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Review Article

Chemical and Biological Warfare preparing to meet the Threat

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Introduction

Till the recent past, there was a widespread tendency to think about defence against chemical and biological warfare (CBW) agents as unnecessary, as someone else's responsibility, or as simply too difficult.[1] The threat of exposure to such agents has traditionally been considered a military issue. Several recent events, however, have demonstrated that civilians may also be exposed to these agents;[2-4] and in the wake of recent atrocities there has been renewed apprehension regarding the deployment of chemical and biological weapons.[5]

Potential sources of exposure for civilian population include acts of terrorism, inadvertent releases from domestic chemical weapon stockpiles, direct military attacks, and industrial accidents. The hostile use of CBW agents would be likely to cause significant impact on health care systems. Patient might present in unprecedented numbers, and demands for intensive care might overwhelm medical resources. Special medications or vaccines, not generally available in standard pharmaceutical stocks, might be required. Health care professionals and laboratory personnel might need added physical protection. All these problems can be overwhelming for the health care system, but the efficacy of the medical response could be improved if health care professionals were better aware of symptoms, pathophysiology and treatment of agents likely to be used.[5] There has been an increasing feeling amongst hospital clinicians that there should be more awareness and preparation about the management of casualties after a terrorist attack using CBW agents.[5-7]

We present an overview of the risk that chemical and biological warfare agents presently pose to the civilian population; and a discussion of the presentation and principles of management of exposure to such agents.

Historical Background

The use of chemical and biological warfare agents as weapons has been attempted throughout the history. Biological warfare has evolved from the crude use of cadavers to contaminate water supplies, to the development of specialized munitions for battlefield and covert use. Recognition of the potential impact of infectious disease on armies resulted in the crude use of filth, cadavers, animal carcasses and contagion as weapons. These have been used to contaminate water supplies since antiquity, through the Napoleonic era, and into the 21st century. Allegations have been made since World War I, but these have not been confirmed because of the absence of microbiological or epidemiological data.[8] These incidents underscore the difficulty in differentiating biological attacks from naturally occurring epidemics and endemic diseases, and emphasize the increased risk of epidemics during hostilities because of deteriorating hygiene, sanitation and public health infrastructure.[9] The practice of ascribing naturally occurring epidemic or endemic diseases to alleged biological attacks for propaganda purposes, demonstrates the perception of psychological vulnerability to the threat of biological warfare. Despite all the limitations, concern continues to grow regarding the possibility of proliferation or enhancement of state sponsored offensive biological weapons programs and the possible use of biological weapons by terrorist organizations.

Potential Sources of Exposure to Civilian Population

1. Terrorist attacks

Terrorism has been defined as the use or threat of violence to sow panic in a society, to weaken or overthrow its leaders, or to bring about political change.[10] Terrorists have previously used conventional means of violence, but several recent events have demonstrated that some terrorists now have access to weapons of greater lethality, including chemical and biological agents.[10-12] Recent technical advances, easy access to raw materials, the ready availability of technical information, and the possible support of terrorists by certain hostile foreign governments have all contributed to the proliferation of CBW agents.[13,14] The most publicized use of chemical agents by terrorists against a civilian population was sarin vapor release in the Tokyo subway, which resulted in 12 deaths and more than 5500 casualties.[4,15] Threats against civilians by terrorists with CBW agents have also been made in California, Chile, and Germany.[16]

2. Military usage / Stockpiles

Uncommonly, civilian populations may become the direct targets of military attacks with CBW agents. They may also potentially sustain unintentional collateral injuries when their own nation's military uses chemical and biological weapons against enemy forces. Few cases of intentional use of CBW agents by military units against civilians have been documented, including the Iraqi attack on Kurdish population, and the recent use of nerve gas in Russia.[17,18] The accidental release of CBW agents from military stockpiles remains another potential threat to the population.

3. Industrial incidents

A number of agents that have been used as chemical weapons, are routinely used in a variety of industrial processes. Inadvertent release of these chemicals, or terrorist attacks aimed at storage and transportation facilities, have the potential to threaten surrounding communities and may result in acutely life-threatening emergencies.[19]

Factors affecting the Severity of CBW Attacks

1. Type of release

The severity of attacks by CBW agents depends in part on the dispersal method used.[5] CBW release into water supplies or food chains produce fewer casualties than airborne release (aerosolized or powdered preparation), as this is not easily detectable, and secondarily causes food and water supply contamination. Several factors influence casualty rates after an air borne attack. Point deployment describes release from a single source. Line deployment is release from a moving vehicle; a wide cone of dispersal occurs if line deployment occurs perpendicular to prevailing wind.[5] Explosive deployment partially destroys the agent as high temperatures resulting from initial explosion degrade chemical agents.

