.

Physical state (20 °C):amber-colored oily liquidOdor:noneMelting point:< -51 °C</td>

| Density:              | of the vapor, 9.2 (compared with air); of the liquid, $1.0083 \text{ g.ml}^{-1}$ (25 |
|-----------------------|--|
|                       | °C)  |
| Boiling point:        | 298 °C (calculated; decomposes)  |
| Volatility:           | 10.5 mg.m <sup>-3</sup> (25 °C)  |
| Vapor pressure:       | 0.0007 mmHg (25 °C)  |
| Rate of hydrolysis:   | 100 days at pH 2-3; 1.3 min at pH 14 (25 °C)   |
| Stability on storage: | relatively stable  |
| Persistency:          | see Table A.2.1. Heavily splashed liquid persists for long periods of                |
| -                     | time under average weather conditions  |

#### A.4.2. Toxins

Agents considered to be toxins used to be limited to compounds produced by living organisms but some can be synthesized from basic chemicals. Toxins may be extremely toxic with some being considerably more toxic than nerve agents. The 1975 Convention on Biological Weapons prohibits all development, production and stockpiling of biological agents and toxins. Two toxins, i.e., saxitoxin and ricin, are also included in as Schedule 1 chemicals in the Chemical Weapons Convention.

In the early 1980s, the US concluded in a number of reports presented to the United Nations that mycotoxins were used in Laos and Kampuchea. The alleged use of these agents, known as "yellow rain", could neither be confirmed nor disregarded. These accusations of possible use, however, focused attention on toxins as possible CW agents, at least temporarily. More recent evidence from India and various US sources support the hypothesis that "yellow rain" was most likely simply bee feces.

Recently, renewed interest emerged due to scientific and technological developments during the last decade. At the time the Convention on Biological Weapons was established, most toxins were obtained by extraction from biological materials, making large-scale production difficult. Modern techniques offer the possibility for synthesis of large quantities of certain toxins, particularly toxins with a peptide structure. With these techniques available, synthesis of derivatives or of slightly modified analogues of toxins has become feasible. The question then arises whether modified synthetic toxins are still covered by the Biological Weapons Convention or whether they should be categorized as chemical warfare agents.

Therefore, some attention is paid to toxins in this chapter. Four toxins have been stockpiled for use as CW agents or have been closely studied as candidate CW agents. Three of these toxins are dealt with in this section. The fourth toxin, Staphylococcus enterotoxin B, falls into the category of the physical incapacitating agents and will be described together with the other members of this class of agents (vide infra). In addition, properties of one representative of the mycotoxins will be described in this section.

#### (i) Botulinum Toxin A

The toxin is produced by the *Clostridium Botulinum* bacterium, which grows and forms its toxin under anaerobic conditions, e.g. in improperly preserved food. Six types of Botulinum toxins are known of which type A seems to have attracted the most military interest. Relatively straightforward techniques permit large-scale production of the toxin. Botulinum toxin A is a white crystalline protein which is stable in storage for long periods provided air

and moisture are excluded. It is stable for a week in non-moving water and persists for a long time in food when not exposed to air. The toxin is rapidly destroyed by boiling or by sunlight.

Botulinum toxin A is one of the most poisonous substances known and is considerably more toxic than the nerve agents. It rapidly decays, however, in the open air and is therefore more likely to be used in operations where it is not exposed to the atmosphere for a prolonged period of time, such as sabotage of water or food supplies.

The compound is not incorporated into one of the Schedules of Chemicals contained in the Chemical Weapons Convention.

## (ii) Saxitoxin

Saxitoxin (CAS registry number 35523-89-8) is a lethal toxin produced by algae of the dinoflagellate plankton *Protogonyaulax*. Mussels and other shellfish that live on these algae can store the toxin without harm to themselves. Consumption of shellfish contaminated with the toxin induces paralytic shellfish poisoning. The toxin can be extracted from cultures of toxin-producing species of Protogonyaulax in high yield. It is a white solid with the following structure.



It is readily soluble in water and attracts water from humid air. It is resistant to heat, but susceptible to oxygen.

It is difficult to disperse saxitoxin on a large scale. Instead, rifle-fired flechettes have been developed that can inject saxitoxin into a man with no more sensation than that of a mosquito bite. Death may then follow in less than 15 minutes.

The agent is listed as a Schedule 1 chemical in the Chemical Weapons Convention.

## iii) Ricin

The beans of the castor plant, *Ricinus communis*, contain the extremely toxic ricin in addition to castor oil. The toxin is a protein. It is soluble in water and stable at room temperature. The toxin is denatured at elevated temperature.

Ricin was patented in 1953 in the US for use as a CW agent. It can be disseminated as an aerosol. At the end of the 1970s, ricin was used in the Bulgarian umbrella murders. With the aid of a special umbrella, 1-mm bullets impregnated with ricin were injected into a victim who died approximately 24 hours later.

The agent is listed as a Schedule 1 chemical in the Chemical Weapons Convention.

## (iv) **T-2 Toxin**

T-2 toxin is one of the trichothecenes, a group of mycotoxins produced by fungi, such as *Fusarium* and *Trichothecium*. In the beginning of the 1980s, the US reported the presence of trichothecenes in samples from Southeast Asia that were said to originate from areas subjected to biological attacks. As already mentioned, definite proof for the alleged use could not be presented.

T-2 toxin is a white solid with the following structure:



T-2 toxin is readily soluble in lower alcohols and slightly soluble in water. It is stable in storage. Its thermal stability is illustrated by the modest degradation during the baking of bread which is contaminated by the toxin. T-2 toxin is detoxified in acidic and alkaline solutions and by prolonged boiling in aqueous solution.

