HISTORY OF NERVE AGENTS:

- GA (Tabun)
- GB (Sarin)
- GD (Soman)
- VX

Nerve agents are highly toxic organophosphate compounds first synthesized in Germany in the late 1930's. These agents are 20 times more deadly than potassium cyanide, 26 times more deadly than cyanide gas and 40 times as toxic as mustard vapor. Just 0.14 milligram per kilogram of body weight (VX), a pinprick sized droplet, will kill a human if gone untreated.

The first compound to be produced was Tabun, followed by Sarin and Soman. These agents were classified by the United States as GA, GB, & GD. The letter G represents Germany, and the following letters A through F designated the order in which the compound was synthesized. GC was not used by the US as it represented gonococcus and GE and GF were not widely produced thus they became obsolete very soon after their synthesis. The United States and England worked very hard after WWII to develop effective forms of protection from these agents. While conducting this research we, as well as the Brit's, were able to synthesize a more stable nerve agent known as V-agents. Ironically, one variant of V-agent was initially produced under the trade name of Amiton and released as an insecticide during the early 1950's in both the US and England. Amiton was soon taken off of the market after consistently finding a large number of mammals killed in the area of usage. In 1958, a British chemist by the name of R. Ghosh synthesized an extremely toxic V-agent known as VX. VX was placed into full-scale production by the United States in April 1961. The Soviet Union was not far behind and produced their own version of VX, which was only slightly different in structure to the US/English version of VX. These V-agents are approximately 170 times more toxic than Sarin (GB). A lethal drop of VX (7-10 mg) will fit between two columns of the Lincoln Memorial on the backside of a penny (approximately 10 mm in diameter).

<table>
<thead>
<tr>
<th>Toxicity</th>
<th>LD50 Chart mg/70kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>GA (Tabun)</td>
<td>1000</td>
</tr>
<tr>
<td>GB (Sarin)</td>
<td>1700</td>
</tr>
<tr>
<td>GD (Soman)</td>
<td>50</td>
</tr>
<tr>
<td>VX</td>
<td>10</td>
</tr>
</tbody>
</table>

The "G" agents tend to be non-persistent and will evaporate at about the same rate as water whereas the VX agents will persist on the ground for three days at 60 degrees F (up to eight days at 14 degrees). Both types of nerve agents present their own unique problems for the rescuer. G agents will tend to "off gas" during the evaporation process thus will present with a "vapor hazard" for emergent personnel even if direct physical contact with the patient is avoided. V agents do not tend to "off gas" but are 5 to 170 times more toxic than G agents and remain in the area where released for much longer periods of time. (Note: Based upon the LD50 chart, VX is 170 times more toxic than Sarin)

The most recent domestic terrorist attack utilizing a nerve agent occurred in Japan in 1995. A Buddhist terrorist group known as the Shoko Asahara attacked a subway full of commuters by concealing the nerve agent Sarin in lunch boxes and soft-drink containers. These containers were placed in various locations on the floor of the subway cars. The agent was then released by puncturing the containers with umbrellas as the terrorists left the trains. Even with this crude method of delivery, over 4000 people were injured in this terrorist incident. At least 493 patients were admitted to area hospitals. Twelve people were killed as a result of this terrorist attack.

The lack of immediate access to personal protective equipment (Tyvek or charcoal impregnated over garments as well as respirators) resulted in 135 ambulance personnel succumbing to the effects of Sarin.

There were also 110 hospital personnel affected due to the "off gassing" of the Sarin from the patient's clothing as well as direct contact with the agent imbedded in the garments. This number could have been reduced if proper decontamination procedures had been implemented prior to allowing patients access to the emergency rooms.

Next week I will cover signs and symptoms of nerve agent exposure as well as emergent treatment procedures.
Nerve Agent Exposure:

Nerve agents may be absorbed through any body surface. When dispersed as a spray or aerosol, droplets can be absorbed through the skin, eyes, and respiratory tract. Vapor is primarily absorbed through the respiratory tract. Nerve agents may also be absorbed through the gastrointestinal tract when ingested with food or water. The rapidity with which organophosphate effects occur is directly related to the amount of agent absorbed in a given period of time.

The respiratory tract (inhalation) is the most rapid and effective route of absorption. Local inhalation effects include bronchospasm and bronchorrhea. Local effects after skin exposure are localized sweating and/or muscular twitching. Local effects after vapor or liquid exposure to the eye include miosis and often conjunctival hyperemia. Local effects of liquid on the mucous membrane include twitching or contracting of the underlying muscle and glandular secretions. Absorption of a nerve agent by any route may result in generalized systemic effects. The mnemonic by which most of us associate organophosphate poisoning is:

**SLUDGE**
- Salivation
- Lacrimation
- Uribation
- Defecation
- Gastrointestinal pain & gas
- Emesis

This mnemonic has been replaced with an updated mnemonic **DUMBELS**, which more accurately depicts signs and symptoms one may find when examining patients exposed to nerve agent vapor, aerosol or liquid.