2. Volatility

Volatility is the tendency of a liquid to evaporate and form vapors. At usual atmospheric temperatures and pressures, most CBW agents are in liquid form.[20,21] After the detonation of ammunition containing a CBW agent; the agent is dispersed primarily as a suspension of fine liquid droplets. The vapors of all CBW agents in general are heavier than air. Therefore, the exposed individuals are safest if they are able to ascend to a higher point, such as the top floor of a building.

3. Persistence

Persistence is inversely related to volatility. The more volatile an agent, the quicker it evaporates and disperses and vice a versa. Military CBW agents are intended to be persistent, or semi-persistent. This is clinically relevant, as persistent agents are slower to evaporate and will remain in contact with body surfaces for longer periods causing harmful effects. Such agents also pose greatest threat to rescue and medical personnel, as there is a risk of secondary exposure and contamination from patients and the surrounding environment.[22,23]

4. Toxicity

Toxicity is defined as the potential for an agent to cause injury to biological systems.[24] Two important concepts related to the toxicity of CBW agents are lethality and incapacitating effects. The cyanides and nerve agents are the most lethal of the CBW agents, and can cause death within minutes.[20] The incapacitating effects of CBW agents can be even more important than their lethality.[22] The military utility of these agents may result in the diversion of military resources to casualty evacuation and the provision of medical care.

5. Latency

Latency refers to the time delay between the exposure or absorption of an agent and the onset of clinical manifestations. This is as much important for chemical weapons as that for biological weapons. The individuals who have been exposed to any agent with significant clinical latency may require medical monitoring and quarantine for many days.[22] This need for monitoring a large number of exposed individuals can potentially overwhelm the resources of medical facilities.

Agents used as Chemical and Biological Warfare Agents

The rapid identification of an unknown agent may assist in determining early medical and public health

interventions.[19]

A. Chemical Agents

I. Nerve Agents (Sarin, Tabun, Soman, VX)

They are extremely toxic, odorless, tasteless and colorless. They are structurally related to organophosphorus compounds (insecticides), and are irreversible inhibitors of cholinesterase enzymes.[25] Their administration results in cholinergic crisis followed by respiratory failure and polyneuropathy. A triphasic clinical syndrome develops after exposure.[5]

1. Cholinergic Phase

Acetylcholine (Ach) accumulation may result in death from bronchoconstriction, vocal cord paralysis, bradycardia and convulsions.[5] This phase lasts for 24-48 hours and requires intensive care. Management includes Atropine, Oximes and Magnesium.

2. Intermediate Syndrome

It begins after the cholinergic phase and lasts for 4-18 days[26] depending on the de-novo synthesis rate of Acetylcholinesterase (AchE). Usually 1% of pre exposure AchE function is recovered per day. This phase is characterized by muscle weakness particularly of diaphragm associated with respiratory failure and cranial nerve palsies.

3. Delayed Polyneuropathy

This phase occurs 7-14 days after exposure.[5] It is characterized by symmetrical peripheral muscle weakness in addition to sensory disturbances. The cause of polyneuropathy is thought to be inactivation of another enzyme (neuropathy target esterase). Persistent postural imbalance, shoulder stiffness and blurring of vision have been reported years after exposure.[5]

Treatment

Pyridostigmine Bromide may be used as a pretreatment by emergency personnel for whom exposure to nerve agents is expected. It is not a true pre treatment but an antidote enhancer.[5] It is a reversible competitive carbamate ester antagonist of AchE. The rationale of treatment is that pyridostigmine carbamylation of AchE binding sites produces a reservoir of temporarily inactivated AchE. After exposure, nerve agents are unable to bind to the carbamylated enzyme.

Atropine and oximes are effective antidotes if administered early after exposure.[27] Atropine antagonizes the muscarinic side effects if administered early after exposure. Atropine is given in repeated dosing till pupillary dilatation occurs[28] and heart rate is more than 80/min. Atropine infusion is advocated for resistant bradycardia.[5]

Oximes reverse nicotinic receptor dysfunction.[5] It reduces or reverses paralysis. Oximes detoxify unbounded agent molecules. Pralidoxime can be used in children and adults. Therapeutic range is 4ug/ml. The possibility of re-inhibition of reactivated AchE should be borne in mind. It maybe amenable to treatment using edrophonium.[5]

Other drugs may also be used including benzodiazepines for convulsions[5,29] and clonidine to control cholinergic symptoms.[30] Magnesium can be used to reduce presynaptic Ach release. Adrenal medulla stimulation in acute toxicity may produce positive inochronotropism necessitating alpha and beta adrenoceptor blockade.[5]

II. Blistering Agents

Vesicants can be classified into 2 main groups: arsenicals and mustards.