Grain that is stored improperly, especially under wet and cold conditions, may be infected by trichothecenes-producing molds. Consumption of such grain can cause severe intoxication in humans. The outbreak of the often fatal Alimentary Toxic Aleukia (ATA) in Siberia at the beginning of the 1940s was caused by trichothecenes. One of the investigators of ATA worked for years with the toxins producing large quantities of mixtures containing T2 in a poorly equipped laboratory without protective means (Prof. Joffe, Hebrew University, directly across from the Israeli Parliament building). He never reported any skin effects and died at a very old age.

The compound is not incorporated into one of the Schedules of Chemicals contained in the Chemical Weapons Convention.

## A.4.3. Incapacitating Agents

Incapacitating agents temporarily disable individuals from performing their assigned duties without causing death or permanent damage. Consequently, the lethal dose of an incapacitating agent should be high in relation to its incapacitating dose, i.e. these agents should have a high safety ratio. Their effects appear shortly after intoxication and last for a significant period of time. During the 1950s and 1960s a number of compounds were considered as potential incapacitating agents. Physical and psychogenic incapacitating agents may be distinguished.

## A.4.3.1. Physical Incapacitating Agents

Physical incapacitating agents may cause, for example, temporary blindness, paralysis, or vomiting and diarrhea, as caused by Staphylococcus enterotoxin B. Also blistering agents, such as mustard gas and Lewisite, can be considered incapacitating agents although their

safety ratio is relatively low. During the First World War, less than 2% of the total casualties inflicted by mustard gas were fatal. Most of the deaths were due to a respiratory intoxication causing irreparable lung damage. Some blistering agents and the Staphylococcus enterotoxin B will be considered.

## (i) Staphylococcus Enterotoxin B

Staphylococcus enterotoxin B is produced by *Staphylococcus aureus* and is responsible for symptoms of food poisoning that follow consumption of food contaminated by Staphylococcus bacteria. The toxin has a specific action on the cells of the intestinal mucosa and is therefore called an enterotoxin. It is a white protein that attracts moisture from humid air and readily dissolves in water. Certain strains of *Staphylococcus aureus* generate the toxin in large and easily isolable yields. It is resistant to freezing and boiling for 30 minutes. Freeze-dried, it is stable on storage when kept below room temperature.

The compound is not incorporated into one of the Schedules of Chemicals contained in the Chemical Weapons Convention.

## **Blistering Agents**

Blistering agents, or vesicants, can produce skin injuries resembling those caused by burns. They also severely affect the eyes. By inhalation, the blistering agents affect the upper respiratory tract as well as the lungs, producing lung edema. In addition, the agents have a systemic effect when absorbed in the tissues. Protection from blistering agents is extremely difficult since they easily penetrate all kinds of materials. The most important substance in this class of CW agents is mustard gas. Some arsenicals including Lewisite also belong to the group of blistering agents.

## (i) Mustard Gas

ClCH<sub>2</sub>CH<sub>2</sub>SCH<sub>2</sub>CH<sub>2</sub>Cl

Chemical name: 2,2'-dichlorodiethyl sulfide.

Code: HD. HD is distilled mustard gas; the non-purified product containing approximately 30 of other sulfur compounds denoted as H or HS. HS originates from <u>H</u>un <u>S</u>tuff, the name given by allied troops. The name mustard originates from the mustard-like tickling sensation on the tongue. Mustard is tasted before one can smell it. In many languages the name yperite is used referring to the first place the agent was used, Ypres, Belgium. In Germany, it is called Lost after <u>Lommel and Steinkopf</u> who were rewarded for the development of a production line CAS registry number: 505-60-2.

Mustard gas, also named sulfur Mustard, yperite or, in German, senfgas, is called "the king of war gases." It was responsible for the majority of chemical warfare agent casualties during the First World War and is still considered one of the most hazardous CW agents. Skin injuries caused by mustard gas are long lasting and heal much slower than burns. Mustard gas is simple to produce and meets most requirements as a CW agent, apart from its delayed effect. It slowly dissolves in water and its droplets tend to float for long periods on water surfaces although it has a higher density than water.

Mustard gas is listed as a Schedule 1 chemical in the Chemical Weapons Convention.

| Physical state (20 °C): | liquid  |
|-------------------------|---|
| Odor:                   | garlic-like   |
| Melting point:          | 14 °C   |
| Boiling point:          | 228 °C (decomposes)   |
| Density:                | of the vapor, 5.4 (compared to air); of the liquid 1.268 (25 °C)  |
| Volatility:             | 610 mg.m <sup>-3</sup> (20 °C)  |
| Vapor pressure:         | 0.072 mmHg (20 °C)  |
| Rate of hydrolysis:     | Mustard gas slowly dissolves in water; the solubility is only approximately 0.8 g/l. When dissolved, mustard gas rapidly hydrolyses in distilled water; the half-life time is approximately 5 minutes (25 °C). In salt water the half-life time is 60 minutes (25 °C) |
| Stability on storage:   | stable  |
| Persistency:            | persistent agent; 1-2 days under average weather conditions, up to several weeks under cold conditions (see Table A.2.1)  |

#### (Ii) Nitrogen Mustard

N(CH<sub>2</sub>CH<sub>2</sub>Cl)<sub>3</sub>

Chemical name: tris (2-chloroethyl)amine. US code: HN-3. CAS registry number: 555-77-1.

Nitrogen mustard is one of three well-known nitrogen analogues of mustard gas. The other two agents are ethyl nitrogen mustard (HN-1,  $C_2H_5N(CH_2CH_2Cl)_2$ ) and methyl nitrogen mustard (HN-2,  $CH_3N(CH_2CH_2Cl)_3$ ). The compounds have toxicological effects similar to mustard gas. They are less stable in storage. Nitrogen mustard is the most stable agent in this respect. It is more resistant to water and oxidation than mustard gas, making decontamination more difficult.