**DUMBELS**
- Diarrhea
- Uribation
- Miosis
- Bradycardia, Bronchorrhea, Bronchospasm
- Emesis
- Lacrimation
- Salivation, Sweating

Mechanism of Action:

The effects of organophosphate nerve agents in general are mainly due to their ability to inhibit acetyl-cholinesterase (AChE) throughout the body. Since the normal function of this enzyme is to hydrolyze acetylcholine (Ach) wherever it is released, such inhibition results in the accumulation of excessive concentrations of acetylcholine at its various sites of action resulting in overstimulation.

Let's review the basics of nerve impulse transmission. Nerve cells are electrically conducting cells, but from one cell to another, the signal is no longer electric, but chemical. When the electrical signal reaches the end of the nerve cell which is conducting it, or reaches the synapse, it causes the pre-synaptic terminal to release packets of the neurotransmitter acetylcholine which diffuse across the space between cells, the synaptic cleft, interacting with post-synaptic receptors on the second cell, and causing the second cell to react. If the second cell is a nerve cell, this will cause a new electrical signal to continue on down the line. If the second cell is skeletal or smooth muscle, the result will be muscle contraction. If the second cell is an exocrine gland, the result will be glandular secretions. The enzyme acetylcholinesterase (AChE) is the turn-off switch to these chemical reactions. It destroys, or hydrolyzes, the neurotransmitter Ach, which ends the reaction and keeps it regulated.

Muscarinic & Nicotinic Receptors:

Prior to discussing the treatment for nerve agent exposure, we must first review the two types of post-synaptic cholinergic receptors. The two types of post-synaptic receptors are muscarinic and nicotinic. Muscarinic cholinergic receptors are found in smooth or non-voluntary muscles, exocrine glands, and certain cranial nerves such as the vagus, which slows the heart. Nicotinic receptors are mostly found in skeletal muscles, but they also sit on pre-ganglionic nerves in the sympathetic nervous system.

If we turn on all of the muscarinic receptors simultaneously, we'll get constriction of all the smooth muscles in the body. In particular, the smooth muscles of the small airways will constrict, causing difficulty breathing. GI tract smooth muscles will constrict, causing
increased peristalsis, increased bowel sounds, and possibly nausea, vomiting, and diarrhea. The pupillary muscle constricts briskly when we turn on the muscarinic receptors resulting in miosis. All of our exocrine neuroglandular junctions will turn on full blast secreting fluid from all of these organs. The most life-threatening reaction will be in the respiratory system, not just from the smooth muscle hyperactivity causing bronchospasm but also from the increased secretions from exocrine glands in the airways.

Turning on all of our nicotinic synapses simultaneously will have effects predominantly at voluntary or skeletal neuromuscular junctions. First we may see fasciculations, tiny involuntary twitchings which don’t cause any movement across a joint. This will proceed to frank twitching, where the muscle now moves a joint. This can be very vigorous and perhaps even mimic tonic-clonic seizure activity, but it’s not a seizure, just a massive overstimulation of the neuro muscular junction itself. When the muscle runs out of energy, ATP, it will fatigue resulting in flaccid paralysis. (Note: Paralysis is never the first thing you’ll see. If you have ever used Raid Wasp & Hornet spray, you can actually see the progression of nerve agent exposure. The insect doesn’t just drop motionless to start with; there is a period of hyperactivity first.) The nicotinic synapses in the sympathetic nervous system can also cause increased blood pressure and heart rate. This activity will counteract the muscarinic effects on the vagus nerve often resulting in normal heart rate and blood pressure initially.

**Atropine** is administered to counteract the effects of muscarinic overstimulation. It works by blocking acetylcholine at the post-synaptic receptor site. Atropine, however, will **not** affect nicotinic receptor sites. This is an important point to remember. Treating nerve agent exposure with Atropine only will allow the overstimulation of the nicotinic to run unchecked. This is the reason Atropine is given in conjunction with Pralidoxime (2 Pam Cl).

**2 Pam Cl** is administered to counteract the effects of both muscarinic and nicotinic overstimulation. It works by binding with acetylcholinesterase (AChE) and actually hydrolyzes the nerve agent allowing the AChE to function unimpeded. 2 Pam must be given early on in the exposure, otherwise, the nerve agent will “age” and bind permanently to the acetyl-cholinesterase enzyme.

Next week, I will continue the discussion of Nerve Agent Treatment as well as discuss methods of personal protection and recognition of “unknown mass casualty scenes” where nerve agent has been released.

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**March 28th, 2003**

**Preventing Nerve Agent Poisoning**

The respiratory tract absorbs nerve agent vapor very rapidly. The protective mask must be put on IMMEDIATELY when it is suspected that nerve agent vapor is present in the air. HOLD YOUR BREATH, put on your mask, clear and seal the mask, then resume breathing. Ensure that you have donned and sealed your protective overgarments, gloves and boots prior to leaving your vehicle. If the nerve agent concentration in the air is high, a few breaths may result in the inhalation of enough nerve agent to be incapacitating or even lethal. When the concentration in the air is low, a longer exposure may precede the onset of symptoms and the detection of nerve agent poisoning. Since the effects of a nerve agent are progressive and cumulative, the prevention of further absorption is urgent once symptoms have begun.