Mustard gas and Lewisite are liquids which cause chemical burns and blistering to all epithelial tissues. After inhalation or ingestion, systemic manifestations include respiratory failure, blindness, vomiting, pancytopenia and cancer.

Mustard gas: [bis (2chloroethyl) sulphide] is a colorless or pale yellow oily liquid that smells faintly of garlic or mustard.[31] Atmospheric release occurs through explosive aerosolization. Its persistence places medical responders at greater risk of intoxication. Wearing of protective clothes and decontamination of casualties and responders is essential.

Mustard gas forms highly reactive sulphonium ions in the body, which alkylate DNA and enzymes. There is a period of latency between exposure and the development of symptoms.[5] Suspected exposure necessitates careful clinical observation and review of the patient. Cutaneous manifestations after 4-12 hours include erythema, edema, and first degree burns; vesication occurs with greater exposure. Necrosis and spreading vesication is seen within minutes of the exposure. Intravenous fluids and burns protocol is required.

Ocular symptoms occur in 85% of patients. Corneal edema is followed by vesication and corneal sloughing. Vision recovers by corneal revascularisation over a period of weeks. Exposure to high doses may lead to permanent blindness.[32]

Respiratory problems occur in over 70% of victims[5] and include dry cough, hoarseness, bronchospasm and airway collapse distal to areas of sloughed respiratory epithelium. Lung damage may be permanent and can induce COPD. Acute upper airway obstruction may require cricothyroidotomy, though this may be technically difficult. Bone marrow suppression may occur.

III. Choking Agents

They are the classical agents of chemical warfare. Chlorine and phosgene were first used in 1915.[5] Chlorine, phosgene and chloropicrin are highly volatile liquids. After inhalation of their vapor, early respiratory distress occurs followed by a variable latent period, following which toxic pulmonary edema and permanent lung damage may occur in survivors of acute phase.

Chlorine is a greenish yellow gas with a distinctive smell which provides adequate warning. It is an oxidizing agent and reaction with water liberates hypochlorous acid, hydrochloric acid and O_2 free radicals; all of which cause tissue damage. Initial exposure causes eye pain, blepharospasm and lacrimation.

Phosgene being 4 times denser than air remains close to the ground. Gas dissolves slowly in water to form CO_2 and hydrochloric acid. Slow dissolution allows phosgene to enter respiratory tissue without significant upper airway damage. Necrosis follows inflammation of delicate terminal airways and alveoli. Alveolar capillaries leak large volumes of serum causing pulmonary edema and respiratory failure. Hypoxemia and ischemia leads to multiorgan dysfunction. Morbidity and mortality are related to the degree of pulmonary damage.

Management of choking agents is supportive. Inhaled or intravenous steroids in high doses are recommended. Antibiotics and leukotriene inhibitors may prove useful. Oral Zafirlukast 40-80 mg 12 hourly for the initial 48 hours and Glutathione may lessen respiratory damage after phosgene poisoning.[5,33]

IV. Vomiting, incapacitating and harassing Agents

Vomiting agents (such as adamsite and diphenylchloroarsine), tear gases [(such as 2-chlorobenzalmalononitrile (Csgas)] and capsacain spray are sensory irritants that are used to temporarily incapacitate targets. Psychoactive drugs (such as LSD and cannabinoids)[5] may also be used, they are severely debilitating but are less likely to require intensive care treatment. With psychochemicals, death is usually accidental while subject is hallucinating or blinded.

V. Blood Agents

Hydrocyanic acid and cyanogen chloride are metabolic poisons which are fatal within 15 minutes of a lethal dose.[5] It is highly volatile, disseminated as a vapor and rapidly disperses throughout the atmosphere to near toxic concentrations. It interrupts cellular respiration by inhibiting cytochrome oxidases. The resulting metabolic acidosis and tissue hypoxia leads to convulsions and cardio-respiratory arrest. Inhalation causes high fatality before hospitalization. Other symptoms are dizziness, confusion and coma. Arterial blood gas shows metabolic acidosis. Antidote is Na-thiosulphate which converts cyanate to non toxic thiocyanate. It is administered in conjunction with sodium nitrite which converts Hb to met-Hb (which binds cyanide) and hydroxycobalamin which reacts with cyanide to form cyanocobalamin. Infusion of norepinephrine may be required to counteract hypotensive side effects of sodium nitrite.