Nitrogen mustards are listed as a Schedule 1 chemical in the Chemical Weapons Convention.

| Physical state (20 °C): | liquid   |
|-------------------------|--|
| Odor:                   | faint odor of amines   |
| Melting point:          | -4 °C  |
| Boiling point:          | 230-235 °C (calculated; decomposes before boiling point is reached)      |
| Density:                | of the vapor, 7.1 (compared to air); of the liquid, 1.24 (25 °C)         |
| Volatility:             | 121 mg.m <sup>-3</sup> (25 °C)   |
| Vapor pressure:         | 0.0109 mmHg (25 °C)  |
| Rate of hydrolysis:     | hydrolysis in water is slow. Furthermore, the solubility in water is low |
|                         | (0.16 g/l at 20 °C)  |
| Stability on storage:   | reasonably stable; it darkens and forms a crystalline deposit on storage |
| Persistency:            | persistent; considerably longer than mustard gas                         |

#### (iii) Lewisite

#### Cl<sub>2</sub>AsCH=CHCl

Chemical name: trans-2-chlorovinyldichloroarsine Code: L. The name and code refer to the developer Lewis CAS registry number: 541-25-3.

Lewisite is both a blistering agent and a toxic arsenic compound. The effects of Lewisite on the eyes and skin are immediately observable. Lewisite hydrolyses very rapidly when dissolved in water or as a vapor in humid air. The arsenic containing hydrolysis product is still toxic, but non-volatile. Lewisite is, however, of little importance as a CW agent due to its low stability in humid atmosphere.

Lewisite (also named L-1) and the analogues bis(2-chlorovinyl)chloroarsine (L-2) and tris(2-chlorovinyl)arsine (L-3) are listed as Schedule 1 Chemicals in the Chemical Weapons Convention.

| Physical state (20 °C): | liquid  |
|-------------------------|---|
| Odor:                   | geranium-like   |
| Melting point:          | -18 °C  |
| Boiling point:          | 190 °C  |
| Density:                | of the vapor, 7.1 (compared to air); of the liquid, $1.89 (20^{\circ})$   |
| Volatility:             | 4,480 mg.m <sup>-3</sup> (20 °C)  |
| Vapor pressure:         | 0.394 mmHg (20 °C)  |
| Rate of hydrolysis:     | rapid when dissolved in water or as a vapor in humid air. The solubility in water, however, is low, approximately $0.5 \text{ g/l}$ |
| Stability on storage:   | stable when dry; otherwise it decomposes into highly corrosive products   |
| Persistency:            | somewhat shorter than mustard gas; very short under humid conditions  |

#### (iv) Mustard Gas-Lewisite Mixture

Code: HL.

Addition of Lewisite to mustard gas lowers the melting point providing a low-freezing mixture for cold weather operations. The ratio Mustard to Lewisite in the mixture having the lowest melting point, the so-called eutectic mixture, is 37 to 63% by weight. The following properties are for the eutectic mixture. Mixtures with higher mustard gas content may also be prepared.

| Physical state (20 °C): | liquid   |
|-------------------------|--|
| Odor:                   | garlic-like  |
| Melting point:          | -25 °C   |
| Boiling point:          | less than 190 °C   |
| Density:                | of the vapor, 6.5 (compared to air); of the liquid, approximately 1.66 |
|                         | (20 °C)  |
| Volatility:             | 2730 mg.m <sup>-3</sup> (20 °C); 240 mg.m <sup>-3</sup> (- 11 °C)      |
| Vapor pressure:         | 0.248 mmHg (20 °C); 0.02 mmHg (- 10 °C)                                |
| Persistency:            | somewhat shorter than that of mustard gas                              |

## (v) Phosgene Oxime

Cl<sub>2</sub>C=NOH

Chemical name: dichloroformaldoxime. Code: CX. Code refers possibly to phosgene  $\underline{C}G$  and  $O\underline{x}$ ime CAS registry number: 1794-86-1.

Phosgene oxime is a solid with a high volatility. It is hygroscopic and tends to polymerize at normal temperatures. Phosgene oxime is known as a nettle gas. It is a powerful irritant which immediately produces pain sensation in the skin. It is difficult to produce and to store. The compound is not incorporated into one of the Schedules of Chemicals contained in the Chemical Weapons Convention.

Physical state (20 °C):solid; or liquid state for technical productOdor:disagreeable, penetratingMelting point:39-43 °CBoiling point:129 °C

A.4.3.2. Psychogenic Incapacitating Agents

Psychogenic incapacitating agents affect the mental state of the individual. Attention has been paid to the psychotomimetic agents in particular, also called hallucinogens or psychedelic agents. Glycolates have been extensively studied in the US as synthetic psychotomimetic agents. Most of these agents are natural products. Examples include LSD 25, mescaline, psilocybin and tetrahydrocannabinol (hashish).

These compounds are easy to manufacture at low cost. They are solids, but can be disseminated as an aerosol. One representative of this group, BZ, was weaponized. BZ, however, is not regarded as a completely satisfactory chemical warfare agent. As is the case for all psychochemical agents, its effects on groups of people under combat conditions are difficult to predict, something military commanders wish to avoid. In addition accidental exposures during weapon filling showed that the effects on troops could not be relied upon.