Military experience in chemical operations has shown that when troops become alarmed, some believe they have been exposed to more chemical agents than they actually have been. Hence, it is important that EMS personnel NOT give themselves more than one Mark 1 kit initially with mild signs and symptoms. Employees who are able to breathe normally, ambulate, and know who they are and where they are will probably not need any additional Mark 1 kits administered. (It should be noted that additional administration of Atropine to co-workers with only MILD symptoms must be approached cautiously with at least 10 to 15 minutes elapsing between successive injections. If the signs of nerve agent poisoning disappear, or if signs of Atropinization, such as a heart rate above 90, diminished bronchial secretions, and dry skin, appear during one of these 10- to 15-minute periods, no further injections should be administered. These individuals should remain under observation without further injections of Atropine unless signs of nerve agent intoxication reappear.) However, if symptoms do recur, additional kits (up to two more for a total of three), can be administered. Personnel should consult with a co-worker to determine if he or she needs additional injections of Atropine and 2-Pam Cl.

Note: Additional Mark 1 kits may have to be given by one’s partner or another EMS employee since personnel requiring additional medication may be unable to administer injections to themselves.

Respiratory effort is the most important criteria in determining whether additional Atropine is needed. Laborered breathing, including coughing, wheezing, and gasping for air, indicates the need for administering additional Atropine. Evaluating heart rate is difficult when dressed in protective overgarments leaving the need for additional Atropine based primarily on the degree of respiratory impairment. When adequate Atropine has been given, labored breathing efforts will be relieved. Any assessment of co-workers must be performed without compromising protective measures (mask, suit, gloves, boots).

**NOTE:** DO NOT give nerve agent antidotes for preventive purposes BEFORE contemplated exposure to a nerve agent. To do so may enhance respiratory absorption of nerve agents by inhibiting bronchoconstriction and bronchial secretion. Atropine will degrade
performance when taken in doses of more than 2 milligram (mg) without nerve agent exposure, especially when maximal visual acuity is required. Also, Atropine will degrade an individual's ability to perform duties in a hot environment.

**Essential Elements of Prevention and Treatment**

The essential prevention and treatment elements of nerve agent poisoning are:

- Donning the protective mask (and hood) at the first indication of a nerve agent attack.
- Dress in your protective overgarment, boots & gloves ensuring all are sealed.
- Administering the MARK I kit as soon as any mild to moderate signs or symptoms are noted.
- Administering Diazepam to Severely poisoned casualties.
- Removing or neutralizing any liquid contamination immediately.
- Suction airway secretions if they are obstructing the airway.
- Establishing a patent airway with an endotracheal tube and administering assisted ventilation, if required.

**Remember…**

The most important tool in the prevention of Nerve Agent exposure is your "common sense". Always be suspicious of mass casualty scenes of unknown origin. Be observant in your approach, especially for the presence of dead animals, birds and insects.

These “subtle signs” may be your first and only indication of a Nerve Agent release prior to actually becoming a victim yourself.

Above all else, use the Buddy System when working in a hazardous environment. Watch your co-workers closely for signs and symptoms of Nerve Agent Exposure.

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**April 4th, 2003**

**RICIN**

Ricin is one of the most toxic natural poisons known to man. It is derived from the Castor Bean Plant (Ricinus communis) and is native to the Ethiopian region of Africa. It can be found in temperate regions of the world and is fast becoming an abundant weed in the Southwestern United States. Castor bean is a herbaceous annual that can reach to nearly 15 feet tall when growing in open spaces in warm climates. Large leaves are alternate, palmately lobed with 5 - 11 toothed lobes. Leaves are glossy and often red or bronze tinted when young. Flowers appear in clusters at the end of the main stem in late summer. The fruit consists of an oblong spiny pod that contains three seeds on average. Seeds are oval and light brown, mottled or streaked with light and dark brown and resemble a pinto bean. Castor plants are very common along stream banks, riverbeds, bottomlands, and just about any hot area where the soil is well drained and with sufficient nutrients and moisture to sustain the vigorous growth. Although the seeds or beans are extremely poisonous, they are the source of numerous economically important products and were one of earliest commercial products. Castor beans have been found in ancient Egyptian tombs dating back to 4000 B.C., and the oil was used thousands of years ago in wick lamps for lighting. To many people the castor plant is just an overgrown, undesirable weed, and yet it produces one of nature's finest natural oils.

Ricin is significant as a terrorist biological weapon due in part to its wide availability and ease of manufacture. Worldwide, one million tons of castor beans are processed annually in the production of castor oil; the waste mash from this process is five percent Ricin by weight. Ricin can be produced relatively easily and inexpensively in large quantities in a fairly low technology setting. There is recent evidence that Ricin is being produced by terrorists for use as a biological weapon of mass destruction. On January 15, 2003 a British police officer lost his life in a counter-terrorism raid in Manchester England where Ricin was thought to have been in production. On March 20, 2003 Ricin was found in a Paris railway station in a luggage depot, thought to have been placed there for later retrieval by terrorist agents. Milligram per milligram, Ricin is as toxic as VX Nerve agent. The most important thing to remember about Ricin is that there IS NO KNOWN ANTIDOTE for this toxin.