VI. Toxins

Saxitoxin, Ricin and Botulinum toxin are biological products and are the most toxic chemicals that are produced by living organisms. They are considered by some to be chemical weapons as their effects do not require replication in humans.

Botulinum Toxin

Clostridium botulinum produces 7 distinct but chemically and functionally distinct neurotoxins [A-G]. Neurotoxin A is 500 times more toxic than sarin nerve gas.[5] Aerosolisation is the most likely method of deployment but sabotage of food supplies may also occur. All serotypes bind to presynaptic receptors at cholinergic synapses. Acetylcholine synthesis is permanently inhibited. Functional recovery occurs by genesis of new terminal boutons. The toxin blocks neurotransmission at neuromuscular junction, postganglionic parasympathetic synapses and peripheral ganglia. One to four days after exposure (depending on the dose inhaled), bulbar palsy and ocular symptoms occur, followed by progressive symmetrical descending weakness that culminates in respiratory failure requiring prolonged ventilatory support.[5] Standard decontamination procedures are sufficient to prevent exposure of health care workers during early phase of treatment after deliberate deployment of botox. Botox is inactivated in 12 hours.[5] Early ventilatory support is associated with a fatality of less than 5%. A trivalent antitoxin exists for serotypes A, B and E, which appears effective if given shortly after oral ingestion of botox. A heptavalent antitoxin exists for all serotypes of botox but its human efficacy is not known.[5]

Ricin is a protein derived from the seeds of the castor plant Ricinus Communis. Waste from the commercial production of castor oil contains 5% ricin, making it easy for such a substance to fall in the hands of terrorists. It acts by interrupting protein synthesis in cells. Ingestion causes abdominal pain and diarrhea. Inhalation of high doses is fatal. Low doses are associated with drowsiness, confusion, convulsions, coma, extreme weakness and cardio respiratory arrest progressing to multi-organ failure and death. Treatment is supportive.

Saxitoxin is produced by dinoflagellate sea organisms. It is concentrated in shellfish and is responsible for cases of paralytic shellfish poisoning. It is 20 times more potent than sarin nerve gas. It inhibits sodium ion channels. Ingestion produces abdominal pain and diarrhea. Inhalation is rapidly fatal and characterized by bulbar palsy, respiratory and cardiac failure. Treatment is supportive and neurological symptoms are not reversible.

B. Biological Agents

These are defined as living organisms, whatever their nature, or infective material derived from them, which are intended to cause disease or death in man, animals or plants. Like chemical weapons, biological weapons are also classified according their intended target. Those chosen for use are similar in character; they are released in low dose into an unprotected population that has poor natural immunity[5] and consistent production of a rapidly occurring high rate of fatality or incapacitance.

1. Viruses

The viruses used in biological warfare are highly infectious and lethal e.g. those producing viral hemorrhagic fever (VHF). VHF describes a range of symptoms that are caused by a variety of RNA viruses e.g. Crimean Congo fever, Ebola virus and yellow fever, viral encephalitides and Variola. Treatment beyond supportive measures is not available. Isolation and contact precautions are required.[5]

Viral Encephalitides: There are three members of the genus Alpha virus that cause viral encephalitis in humans, Venezuelan Eastern and Western[5,34,35] and equine encephalitis viruses (VEE,EEE and WEE). They are highly infectious (10-100 organisms cause clinical symptoms) and stable when weaponised. Mortality may be as high as 70%. No specific therapy exists, and treatment is therefore supportive. Vaccines are available but the WEE and EEE vaccines are poorly immunogenic requiring repeated immunization.[5]

Smallpox is caused by Variola, which is highly infective (10-100 organisms cause infection) when aerosolized, and stable when weaponized. It has a high mortality rate (3% in vaccinated, 30% in unvaccinated); death resulting from pneumonia.[36] Cessation of routine vaccination has increased the susceptibility of the population to variola infection.[5] Cidofovir, a DNA polymerase inhibitor used to treat cytomegalvirus in AIDS patients appears to be effective in vitro when given early after infection.[5]

2. Bacteria

They are easy to culture and have high infectivity and lethality. Common agents used include Bacillus anthracis (anthrax), Yersinia pestis (plague) and Francisella tularensis (tularemia).