(i) **BZ** 

OH O \_\_\_\_\_\_ \_\_\_\_C\_\_\_O\_\_

Chemical name: 3-quinuclidinyl benzilate. Code: BZ (<u>Benzilate</u>) CAS registry number: 6581-06-2. BZ is listed as a Schedule 2 chemical in the Chemical Weapons Convention.

| Physical state (20 °C): | white solid  |
|-------------------------|--|
| Melting point:          | 168 °C   |
| Density:                | of the vapor, 11.6 (compared to air)                     |
| Volatility:             | negligible; approximately 0.5 mg.m <sup>-3</sup> (70 °C) |
| Vapor pressure:         | negligible; approximately 0.03 mmHg (70 °C)              |
| Rate of hydrolysis:     | 3-4 weeks at pH 7; 1.8 minutes at pH 13 (25 °C)          |
| Stability on storage:   | stable   |

#### A.4.4. Irritating Agents

The ratio between the lethal dose and the effective dose of irritating agents is high, as for incapacitating agents. Unlike incapacitating agents, however, irritating agents induce immediate effects upon exposure, which disappear relatively rapidly after cessation of exposure. Two groups of irritating agents will be dealt with: tear gases and vomiting agents. Tear gases are included in the police arsenals of many countries as riot control agents. The irritating agents are not incorporated into one of the Schedules of Chemicals contained in the Chemical Weapons Convention.

#### A.4.4.1. Tear gases

In addition to their use as riot control agents, tear gases are well known from their use in the Vietnam War. They are also used as training agents. Tear gases cause instant pain in the eyes, flow of tears, eyelid spasms, and irritation of the skin. Some may also have an effect on the respiratory system. When released indoors, they can cause serious illness or death.

#### (i) Chloroacetophenone

O ∥ CCH₂CI

Chemical name: ω-chloroacetophenone. Code: CN. CAS registry number: 532-27-4.

The solid agent is mainly disseminated as an aerosol. Although its volatility is relatively low, its efficacy is high enough for dissemination of an effective concentration. In addition to its lacrimatory effects, the agent is an irritant to the respiratory system.

| Physical state (20 °C): | solid   |
|-------------------------|---|
| Odor:                   | similar to apple blossoms                                       |
| Melting point:          | 54 °C   |
| Boiling point:          | 248 °C  |
| Density:                | of the vapor, 5.3 (compared to air)                             |
|                         | of the liquid, $1.187 \text{ g.ml}^{-1}$ (58 °C)                |
| Volatility:             | 2.36 mg.m <sup>-3</sup> (0 °C); 34.3 mg.m <sup>-3</sup> (20 °C) |
| Vapor pressure:         | 0.0026 mmHg (0 °C); 0.0041 mmHg (20 °C)                         |
| Rate of hydrolysis:     | very slowly   |
| Stability on storage:   | stable  |
| Persistency:            | short   |

#### (ii) Bromobenzyl Cyanide

CHCN Βr
Chemical name:  $\alpha$ -bromobenzyl cyanide. Code: CA. CAS registry number: 5798-79-8.

| Physical state (20 °C): | solid   |
|-------------------------|---|
| Odor:                   | like soured fruit   |
| Melting point:          | 25.4 °C   |
| Boiling point:          | 242 °C (decomposes)   |
| Density:                | of the vapor, 6.7 (compared to air); of the liquid, 1.47 g.ml <sup>-1</sup> (25 °C) |
| Volatility:             | 17 mg.m <sup>-3</sup> (0 °C); 115 mg.m <sup>-3</sup> (20 °C)                        |
| Vapor pressure:         | 0.011 mmHg (20 °C)  |
| Rate of hydrolysis:     | very slow   |
| Stability on storage:   | fairly stable in glass; vigorously corrosive on metals                              |
| Persistency:            | rather persistent; heavily splashed liquid persists 1-2 days under                  |
|                         | average weather conditions  |

#### (iii) ortho-Chlorobenzylidenemalononitrile

Chemical name: 2-chlorobenzylidenemalononitrile. US code: CS CAS registry number: 2698-41-1.

CS is a water-insoluble, white crystalline solid. It is disseminated either as a spray of solution, or as a cloud of dust or powder, or as an aerosol. The formulation known as CS1 comprises micronized CS powder mixed with 5 per cent silica aerogel to reduce agglomeration. It remains active for up to 5 days when dusted on the ground. The formulation CS2 is CS containing the silicone water-repellent Cab-O-sil, which reduces both agglomeration and hydrolysis. CS2 may be persistent for as long as 45 days.

| Physical state (20 °C): | solid   |
|-------------------------|---|
| Odor:                   | pepper-like   |
| Melting point:          | 93-95 °C  |
| Boiling point:          | 310-315 °C (decomposes)   |
| Volatility:             | $0.71 \text{ mg.m}^{-3} (25 \text{ °C})$                            |
| Rate of hydrolysis:     | rapid when dissolved in water. The solubility in water, however, is |
|                         | low (approximately 0.8 g/l)   |
| Stability on storage:   | stable  |
| Persistency:            | depending on degree of contamination and on the formulation used    |

A.4.4.2. Vomiting agents

This group of irritating agents causes coughing and vomiting in addition to irritation of mucous membranes. The agents are also called sternutators.

# (i) **Diphenylchloroarsine**

. AsCl

Chemical name: <u>diphenylchloroa</u>rsine. Code: DA. CAS registry number: 712-48-1.

Diphenylchloroarsine was used in the First World War and is also known as Clark I.

| Physical state (20 °C): | solid   |
|-------------------------|---|
| Odor:                   | none  |
| Melting point:          | 40 °C   |
| Boiling point:          | 333 °C (decomposes)   |
| Density:                | of the liquid, $1.387 \text{ g.ml}^{-1}$ (50 °C)                          |
| Volatility:             | 0.68 mg.m <sup>-3</sup> (20 °C)   |
| Vapor pressure:         | 0.0005 mmHg (20 °C)   |
| Rate of hydrolysis:     | low; the first hydrolysis product is a relatively potent irritating agent |
| Stability on storage:   | stable  |
| Persistency:            | short   |

# (ii) Diphenylcyanoarsine

AsCN

Chemical name: <u>diphenylcyanoarsine</u>. Code: DC. CAS registry number: 23525-22-6.