Next week I will cover the mechanism of toxicity, routes of delivery, as well as signs & symptoms of exposure.
April 11th, 2003

Terroristic Use of Ricin

Ricin, although extremely toxic, must be delivered in sufficient amounts to cause mortality in humans. To be used as a terrorist weapon, Ricin must be distributed either through a food source for human consumption or aerosolized for inhalation. (It would be highly unlikely that Ricin would be delivered intravenously or subcutaneous in this setting). Due to modern methods of food packaging, the most likely distribution of Ricin as a weapon of mass destruction would be through aerosolization.

Mechanism of Toxicity

The toxins are made up of two polypeptide chains, an A chain and a B chain, which are joined by a disulfide bond. Ricin is very toxic to cells. It acts by inhibiting protein synthesis. The B chain binds to cell surface receptors and the toxin-receptor complex is taken into the cell; the A chain has endonuclease activity and extremely low concentrations will inhibit protein synthesis. A single molecule of Ricin entering a cell can inactivate over 1500 ribosomes per minute.

When inhaled as a small particle aerosol, this toxin may produce pathologic changes within 8 hours and severe respiratory symptoms followed by acute hypoxic respiratory failure in 36-72 hours. When ingested, Ricin causes severe gastrointestinal symptoms followed by vascular collapse and death.

When tested on rodents, there are microscopic cellular changes after aerosol exposure. These changes are characterized by necrotizing airway lesions causing tracheitis, bronchitis, bronchiolitis, and interstitial pneumonia with alveolar edema. There is a latent period of 8 hours post-inhalation exposure before microscopic lesions are observed. In rodents, Ricin is more toxic by the aerosol route than by other routes of exposure.

There is little toxicity data in humans. The exact cause of morbidity and mortality would be dependent upon the route of exposure. During the 1940’s there were several accidental sub lethal aerosol exposures of Ricin, which were characterized by an onset of fever, chest tightness, cough, dyspnea, nausea, and arthralgias, which occurred in the first four to eight hours. The onset of profuse sweating some hours later was commonly the sign of termination of most of the symptoms. Although lethal human aerosol exposures have not been observed, the severe pathophysiologic changes seen in the animal respiratory tract, including necrosis and severe alveolar flooding, are probably sufficient to cause death if enough toxin is inhaled. Time to death in experimental animals is dose dependent, occurring 36-72 hours post inhalation exposure. Humans would be expected to develop severe lung inflammation with progressive cough, dyspnea, cyanosis, pulmonary edema, and eventual acute hypoxic respiratory failure.

Diagnosis

An attack with aerosolized Ricin would be, as with many biological warfare agents, primarily diagnosed in the clinical setting. Acute lung injury affecting a large number of cases in a suspected terrorist attack should raise suspicion of an attack with a pulmonary irritant such as Ricin, although other pulmonary agents could present with similar signs and symptoms. Biological agents such as Anthrax and SEB as well as some chemical warfare agents like phosgene need to be included in the differential diagnosis.

- There would be no mediastinitis as seen with inhalation Anthrax.
- SEB would be different in that most patients would not progress to a life-threatening syndrome but would tend to plateau clinically.
- Phosgene induced acute lung injury would progress much faster than that caused by Ricin.

An important point to remember is that Ricin intoxication would be expected to progress, despite treatment with antibiotics.

Essential Elements of Prevention and Treatment

The essential prevention for Ricin in the emergent response setting is the donning of protective masks prior to arrival on scene. Secondary aerosols (similar to off gassing in Nerve agents) should generally not be a danger to health care providers. Clorox diluted to a 0.1% sodium hypochlorite solution and/or soap and water should be sufficient to decontaminate skin surfaces.

- Patients with pulmonary intoxication are managed by appropriate treatment for pulmonary edema and respiratory support.
- Gastrointestinal intoxication is best managed by vigorous gastric decontamination with activated charcoal, followed by use of cathartics such as magnesium citrate.
- Volume replacement of GI fluid loss is also important.
- In percutaneous exposures, treatment would be primarily supportive.

Treatment is based upon the route of exposure. Follow the guidelines on page(s) 44 & 45 of your NBC Field Manual.
April 18th, 2003

Dirty Bomb

The Threat

There has been much discussion, since 9/11, surrounding the use of a “Dirty Bomb” as a weapon of mass destruction. In fact, on May 8, 2002, the FBI captured Abdullah Al Muhajir, a U.S. citizen allegedly working with al-Qaeda to set off a dirty bomb in an American city. The Washington Post reported in March 2002 that the Bush administration’s consensus view was that Osama bin Laden’s al-Qaeda terrorist network probably had in their possession enough stolen radioactive contaminants such as strontium 90 and cesium 137, which could be used to make a dirty bomb. Getting this material into the United States undetected poses the biggest problem for the terrorists at this time.

In January 2003, British officials found documents in the Afghan city of Herat that led them to conclude that al-Qaeda had successfully built a small dirty bomb. It has also been reported that Iraq tested a one-ton radiological bomb in 1987 but gave up on the idea because the radiation levels it generated were not deadly enough. Based upon this information it appears that the threat is real, however, the jury is still out on how effective a “Dirty Bomb” would be as a weapon of mass destruction.