Bacillus Anthracis is an aerobic Gram positive, rod-shaped spore forming bacteria that primarily infects the herbivores, particularly cattle, sheep, goats, and horses. The reservoir of B. Anthracis is the soil and the organism is distributed worldwide.[5] Humans usually contact anthrax through close contact with infected animal products particularly hair and hides. Three clinical presentations are usually seen in humans.[37]

1. Cutaneous anthrax results from inoculation of spores through skin abrasions. It appears within 5 days of exposure, beginning with small pruritic papules which form vesicles. These rupture within a week to leave an ulcer that resolves as a black eschar.

2. Inspiration of anthrax spores can result in the highly lethal inhalational form of the disease (woolsorters disease). Inhaled spores reach the alveoli and are phagocytosed to hilar and mediastinal lymph nodes and a large amount of toxin is released into the circulation. Initially there is an insidious onset with malaise, fatigue, myalgia, non-productive cough and fever. Over 1-4 days, a necrotising hemorrhagic mediastinitis ensues causing chest discomfort, acute dyspnea and stridor. Hemorrhagic meningitis with meningism and coma occurs in 50% of patients. Multi-organ failure which is refractory to treatment is the cause of death within 24-36 hrs. Historically, Penicillin was used for treatment, but now it has proved possible to bioengineer penicillin resistance in B. Anthracis. Currently, treatment with Ciprofloxacin is commenced as soon as possible. Chemoprophylaxis can be done with ciprofloxacin or doxycycline, which is continued for 4 weeks or until 3 doses of vaccine are given. Attenuated vaccine is available (Michigan vaccine) and injected subcutaneously at 0, 2 and 4 weeks then at 6, 12 and 18 months with annual boosters. New vaccines that target the protective antigen moiety of the anthrax toxin are being developed.[5]

In a recent event on October 9, 2001, a letter containing anthrax spores were mailed from New Jersey to Washington, D.C. Five postal workers who handled the mail suffered from inhalational anthrax. The 2 postal workers who died had nonspecific prodromal illnesses. One developed predominantly gastrointestinal symptoms including nausea, vomiting, and abdominal pain. Both ultimately developed respiratory failure, requiring mechanical ventilation. The duration of illness was 5 days from onset of symptoms to death. Both died within 24 hours of hospitalization. Without a clinicians high index of suspicion, the diagnosis of inhalational anthrax is difficult during non specific prodromal illness.[5,38]

3. Ingestion of infected meat can lead to gastrointestinal anthrax. Pharyngeal ulcers and edema necessitate an artificial airway. Hemorrhagic mesenteric adenitis, ascites, bleeding per rectum and hematemesis may occur.

Plague is caused by Yersinia pestis, which is an anaerobic, gram-negative coccobacillus. There is documented evidence of the use of plague as a biological weapon, as far back as the 14th century. Plague is transmitted to humans in one of three ways, by flea vectors (Xenopsylla Cheopsis) from rodent reservoirs, by animal to human droplet infection, or by human to human droplet infection.[5] Bubonic, septicemic and pneumonic forms of infection are recognized. Pneumonic plague is the most likely result of a deliberate epidemic.[5] Bubonic plague has a mortality rate of 40% and pneumonic plague has a mortality of 100%, unless treatment is commenced within 24 hours. Treatment is with Streptomycin 30mg/kg twice daily for 10 days, the alternate are gentamycin, doxycyclin, or chloramphenicol.[5]

Tularemia is caused byFrancisella tularensis[39], a small, aerobic, intracellular, Gram negative coccobacillus.[5] Transmission to humans normally takes place after inoculation by arthropod vectors. The ingestion of infected meat or inhalation of aerosolized bacteria may also result in infection. It may be produced by as few as 10-50 organisms. The more common ulceroglandular form of the disease occurs after inoculation. The less common but more fatal (35%, if untreated) typhoidal form of tularemia occurs after inhalation and presents with fever, anorexia and non productive cough. Pneumonia may develop and can be complicated with pleural effusion.