The agent was used in the First World War and is known as Clark II.

| Physical state (20 °C): | solid   |
|-------------------------|---|
| Odor:                   | similar to a mixture of garlic and bitter almonds |
| Melting point:          | 31.5-35 °C  |
| Boiling point:          | 350 °C (decomposes)                               |
| Density:                | of the liquid, $1.334 \text{ g.ml}^{-1}$ (35 °C)  |
| Volatility:             | $1.5 \text{ mg.m}^{-3} (20 \text{ °C})$           |
| Vapor pressure:         | 0.0002 mmHg (20 °C)                               |
| Rate of hydrolysis:     | very slow   |
| Stability on storage:   | stable  |
| Persistency:            | short   |

#### (iii) Adamsite



Chemical name: 10-chloro-5,10-dihydrophenarsazine. Code: DM. CAS registry number: 578-94-9.

Adamsite was stockpiled during the Second World War. Its volatility is very low. Hence, intoxication by its vapor is not possible. It can effectively be disseminated as an aerosol.

| rm |
|----|
|    |
|    |
|    |

# A.4.5. Herbicides

Herbicides or anti-plant agents may be used in a military conflict for destruction of crops or for defoliation of natural vegetation to reduce the enemy's possibilities of finding cover. The potential agents can be divided according to three distinct types of action on the plant: defoliation, poisoning to death and prevention of growth. Defoliation is induced by so-called defoliants which interfere with the mechanisms that normally bring about seasonal leaf falls. Defoliation can also be secured by desiccants which dry out the leaves thus becoming easily detached by wind or rain. Toxic actions are caused by plant growth regulators which promote, inhibit of otherwise modify physiological processes in plants. Employment of soil sterilants prevents the growth or re-growth of plants.

Dispersion of herbicides from the air is the only feasible method of covering areas of any considerable size rapidly.

Herbicides were used on a large scale in the Vietnam War. The agents used were given their names from the colored tapes which secured the packing cases, such as Agent Orange, Agent White, Agent Purple and Agent Blue.

The herbicides are not incorporated into one of the Schedules of Chemicals contained in the Chemical Weapons Convention.

Chemical name: 2,4-dichlorophenoxyacetic acid. CAS registry number: 94-75-7.

2,4-D is a white crystalline compound with a melting point of 139-140 °C. It is slightly soluble in water and miscible with oil. It is non-corrosive to metals.

2,4-D is generally used as a salt in dilute oil/water emulsions. Esters of 2,4-D, which are liquids of low volatility, have been employed in various mixtures (see Agent Orange and Agent Purple). Esters are more effective than the salts in penetrating the leaf cuticle and in killing species. Immediately after application, rain slightly reduces the effectiveness of sprays of the salts in aqueous solution, but rain does not influence the effectiveness of ester sprays.

Plant injuries will generally be apparent within 24 hours after application. In some plants the stems and leaves dry until the plants are dead. In other plants the stems remain green for several weeks but swell, develop cracks and form callous tissue. Numerous watery, translucent buds often appear at the crown but do not grow into new shoots. Seriously affected plants may develop spongy, enlarged roots several weeks after application.

(ii) 2,4,5-T

Chemical name: 2,4,5-trichlorophenoxyacetic acid. CAS registry number: 93-76-5.

2,4,5-T is a white crystalline compound with a melting point of 153 °C. It is slightly soluble in water and miscible with oil. It is non-corrosive to metals.

Technical preparations of 2,4,5-T may contain small quantities of the highly toxic 2,3,6,7-tetrachlorodibenzo-p-dioxin, also known as dioxin. Dioxin is very toxic and in addition has carcinogenic and teratogenic properties.

Like 2,4-D, 2,4,5-T can be used as a salt or as an ester. The effects of 2,4,5-T on plants are similar to those of 2,4-D. On certain plants, 2,4,5-T is more effective.

#### (iii) Agent Orange



Agent Orange is a mixture of 50% n-butyl ester of 2, 4-D and 50% n-butyl ester of 2, 4, 5-T, with a melting point of 7 °C. It consists of 95-100% active components. The agent is miscible with alcohol, acetone, ether and oil. The agent softens rubber and paint. It may deteriorate rubber equipment and remove coatings of paint. The agent will mainly be used for defoliation, but also for destruction of crops.

#### (iv) Agent Purple



Agent Purple is a mixture of 50% n-butyl ester of 2, 4-D, 30% n-butyl ester of 2, 4, 5-T and 20% isobutyl ester of 2, 4, 5-T. Properties of the agent are similar to those of Agent Orange.

#### (v) Cacodylic Acid (Agent Blue)

Cacodylic acid

(CH<sub>3</sub>)<sub>2</sub>ÄsOH

Chemical name: dimethylarsenic acid. CAS registry number: 75-60-5.

As a pentavalent arsenic compound, it has a low toxicity to warm-blooded species including humans. A toxic trivalent arsenic compound will be formed by reduction, which may take place in tropical areas when applied to rice fields.

Cacodylic acid has been applied in a formulation called Agent Blue. This is a 40% solution of the sodium salt of Cacodylic acid in water containing a proper amount of surfactants and a corrosion inhibitor.

Agent Blue causes foliage to dry and shrivel by absorption of moisture from the leaves. Although the agent can produce relatively rapid defoliation, re-growth may occur in one month. Agent Blue prevents grain formation in rice at low doses without apparent external effect. Higher spray rates usually kill the crop.

## (vi) Agent White



The active components of Agent White are the triisopropanolammonium salt of 2, 4-D (80%) and picloram (20%). Picloram (4-amino-3, 5, 6-trichloro-2-pyridinecarboxylic acid; CAS registry number: 1918-02-1) is soluble in water and not miscible with oils. It is very persistent in soil. Although it is washed away from the upper layer of the soil by rain fall, picloram can be taken up in plants as soon as their roots reach deeper into the soil (60-120 cm). Picloram is more toxic to plants than 2, 4-D, but its effects appear more slowly.