What is a Dirty Bomb?

A “Dirty Bomb”, also known as a Radiological Dispersal Devise (RDD), is in no way a conventional nuclear device. A nuclear weapon detonation involves a fission reaction that generates an extreme amount of heat (several tens of millions of degrees centigrade), neutrons, x and gamma rays, electromagnetic pulse, and a large area of thermal blast devastation. The triggering device for a nuclear weapon is extremely sophisticated, well beyond the technical abilities of most terrorist agents.

In contrast, a “Dirty Bomb” is quite easy to manufacture because it is assembled utilizing conventional explosives such as dynamite, ANFO, C-4, etc. combined with low level radiological material in the form of powder or pellets. However, since September 11, 2001, stringent reporting measures involving theft of radiological material have been put into place regulating this material and hospital waste products. Only one stolen high-risk radioactive source, Iridium-192, has not been recovered in the last five years in the United States. However, this source (Iridium-192) would no longer be considered high-risk because much of the radioactivity has decayed away since it was reported stolen in 1999. In fact, the combined total of all un-recovered sources over a 5-year time span would barely reach the threshold for one high-risk radioactive source.

The idea behind a dirty bomb is to blast radioactive material into the area surrounding the conventional explosion. This could possibly cause buildings and people to be exposed to radioactive material. However, at the levels created by most probable sources, not enough radiation would be present in a dirty bomb to cause severe illness from exposure to radiation. In fact, the primary cause of death in the use of such a device would be from the conventional blast itself.

The main purpose of a “Dirty Bomb” is to terrorize citizens and make buildings or land unusable for a long period of time. Cleanup after such an event would take several months costing perhaps tens of millions of dollars. This is why the “Dirty Bomb” has often been referred to as a Weapon of Mass Disruption.

Response Profile

There are several things that must be considered when responding to events involving explosions. First of all - Don’t assume that the event is over after the initial explosion. Treat every event involving explosion as if there were secondary material involved, in other words, as if it were a Haz-Mat event. There could even be secondary explosive devices timed to explode after the area is saturated with emergent personnel. Use extreme caution when approaching the scene. Always approach upwind and maintain a safe distance from the scene. Remember: Time, Distance, & Shielding. Always wear your bunker gear and respirator when outside of your vehicle. This will provide you the best protection from radiological emissions as well as from biological contaminants both at the scene and when treating patients or the walking wounded. Above all else, use your common sense. Don’t rush in and become a victim yourself.

Next week I will continue the explosives series with suicide bombings and soft targets of opportunity.

April 25th, 2003

Suicide Bombing
The Threat

Suicide terrorism is the readiness to sacrifice one's life in the process of destroying or attempting to destroy a target to advance a political goal. The aim of the psychologically and physically war-trained terrorist is to die while destroying the enemy target.

In the 1980s suicide terrorism was witnessed in Lebanon, Kuwait and Sri Lanka. In the 1990s it had spread to Israel, India, Panama, Algeria, Pakistan, Argentina, Croatia, Turkey, Tanzania and Kenya. With enhanced migration of terrorist groups from conflict-ridden countries, the formation of extensive international terrorist infrastructures and the increased reach of terrorist groups in the post Cold War period, suicide terrorism is likely to affect Western Europe and North America in the foreseeable future.

Key Characteristics

Examination of suicide terrorism across a range of groups has revealed that terrorist groups use suicide bombers when they are both strong and weak. Suicide-capable groups differ in form, size, orientation, goal and support. There are now 10 religious and secular terrorist groups that are capable of using suicide terrorism as a tactic against their governments and/or foreign governments. They are: the Islam Resistance Movement (Hamas) and the Palestinian Islamic Jihad of the Israeli occupied territories; Hezbollah of Lebanon; the Egyptian Islamic Jihad (EIJ) and Gamaya Islamiya (Islamic Group - IG) of Egypt; the Armed Islamic Group (GIA) of Algeria; Barbar Khalsa International (BKI) of India; the Liberation Tigers of Tamil Eelam (LTTE) of Sri Lanka; the Kurdistan Worker's Party (PKK) of Turkey; and the Osama bin Laden network (Al Qaeda) of Afghanistan. A review of the key characteristics of the 10 suicide-capable groups reveals that any group can acquire suicide bomb technology and engage in suicide terrorism.

Method of Operation

The organization of suicide operations is extremely secretive. The success of the mission depends on a number of elements: level of secrecy; thorough reconnaissance; and thorough rehearsals. Secrecy enables the preservation of the element of surprise, critical for the success of most operations.

Thorough reconnaissance enables the group to plan, often by building a scale model of the target. Thorough rehearsals allow the bomber to gain stealth and speed. There are other elements, such as getting the bomber to the target zone and then to the target itself. The bomber is usually supported by an operational cell, responsible for providing accommodation, transport food, clothing and security to the bomber until he/she reaches the target. Resident agents help generate intelligence for the operation, from target reconnaissance to surveillance. The cell members confirm the intelligence. Often, immediately before the attack, the bomber conducts the final reconnaissance.

