Chapter 12

RIOT CONTROL AGENTS

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INTRODUCTION

Riot control agents are compounds that cause temporary incapacitation by irritation of the eyes (tearing and blepharospasm), causing them to close, and irritation of the upper respiratory tract. They are often called irritants, irritating agents, and harassing agents; the general public usually calls them tear gas. Like most of the other chemical agents discussed in this textbook, riot control agents are known by two-initial designators that are neither abbreviations nor acronyms of their chemical names but are most akin to code names. Hence an explanation of the derivations of the names is usually not attempted here.

Three types of riot control agents are recognized: lacrimators, which primarily cause lacrimation and eye irritation; sternutators, which mainly cause sneezing and irritation of the upper respiratory tract; and vomiting agents, which additionally cause vomiting. Because these compounds—CS, CN, DM, CR, and CA—have a number of characteristics in common, they are grouped together as riot control agents in this chapter. The small distinctions among them are noted in the discussion of each agent. Table 12-1 lists the chemical, physical, environmental, and biological properties of the three major agents: CS, CN, and DM. Characteristics common to all compounds in this category are

- a rapid time of onset of effects (seconds to several minutes),
- a relatively brief duration of effects (15–30 min) once the victim has escaped the contaminated atmosphere and has decontaminated (ie, removed the material from his clothing), and
- a high safety ratio (the ratio of the lethal dose [estimated] to the effective dose).

Riot control agents all produce effects by sensory irritation, causing extreme discomfort or pain in the organs affected. The eyes, nose, and respiratory tract are the primary organs affected, although the skin is also often involved. The compounds produce temporary disability because the extreme eye irritation and blepharospasm cause the eyes to close temporarily, and the irritation of the airways causes coughing, shortness of breath, and sometimes retching or vomiting. One of these compounds, DM, is noted for also causing vomiting and malaise.

The United States does not recognize riot control agents as chemical warfare agents as defined in the Geneva Convention of 1925. The Geneva Gas Protocol of 1925 was ratified by the United States on 22 January 1975. At that time, the United States interpreted the protocol as prohibiting the first use of lethal chemicals, but not of nonlethal ones such as riot control agents or herbicides.

During the Vietnam War, before the protocol ratification, the United States had used the riot control agent CS (*o*-chlorobenzylidene malononitrile) extensively. On 8 April 1975, President Ford signed Executive Order 11850, which unilaterally renounced first use of riot control agents in armed conflict, with specified exceptions. These exceptions include first use for riot control in areas under direct U.S. military control (including control of rioting prisoners of war), use in rescue operations, use in situations in which civilians screen or mask attacks, and use in rear echelons to protect convoys from terrorists or similar groups. Presidential approval is required in advance for either first or retaliatory use of riot control agents in war.

Of all the compounds discussed in this book, riot control agents are perhaps the most scrutinized by the public. In civilian life, law enforcement agencies use riot control agents in civil disturbances, riots, or to avoid using deadly force. The military commonly uses them in training. The symptoms described below, therefore, will be familiar to most military personnel.

HISTORY

Irritant compounds were allegedly used by Marcus Fulvius against the Ambracians in the second century BC. The Byzantines apparently knew of the efficacy of using irritant substances to harass the enemy. Plutarch described a Roman general who used an irritant agent cloud in Spain to drive the enemy out of concealment in caves,[1] a use similar to that of the United States in Vietnam 2,000 years later.

Modern use probably began in the 1910–1914 period, when ethylbromoacetate was employed against criminals by French police. At the beginning of World War I, some of these former policemen, who were then in the French army, began to use some of these munitions on the battlefield with some degree of success. Although the German use of chlorine at Ypres, Belgium, on 22 April 1915 is generally heralded as the