Agent White is a white mixture, soluble in water and not miscible with oil. It is applied as a solution in water. The maximum concentration of the active components is 25%. Initially, its toxic effects on plants are due to the anti-plant action of 2, 4-D. The long duration effects are caused by the persistent picloram.

#### A.4.6. Biological Agents (Replication)

The US Centers for Disease Control and Prevention publishes a list of restricted biological agents. The restrictions hold for those agents with a potential application for biological warfare or terrorist attacks. The list contains a number of toxins; some of them have been mentioned previously. Many more lists of potential biological warfare agents and toxins circulate in the literature. A selection of those agents will be dealt with in the framework of a course for the OPCW assistance and protection program. The biological agents are solids or suspensions in a liquid. Viruses need a host to survive in the environment. They must be dispersed as aerosol in order to infect the lungs or must be distributed in food or drinking water. In the later case, only a limited number of microorganisms can survive in water. Many of the other biological agents lose their infectivity rapidly when exposed to the environment, in particular due to UV radiation. In order to stay airborne, the particles must be small, approximately a few microns. Those particles are difficult to make and it is not possible to spray them from aircraft such as the case for chemical agents. The particles would be either too large and settle rapidly to the ground, or be too small and stay up high in the air and only reach ground level in a much diluted concentration. In addition, for many biological agents the infectivity goes down when the particle size increases. These kinds of considerations make it very difficult to state exactly what the infectivity or toxicity of biological agents is. The example of ricin will make this partly clear. Ricin as a pure solid has a toxicity comparable to the nerve agent Sarin. However, the dispersion efficiency of a solid in a particle size that stays airborne and forms a respiratory hazard is about two order of magnitude less than that of a liquid that turns quickly into a vapor, such as Sarin. In the Tokyo Metro incident about 100 kg ricin had to be dispersed to arrive at similar casualty numbers than that for the case of about 1 kg Sarin release. There is no doubt such dispersal would have been noted and at least partly prevented.

Biological agents have many different forms.

- The smallest type of microorganisms is viruses. They consist of RNA surrounded by an outer layer of protein; sometimes an additional outer layer of lipids is present. Single viruses are very small, 0.02-0.2 micron. A virus is strongly dependent on its typical host cells in which it can multiply.
- Bacteria are single cell microorganisms. They contain all the DNA and information to multiply provided the environment is correct. The size is approximately 1 micron. Some bacteria can be transformed into spores that are more resistant to environmental factors. The spores can germinate again provided the environment is correct. Bacteria can act upon humans in two ways. In one mechanism the bacteria penetrates into the tissue and attack it. In the other mechanism the bacteria starts to produce a toxin. Some bacteria can employ both mechanisms.
- Rickettsiae are a special type of bacteria because they need the help of other cells to multiply. They first have to penetrate into the cell of the host and multiply in the cell as a parasite.
- Fungi are the largest type of microorganisms. They are more likely to attack plants than humans, but many produce toxic compounds that are highly resistant to environmental factors. Grain infected by Fusarium can produce trichotecenes which can withstand the temperatures involved in baking bread.

One of the problems in the use of biological agents for warfare purposes is the often uncertain and sometimes long incubation period. One could safely say that for military tactical purposes any agent with an incubation period of more than 5 days is not attractive. Additionally, the range of possible incubation times increases when the incubation time itself increases. So agents with an incubation period of 1-2 weeks lack the predictability required for military purposes. It is obvious that arguments which are of concern to military operations do not hold for terrorist activities. If a terrorist group decides to use biological warfare agents, they can use what ever they think will do the job.

# Viruses

# Crimean-Congo hemorrhagic fever

The virus is endemic in Africa south of the equator, Central Asia and the Middle East. Transmission is through ticks and by contact with blood and body fluids from infected patients. Animals can be infected as well and slaughter of these animals may generate aerosols. The incubation period is 1-2 weeks. Mortality is approximately 20% in an illness period of about ten days.

## **Dengue fever virus**

Dengue virus is present in more than 100 countries with tropical regions and mainly in urban areas. Mosquitoes carry the virus, and the spread of the insect is responsible for the geographic expansion of the virus. There are four different strains, which are so different that immunization for one type does not protect against another strain. The incubation period can vary from 0.5 to 2 weeks. When infection progresses to hemorrhagic fever, it is potentially lethal. There is no specific therapy and no vaccine available.

#### Eastern equine encephalitis virus

Eastern equine encephalitis virus is one of the many forms of encephalitis type of viruses. The virus is transmitted by insects from animal carriers to humans. The incubation period is usually 1 to 2 weeks and the disease last for 2 to 5 weeks. Mortality is low and transfer from human to human is not observed.

## Equine morbillivirus or Hendra virus

A virus circulating in wildlife reservoirs (fruit bats) in Australia and can jump from bats to horses and humans. It is often fatal. The disease had been described as being due to a morbillivirus in 1994 but that has proven to be incorrect.

#### Ebola viruses

A Filovirus discovered in Africa (Congo). It is one of the most pathogenic viruses known to man and has a mortality rate of 50-90%. The 50% refers to an outbreak in Sudan. The strain present in the Congo is more deadly. Transmission is through contact with blood, bodily fluids or tissue from infected patients. The incubation period is 2-20 days.

#### Lassa fever virus

Lassa fever is caused by a virus mainly occurring in Africa. It was discovered in 1969 in Nigeria. The infection is transferred from rodents to humans. The virus is very infectious.

#### Marburg virus

Marburg virus is a Filovirus closely related to Ebola. The virus was discovered among monkey caretakers in Marburg, Germany, when handling a new shipment of monkeys. The patients develop high fever and the internal organs are attacked, in particular liver and lymphatic glands. The incubation period is just under one week. The disease is highly infectious and mortality of the known cases is around 25%.