As a comprehensive knowledge of the target is essential for the success of a suicide operation, terrorist groups depend on building solid agent-handling networks. Some security and intelligence agencies have succeeded in penetrating the agent-handling network of various terrorist groups. In some cases, the only form of defense is to penetrate the terrorist group itself. This is because bombers penetrate governments or societies as sleepers and gradually gain acceptance as a trusted member. Thus the bomber can reach and destroy a valuable target - human or infrastructure.

Method of Delivery

There are six types of suicide improvised explosive devices (IEDs). These are: the human-borne suicide IED, also known as the suicide bodysuit; the vehicle-borne suicide IED; the motorcycle-borne suicide IED; naval craft-borne suicide IED; scuba diver-borne suicide IED; and aerial (microlight, glider, mini-helicopter) borne suicide IED. All these categories have been used in South Asia and the Middle East.

The largest number of suicide IEDs used has been the suicide bodysuit. The suicide body suit has evolved to improve concealment and is becoming increasingly small. Initially, the device was a square block of explosives worn in the chest and the belly area. Gradually, the device evolved into a heart shaped block of explosives placed just above the navel. As body searchers for suicide devices are usually conducted around the abdomen, a group is also developing breast bombs.

Most suicide body suits have no/little electronics, making it difficult for security agencies to develop counter-technologies to detect these devices. A suicide body suit can be made from commercial items. With the exception of the malleable plastic explosives and detonator, all the other components can be purchased from a tailor shop (stretch denim) and an auto shop (steel ball bearings, wires, batteries and switches). Furthermore, when a device is sophisticated it becomes difficult to operate, as well as fixing it when it fails to function. Suicide devices will thus remain simple.
However, there are likely to be variations of suicide devices. Terrorists tend to select from a repertoire of tactics. This is to retain an element of surprise and to evade the attention of security authorities directed at countering a standard set of tactics.

A Growing Threat

The threat of suicide terrorism is likely to spread with time. It is likely that suicide terrorism will affect Western Europe and North America in the future.

Terrorist groups are increasingly providing intensive training to their bombers, with the intention of increasing their endurance. For instance, the suicide bomber who destroyed the U.S. embassy in Nairobi in 1998 had been resident in Kenya for four years. He had married in Kenya and lived in the capital before carrying out the suicide operation. Similarly, the suicide bomber who assassinated President Premadasa of Sri Lanka had lived in the capital, Colombo, for three years before carrying out the attack.

Terrorist groups are setting a dangerous trend of using suicide bombers to destroy targets far away from their traditional theatres of operation. Many groups are likely to use suicide bombers to infiltrate target countries and conduct suicide attacks against Western VIPs and critical infrastructure in the foreseeable future.

May 2nd, 2003

Targets of Opportunity

The Target

Over the past couple of weeks, I have looked around Sedgwick County in an attempt to find areas where terrorists might find “targets of opportunity”. Traditional sights for terrorism such as federal and state buildings, airports, county offices, and city facilities have become somewhat “hardened” over the past two years. Security measures at these sights have become a priority for State and Local officials limiting their “attractiveness” for terrorist attack. As an alternative, terrorists may seek out “soft targets” such as malls, shopping centers, and other public places as they have done in Israel over the past several years, however, these type of attacks are limited in scope and will not have the potential for creating a significant number of casualties that would overwhelm our emergent response for an extended period of time. There is, however, one “target of opportunity” in which a terrorist might seek out that could create a catastrophic scenario for Sedgwick County and the surrounding area. This target is located approximately 6 miles southwest of Wichita in the area of 55th south and Ridge road.

On May 1st 2003 at approximately 1100 hours, I drove to the area of 55th and Ridge just to observe the region around Vulcan and Atofina Chemicals. I was in the area for approximately 30 minutes. During that time I observed less than six civilian vehicles on 55th south, no law enforcement officers and no Vulcan security vehicles. I did, however, notice at least 3 Vulcan transport vehicles leaving the area east bound on 55th. I also noted several Hydrogen Fluoride rail cars parked along the west side of the Garvey grain elevators, well outside of the “secured” fence line of Vulcan and Atofina. In fact, one could walk along the tracks from either Ridge road or 55th south and not encounter any significant barriers that would limit access to these HF rail cars. There were also several chlorine rail cars parked along side the HF cars well outside of the Vulcan fence line. Both the HF and Chlorine vehicles were readily accessible along the dirt access road located on the west side of the grain elevators.

The following is a list of chemicals found at Vulcan. This data was obtained from year 2000 Risk Management Plans (RMP) located on the RTK website.

Atofina Chemicals
No longer in business, however, Hydrogen Fluoride rail cars are visible at the site, of which are not listed in Vulcan’s RMP report. Quantity therefore may be inaccurate as it relates to Atofina & Vulcan Chemicals.