Diagnosis is confirmed by isolation of the organism in the sputum or blood. Treatment is with streptomycin 30mg/kg every 12 hours for 10-14 days[5] or Gentamicin 3-5 mg/kg/day for 10-14 days. A live attenuated vaccine is available.[5]

Principles of Management

A multi disciplinary approach will be necessary to address emergency medical and emergency public health needs.[19] The first step is to identify, demarcate and cordon off the contaminated area. Definitive identification of the agent may take several hours or even days, clinical signs and symptoms in exposed individuals are useful indicators of the likely agent, and will be critical in guiding emergency medical care.[19]

1. Initial Management in Hospital and ICU

Disaster planning for managing a chemical or biological attack must be developed and realistic training should be provided to ensure effective response to an actual terrorist event.[40] Triage of individuals exposed to a CBW attack poses several challenges, but the underlying principles are the same as for any multiple casualty incident.[19] A triage station should be established in the hot zone to assist in determining priorities for resuscitation, decontamination, pharmacological therapy, and site evacuation. It is a dynamic process and should occur at every stage of patient management.

Triage can be classified as:

i. Primary: at site of disaster, and

ii. Secondary: when patient arrives in hospital.

Patients are divided into 4 groups of treatment category:[9]

1.	Immediate	life saving treatment required immediately
2.	Delayed	treatment can be delayed without harm
3.	Minimal	walking wounded
4.	Expectant	death inevitable

Initial categorization of the patients is very important in the management of mass casualties involving the chemical and biological agents.

II. Decontamination

Decontamination may not be necessary if the victim has been exposed to vapor alone, but rapid decontamination may be required if exposure is to liquid or aerosolized form of an agent.[3,41] When indicated, decontamination should be performed as close to the scene as possible (i.e. in the warm zone), and ideally before patient transportation.[19] Removal or neutralization of CBW agents should be done to limit human exposure. Personal or small-scale decontamination may be instigated by dilution [showering], or use of chemical agents [soap or hypochlorite solution]. Decontamination in the ambulatory reception area will minimize exposure of hospital staff. Health personnel should be trained in the use of protective equipment which includes face masks, chemical resistant gloves, splash suits, and air purifying respirators. Detection equipment is required to monitor levels of CBW agents. Priority is given to lifesaving treatment over decontamination. It is preferable for the patients to decontaminate themselves.

III. Protection of the Staff

Medical responders may be required to work in protective clothing and masks[19,40], but the hospital staff may not have access to protective equipments. Therefore, it is essential to insist on effective decontamination procedures to be carried out before hospital admission. Barrier protection will make care of patients more difficult and

increase the risks of heat, fatigue, and isolation stress for medical personnel.[40,41] Strategies to decrease infection rates include:

1. Isolation of patients for droplets, body fluids, blood and secretions
2. Correct disposal of clinical waste
3. Basic hygiene
4. Vaccination
5. Post exposure prophylaxis

IV. Definitive Management

The rapid identification of the causative agent may assist in determining early medical and public health interventions. Definitive identification may require the facilities of specialized analytical laboratory and generally requires some time. Clinical manifestations in the exposed individuals may be the most useful indicators of the likely agent and will be critical in guiding emergency medical care. In addition, the samples of air, soil, water, munitions, and biological materials may be required to precisely identify the agent and to quantify the level of exposure. Management is generally supportive, unless a definitive diagnosis has been established, and a specific antidote is available.

V. Public Education

The use of public warning and information systems will be critical to inform the community about the nature of the incident and the appropriate measures that they can take to protect themselves.[42] Timely and accurate information will assist in minimizing panic in the affected community. Measures to reduce exposure of the public to chemical agents include evacuation, sheltering in place, and the distribution of gas masks when nerve gases are suspected.[43-45]

Apart from controlling a specific event, continuing public education regarding disaster planning and management may play a very significant role in the ultimate outcome.

Conclusion

Education of the public and institutional preparedness can mitigate the horror of CBW agents. The media can play an active prevention role, by realistically educating the public about the impact of CBW attack, as the threats posed by biological weapons are likely to continue into the future.[19] The use of public warning systems, will be critical to inform the community about the nature of the incident, and the appropriate measures that they can take to protect themselves.[46] The stresses associated with a biological terrorist attack could create, high numbers of acute and potentially chronic psychiatric casualties who must be recognized, diagnosed and treated to facilitate triage and medical care.[40]

Every effort should be made to ensure safety of personnel and other patients. Inadequate personnel protection reduces the efficiency and efficacy of the medical response. Despite the alarming projected mortality statistics quoted for a significant CBW attack, actual mortality has been relatively low. Morbidity, however, has been high; reflecting a lack of medical preparedness for such an attack. Only by planning, and investing, in the right training and defensive measures, we can decrease the risks, disruptions, and casualty morbidity and mortality. By pre-empting and improving our readiness to respond to terrorism, many lives can be saved and terrorists denied their goal of creating panic and crises situations.[46]

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