#### Rift Valley fever virus

Rift Valley fever virus is a natural occurring disease in parts of Eastern Africa. Infection can take place via the air (rare) but mostly through mosquito bites. The incubation period is 4-6 days. When the illness develops, the patient has fever, headache and muscle pain. Mortality is low. A vaccine is available. The virus also attacks cattle, sheep and goats.

#### South American hemorrhagic fever viruses

(Junin, Machupo, Sabia, Flexal, Guanarito). There are several forms of this virus; the best known is Junin causing Argentine hemorrhage fever. The virus is present in rodents and spread though their excreta. When dried, this might be transferred to aerosols. The incubation period is 1-2 weeks. The mortality is just under 20%.

#### Tick-borne encephalitis complex viruses

Ticks present in Russian forests carry the disease and transmit it to humans. The symptoms are much the same as for the other encephalitis viruses.

#### Variola major virus (smallpox virus)

The virus causes smallpox and is highly contagious. After an incubation period of 1-2 weeks the patient develops high fever and skin eruptions. These eruptions when dried out leave the well-known pockmark scarring behind. This is one of the few viruses that are relatively stable in the environment. Mortality can be up to 30%. The eradication of smallpox through a world wide vaccination program is one of the success stories of the WHO.

#### Venezuelan equine encephalitis virus

A virus present in birds in Central and South America, it can be transferred to humans by infected insects. After a widely varying incubation period of 4-20 days, it results in fever and headaches and many other symptoms. The disease is not contagious but it seems that the virus is very infectious.

#### Yellow fever virus

Yellow fever occurs mostly in areas where there are tropical rain forests, but during the rainy seasons, it can also spread to regions with a more temperate climate. The disease is spread through mosquito bites. The incubation period is 3-6 days, and the virus grows in the lymphatic system. It attacks internal organs, especially the liver, and gives rise to skin eruptions. Bleeding of the skin and mucous membrane is common. The mortality rate is 20-30%. A vaccine is available.

# (ii) Bacteria

#### Bacillus anthracis

Anthrax is caused by the spores of the bacterium *Bacillus anthracis*. Spores are present in animal products, and people who tend the animals or consume these products might get infected. The spores are highly resistant. When cattle graze on lands where tens of years before infected animals were buried, they are likely to become infected. Forty years are required before an anthrax contaminated island of the UK can be returned to the owners anthrax free. The island was contaminated in order to investigate the potential of anthrax as a biological warfare agent in the early stages of WW I. There are two known cases of deliberate use of anthrax. One was during WW I when some German travelers tried to infect horses used by the allied troops. The other case refers to the anthrax letter incidents in the US in October

2001. Anthrax occurs naturally in many countries, especially amongst wild animals in southern Africa. Consumption of contaminated meat was at first given as the cause of the anthrax victims in Sverdlovsk in 1979. Later it appeared that the casualties resulted from an accidental release from a military research institute involved in biological warfare. The bacterium attacks cattle, horse, sheep and pig.

## Brucella abortus, Brucella melitensis, Brucella Suis

Brucella species are found in cattle and transferred to humans from infected milk or from sick animals. The patients slowly develop a fever, suffer headaches, and become exhausted and confused. The illness can be treated with antibiotics. The bacterium attacks cattle, sheep and pigs.

# Burkholderia (Pseudomonas) mallei

Also known as glanders. *Burkholderia* infects animals such as horses, dogs, goats, etc. Humans contract the infection from infected animals through broken skin or the respiratory system. Aerosols are highly infectious and penetrate the human system via the lungs. This form has an incubation period of 10-15 days. Cutaneous glanders incubates after 3-5 days. When the blood stream is invaded, septicemic glanders develops which is almost always fatal.

## Burkholderia (Pseudomonas) pseudomallei

*Burkholderia pseudomalleus is* a bacterium present in the soil and waters of Southeast Asia and tropical areas. It causes the disease Melioidosis. Depending on the site of the human body that is infected, several symptoms might become apparent. This bacterium is closely related to *Burkholderia*, and the effects are closely related as well. Incubation period is again 3-5 days, and blood stream invasion is almost always fatal.

#### Clostridium Botulinum

*Clostridium Botulinum* produces the nerve toxin Botulinum toxin. It has a paralyzing effect, it occurs naturally in water, and at least 6 different types of toxins are produced by different strains, e.g. Botulinum toxin A, B, etc.

#### Francisella tularensis

*Francisella tularensis* causes the disease known as rabbit fever or tularemia. The bacterium not only attacks rabbits but may infect other small animals. It can be transferred to humans through the bites of mosquitoes, from inhaling contaminated hay dust or through contact with infected rodents. The onset time to effects is highly variable from 1-10 days, but for inhalation 2-3 days. Mortality strongly depends on the type of strain. European forms of tularemia have a mortality rate usually below 10% of the infected population. The strain naturally occurring in the US has about a 5 times higher mortality rate.

#### Yersinia pestis

Better known as plague, *Yersinia pestis* was the cause of many epidemics throughout history. One example is the Black Death epidemic in Europe in the 14th century, killing 50% of the population in three years. The disease is transferred by fleas that live on infected rodents.

When transferred to humans, they suffer from the bubonic plague. The incubation time is 2-6 days and patients will show a high temperature and swollen lymphatic glands. Transmission via the air between humans is possible, and infection of the lungs causes pulmonary plague. Again a high temperature will develop after 2-4 days. The patient feels exhausted and has difficulties breathing. Without treatment, the mortality approaches 100%. The bacterium produces a toxin, called plague toxin that is responsible for killing tissue.

# (iii) Rickettsiae

# Coxiella burnetii

Q-fever, as the disease is called, results from the inhalation of spores. The corresponding bacterium is a parasite that can be grown in hen eggs. Sometimes goats are infected and transfer the infection to humans. The incubation time is 2 weeks, which makes it not attractive for tactical warfare. The infectious dosage is very low, on the order of 10 spores. The infected persons develop headaches, muscle pain and a dry throat. Patients are usually ill for a couple of weeks. The bacterium also attacks cattle and sheep.