6010 S. Ridge
Wichita, KS 67215

HF Railcar Storage & Unload
   Hydrogen Fluoride - 3,400,000 lbs.
Bulk HF Storage
   Hydrogen Fluoride - 850,000 lbs.
Unit III Reactor System
   Hydrogen Fluoride - 3,600 lbs.
Unit IV Reactor System
   Hydrogen Fluoride - 6,800 lbs.
HF Absorption/Recovery System
   Hydrogen Fluoride - 77,000 lbs.
HCl Purge Acid Storage
   >37% Hydrochloric acid solution - 120,000 lbs.
Chloroform Storage

6020 S. Ridge
Wichita, KS 67215

HF Railcar Storage & Unload
   Hydrogen Fluoride - 3,400,000 lbs.
As you can see from the RMP data, there is several thousand pounds of Hydrogen Fluoride and over 2 million pounds of Chlorine located within one mile of this site. Each Hydrogen Fluoride rail car alone is capable of holding over 167,000 lbs. Each Chlorine car is rated at 90-ton capacity. (Pictures depicting this data are located on pages 4 & 5) As you can see from the photographs, there are several railcars containing these two toxic chemicals both on and off site.

**Worst Case Scenario**

I wondered what would happen if a terrorist targeted just one rail car with an explosive device that would rupture the tank causing the sudden release of the entire contents. To satisfy my curiosity, I obtained several programs from the EPA website that would assist me in making such a determination. Utilizing a program called the RMP Computer, I was able to determine the following for Hydrogen Fluoride (results for Chlorine dispersal are almost identical):

RMP*Comp Ver. 1.07
Results of Consequence Analysis
Chemical: Hydrogen fluoride (anhydrous)
CAS #: 7664-39-3
Category: Toxic Gas
Scenario: Worst-case
Liquefied under pressure
Quantity Released: 167000 pounds
Release Duration: 10 min
Release Rate: 16700 pounds per min
Mitigation Measures: NONE
Topography: Rural surroundings (terrain generally flat and unobstructed)
Toxic Endpoint: 0.016 mg/L; basis: ERPG-2
**Estimated Distance to Toxic Endpoint: >25 miles (>40 kilometers); report as 25 miles**

------ Assumptions About This Scenario ------
Wind Speed: 1.5 meters/second (3.4 miles/hour)
Stability Class: F
Air Temperature: 77 degrees F (25 degrees C)

Applying the same information regarding such a release utilizing CAMEO, ALOHA, and MARPLOT computer programs, I was able to depict what the toxic cloud emanating from a single HF tank car (167,551 lbs) or a single Chlorine tank car (180,000 lbs) would look like superimposed over a map of Wichita.

As you can see from the picture, results of such an attack would be devastating to our community. The toxic cloud release from just one Hydrogen Fluoride or Chlorine railcar would create an area of devastation over 16 miles long and 2 miles wide affecting several thousands of citizens.

**Conclusion**

Based upon my 30 minute observation of security in the area surrounding Vulcan Chemicals and Atofina, it appears that this area may well be classified as a “Soft Target of Opportunity”. We have no way of knowing if terrorist organizations have evaluated this site as a possible target, however, based upon what I observed on May 1st and from information readily available on the internet it appears to me that a potential terrorist target of mass destruction does indeed exist within our community.
Overhead View of Vulcan – 1999

View of HF Tank Car looking SW from 55th S.

View of HF and Chlorine Tank Cars looking E. from 6000 Block S. Ridge Road (Arrows depict HF cars)

View of HF and Chlorine Tank Cars on Siding at Vulcan (Arrows depict HF cars)

Photographs taken at approximately 1100 hrs. – May 1st, 2003
Chlorine
Railroad Tank Car
Terrorist Attack with complete rupture of RR car and contents releasing 180,000 lbs of Chlorine
Temp 70 F - Wind SSW 10 MPH
Location 63rd S & Ridge Road
Toxic Cloud 16 Mi. Long - 2 Mi. Wide
Note: Vulcan has up to 2.2 Million pounds on site
Chlorine (Pulmonary Agents) – by Steve Albright

History

After last week’s article depicting worst-case scenarios involving Vulcan, I thought it might be important to review the effects of pulmonary agents, in particular Chlorine. Chlorine gas was first used as a weapon of mass destruction during the First World War. In order to break a stalemate on the front in Belgium in early 1915, the German Army spent two months implanting 6,000 commercial cylinders of chlorine in their trenches along a 7-kilometer salient near Ypres, Belgium. On the 22nd of April the prevailing westerly winds shifted in favor of the Germans. Over a ten-minute period, they released the contents of all 6,000 cylinders and a six-foot tall, yellow-green cloud wafted toward the French lines. Neither side anticipated the effectiveness of this chemical assault. Those allied troops that did not die in place, broke and ran, leaving a four-mile wide gap in the allied line. While it is unlikely that an adversary would use chlorine to attack US Forces on today’s battlefield, this agent and related compounds (Phosgene) still pose a serious threat to the US military and the American people. Thousands of tons of Chlorine are produced, stored and transported each year in this country, creating the potential for a large-scale release from an industrial accident. The potential for domestic terrorism is also a significant concern. Terrorists use weapons of opportunity. Simply opening the valve on a chlorine or phosgene tank-car near a large metropolitan area could produce mass casualties. Both chlorine and phosgene are ever-present in the chemical industry. These compounds are used as precursors for more complex chlorinated hydrocarbons, the primary components of modern plastics and dyes.