## Rickettsiae Prowazekii

This is a disease-causing bacterium which lives in lice, which can jump from one person to the other. The disease is better known as spotted typhus. After a highly varying incubation period of 5-15 days, patients develop a high fever, coughing and bleeding from the nose. Later the skin will show eruptions and the patients get confused. Mortality is between 10-40%. For treatment of spotted typhus, broadband antibiotics are required.

#### Rickettsiae rickettsi

This is a disease-causing bacterium that lives in ticks. The disease is called Rocky Mountain spotted fever. The incubation period varies from 3-10 days. Besides fever, headache, muscular pains, and photophobia develop, and after some days a skin rash spreads from the ankles and wrist to legs arms and chest. The disease last for about two weeks is not contagious. Mortality rate is about 25%. Vaccines are not available, and broadband antibiotics therapy reduces the mortality rate.

#### (iv) Toxins

The description of the properties of toxins will be restricted to those high on the list of potential biological warfare agents and the two mentioned in the schedules of the CWC. It is felt that it is of little use to provide detailed information on all the toxins on the CDC list because many of the agents mentioned on that list are not seen as a terrorist threat or warfare agent threat. As an example, Aflatoxin causes a cancer to develop in the liver after about ten years. Diacetoscirpenol is one of the agents that appeared on the list after the yellow rain incidents. A more likely explanation for those incidents is swarms of bees dropping their feces in a rain storm.

#### Clostridium Botulinum

This organism produces the Botulinum toxin (See A.4.2. (I)) of which at least six types are known over which the toxicity varies. The toxin paralyses the diaphragm and chest muscles

with death resulting from asphyxia. The toxin is unstable in air, and when inhaled, higher dosages are required than if the toxin was ingested. The disease is not contagious, and the mortality is 60%. Botulinum toxin is usually regarded as the most toxic compound known to man. Compared on a weight basis, it is about 1000 times more toxic than nerve agents. However, the efficiency of dispersal for nerve agents is one to two orders in magnitude higher, making Botulinum toxin 10-100 times more "hazardous" than nerve agents. An antitoxin is available and should be given in the early stages of the development of the intoxication. An important part of the treatment is to keep the patients breathing.

## Diphtheria toxin

It is produced by *Corynebacterium diptherae*. The bacterium does not invade the cells but adheres to the mucous membrane of the heart, kidneys and liver and produces the toxin that invades the cells. The disease has an incubation period of 2-7 days and may last for up to 70 days. The mortality is high and the disease is contagious. Vaccine is available. Therapy is either through an antitoxin given in the early stages or through antibiotics treatment (penicillin).

## **Ricin** (see A.4.2. (iii))

Ricin is a toxin naturally present in castor beans. When the beans are processed for the oil, the toxin remains in the residue. Castor beans are produced in large quantities, and the toxin is relatively easily to extract from the mash left over from the processing. The toxicity is of the same order as those of nerve agents but the dispersal efficiency is again 1 to 2 orders of magnitude smaller. The symptoms depend on whether the agent was ingested; inhaled or injected (it has been used for political assassinations). When inhaled, the effects mainly appear in the lungs and failure of the respiratory and circulatory systems causes death. When ingested, bleeding of the stomach and intestines, failure of the liver and kidneys and death by circulatory failure will result. Death from injected ricin will follow due to circulation of the toxin through the body and attack on all major organs. Except for supportive care, there is no therapeutic treatment available and no vaccine

Saxitoxin (see A.4.2.(ii)

Saxitoxins are a family of neurotoxins. They operate by blocking sodium channels in cells which transmit signals to muscles. They are produced by lower organisms in the oceans. Human intoxications are mostly due to eating mollusks that have been feeding on the infected organisms. The resulting disease is called paralytic shellfish poisoning. Onset of symptoms is usually within 1 hour. Nerves responsible for eye movement and those in the throat are often attacked. In general there is a lack of muscular coordination. In severe poisonings, death results from respiratory paralysis. Supportive care such as assisted breathing might reduce fatalities. Fortunately the disease appears relatively quickly after eating infected mollusks. Where delayed onset occurs, it has sometimes been diagnosed as a nerve agent poisoning. The atropine treatment that would follow makes things worse and will increase lethality.

#### Shigatoxin

The *Shigella* bacterium produced Shigatoxin, and only few organisms are sufficient to cause dysentery. The incubation period is 2-3 days, and the resulting disease manifests stomach pains and bloody diarrhea. The disease is contagious and there is no vaccine. The treatment is

to drink large quantities of fluid. Shigatoxin is a neurotoxin and can cause paralysis in addition to the bleeding. The toxicity is in the same range as that of Botulinum toxin.

# Staphylococcus enterotoxin B (SEB) (see A.4.3.1. (i))

SEB is produced by *Staphylococcus aureus* and is responsible for symptoms of food poisoning that follow consumption of food contaminated by *Staphylococcus* bacteria. The toxin has a specific action on intestinal mucosal cells and is therefore called an enterotoxin. It is a white protein that attracts moisture from humid air and readily dissolves in water. Certain strains of *Staphylococcus aureus* generate the toxin in large and easily isolable yields. It is resistant to freezing and boiling for 30 minutes. Freeze-dried, it is stable on storage when kept below room temperature.

The compound is not incorporated into one of the Schedules of Chemicals contained in the Chemical Weapons Convention. Another type of toxin produced by Staphylococcus bacteria is the alpha toxin leading to similar effects.

## Tetrodotoxin

A toxin present in the liver and ovary of the puffer fish (*Takifugu*). It acts very rapidly, usually within an hour. Poisoning is by ingestion or injection.