Recent Events

The second largest release of Chlorine in the United States occurred near Alberton Montana on April 11th, 1996. Around 4:00 AM on that date, a 72-car train derailed approximately 1 mile west of Alberton. At least one tank car of pressurized Chlorine ruptured creating a 24-inch gap in the cylinder, venting approximately 122,000 lbs of Chlorine. Over 1000 people were evacuated from an 8 to 15 square mile area, including the entire town of Alberton (population 374). At least one person died as a result of this release and over 352 people were hospitalized. People exposed to the toxic chemical fumes reported a number of health effects: burning eyes and nose, lung irritation and inflammation, sore throats, difficulty breathing, wheezing, coughing up yellow or green sputum, nose bleeds, coughing up blood, headaches and dizziness, and other symptoms or reactions including, depression, lack of motor skills, hopelessness, and anxiety. Exposed animals and livestock also developed reactions: including eye lesions, difficulty breathing, wheezing, which are indicative of lung irritation.

Chemical Properties

Chlorine was discovered in 1774 and was named from the Greek word Khloros, when translated, means “green”. Because it is highly reactive, chlorine is usually found in nature bound with other elements like sodium, potassium, and magnesium. When chlorine is isolated as a free element, chlorine is a greenish yellow gas, which is 2.5 times heavier than air. It turns to a liquid state at -29°F, and it becomes a yellowish crystalline solid at -153°F. Chlorine can be liquefied under pressure for transport by rail and is slightly soluble in water. When released to air, chlorine will react with water to form hypochlorous acid and hydrochloric acid, which are removed from the atmosphere by rainfall. The hypochlorous acid breaks down rapidly. The hydrochloric acid also breaks down; its breakdown products will lower the pH of the water making it more acidic.

Health Considerations

The toxic effects of chlorine are primarily due to its corrosive properties. The action of chlorine is due to its strong oxidizing capability, in which chlorine splits hydrogen from water in moist tissue, causing the release of nascent oxygen and hydrogen chloride which produce major tissue damage. Alternatively, chlorine may be converted to hypochlorous acid which can penetrate cells and react with cytoplasmic proteins to form N-chloro derivatives that destroy cell structure. Symptoms may be apparent immediately or delayed for a few hours. Respiratory Chlorine is water-soluble and therefore, primarily removed by the upper airways. Exposure to low concentrations of chlorine (1 to 10 ppm) may cause eye and nasal irritation, sore throat, and coughing. Inhalation of higher concentrations of chlorine gas (>15 ppm) can rapidly lead to respiratory distress with airway constriction and accumulation of fluid in the lungs (pulmonary edema). Patients may have immediate onset of rapid breathing, blue discoloration of the skin, wheezing, rales or hemoptysis. In symptomatic patients, pulmonary injury may progress over several hours. Lung collapse may occur. The lowest lethal concentration for a 30-minute exposure has been estimated as 430 ppm. Exposure to chlorine can lead to reactive airways dysfunction syndrome (RADS), a chemical irritant-induced type of asthma. Cardiovascular Tachycardia and initial hypertension followed by hypotension may occur. After severe exposure, cardiovascular collapse may occur from lack of oxygen. Metabolic Acidosis may result from insufficient oxygenation of tissues. Dermal Chlorine irritates the skin and can cause burning pain, inflammation, and blisters. Exposure to liquefied chlorine can result in frostbite injury. Ocular Low concentrations in air can cause...
burning discomfort, spasmodic blinking or involuntary closing of the eyelids, redness, conjunctivitis, and tearing. Corneal burns may occur at high concentrations. Pulmonary function usually returns toward baseline within 7 to 14 days. Although complete recovery generally occurs, symptoms and prolonged pulmonary impairment may persist. Exposure to chlorine can lead to reactive airways dysfunction syndrome (RADS).

If victims show signs and symptoms of skin burning or eye injury, remove clothing and decontaminate with soap and water. Victims who present with signs of respiratory distress, without s/s of skin injury, usually do not need decontamination. There is no antidote for Chlorine poisoning so treatment is mainly supportive. Quickly access for a patent airway, ensure adequate respiration and pulse. If trauma is suspected, maintain cervical immobilization manually and apply a cervical collar and a backboard when feasible. High flow 02, ventilation assist with bag-valve mask, and intubation may be required to maintain the victims airway. Treat pulmonary edema as required. (Note: Health information obtained from CDC documents).

**Personal Protection**

EMS personnel should stay upwind and avoid low-lying areas. If entry into the area of contamination is required, ensure that you wear your chemical protective clothing, hood, gloves, and gas mask with a cartridge designed for chlorine (multi-gas) exposure (M-95, 3M P100, etc). Always use the buddy system when working in hazardous areas. Expect to be overwhelmed by the sheer volume of patients if an event as described in last weeks article should occur. Above all else, use your common sense and ensure that you have donned all appropriate gear before entering the area of contamination.

**Photographs of Alberton Montana 1996 Chemical Release**