TABLE 12-1

CHEMICAL, PHYSICAL, ENVIRONMENTAL, AND BIOLOGICAL PROPERTIES OF CS, CN, AND DM

Properties	*o*-Chlorobenzylidene Malononitrile (CS)	1-Chloroacetophenone (CN)	Diphenylaminearsine (DM)
Chemical and Physical			
Boiling point	310°C	248°C	410°C with decomposition
Vapor pressure	0.00034 mm Hg at 20°C	0.0041 mm Hg at approx 20°C	4.5×10^{-11} mm Hg at 25°C
Density:			
Vapor	—	5.3*	—
Liquid	—	1.187 g/mL at approx 58°C	—
Solid	Bulk: 0.24–0.26 g/cm^3 Crystal: 1.04 g/cm^3	1.318 g/cm^3 at approx 20°C	Bulk: < 1 g/cm^3 Crystal: 1.65 g/cm^3 at 20°C
Volatility	0.71 mg/m^3 at 25°C	34.3 mg/m^3 at approx 20°C	Not of practical significance
Appearance and odor	White crystalline powder with pungent odor (pepper)	Fragrant (like apple blossoms)	Yellow-green, odorless, crystalline substance
Solubility:			
In Water	Insoluble	Insoluble	0.0064 g/100 g at room temperature
In Other Solvents	Organic solvents: complete	Organic	Best: acetone, 13.03 g/100 g at 15°C
Environmental and Biological			
Detection	No detector	No detector	No detector
Persistency:			
In Soil	Varies	Short	Persistent
On Materiel	Varies	Short	Persistent
Skin Decontamination	Soap and water	Soap and water	Soap and water
Biologically Effective Amount:			
Aerosol (mg•min/m^3)	LCt_{50}: 60,000 ICt_{50}: 3–5	LCt_{50}: 7,000–14,000 ICt_{50}: 20–40	LCt_{50}: 11,000–35,000 ICt_{50}: 22–150; nausea, vomiting: approx 370

*Compared with the density of air
LCt_{50}: the concentration • time (C*t*) that is lethal to 50% of the population exposed
ICt_{50}: the C*t* that incapacitates 50% of the population exposed

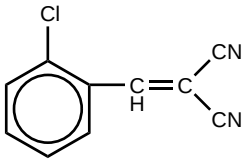
beginning of chemical warfare on the modern battlefield, irritating substances had already been in use for about a year. During World War I, approximately 30 different compounds were tried for their irritant effects, usually without much success.[2] As noted above, a riot control agent was widely used in the Vietnam War.

Riot control agents gained some notoriety when they were used in civil disturbances in Paris, France, in 1968; in Londonderry, Northern Ireland, in 1969; in several protest demonstrations in the

United States in the late 1960s; and in prison riots. More recently, riot control agents were used in an unsuccessful attempt to drive the Branch Davidians from their compound near Waco, Texas, in February 1993.

Probably the best known of these compounds is CN (1-chloroacetophenone); it has been used for many years and is commercially available in devices for self-protection under its proprietary name, Mace (the chemical, not the devices, is manufactured by General Ordnance Equipment Corp., Pittsburgh, Pa.). CS is the compound that is used by the military in most countries and almost exclusively by law enforcement agencies throughout the world.

CS (*o*-CHLOROBENZYLIDENE MALONONITRILE)



CS

The riot control agent known as CS (*o*-chlorobenzylidene malononitrile) was first synthesized in 1928 by Corson and Stoughton (hence its code name). It replaced CN as the standard riot control or irritant agent in the U.S. Army in 1959. In the late 1950s, CS was also adopted by most U.S. law enforcement agencies and by the military and law enforcement agencies of other countries, because CS is more effective than CN (it causes effects at lower doses) and is less toxic (ie, its LCt_{50}, the vapor or aerosol exposure [concentration • time] that is lethal to 50% of the exposed population, is higher).

Physical Characteristics

CS is a white, crystalline solid with a low vapor pressure. It is almost insoluble in water and only slightly soluble in ethyl alcohol and carbon tetrachloride. Because of these physical characteristics, decontaminating buildings, furniture, and other material after CS use in urban riots is difficult. Dissemination of CS can be by explosive dispersion of a powder or solution, by dispersion of the powder in a fine state, by spraying a solution, or by releasing as smoke from a pyrotechnic mixture.[3] The method of dissemination may influence the severity of the injury (see eye injury for CN). The Material Safety Data Sheet, which the manufacturer includes in each package, assigns it a flammability rating of 4 (on a scale of 0 to 4). The agent was a large contributor in the conflagration that burned the Branch Davidian compound and its inhabitants in Waco, Texas, in 1993.

CS tends to agglomerate when used and resists weathering poorly (losing its effectiveness). During the mid 1960s, hydrophobic formulations of CS, CS1 and CS2, were developed. The former is a micronized powder with 5% hydrophobic silica aerogel; the latter is a siliconized, microencapsulated form of CS1. CS1 and CS2 last for several weeks and are a persistent hazard during military operations. Because of their persistence, they have not been used for civil disturbances.

Clinical Effects

Clinical effects common to all of these riot control agents are listed in Exhibit 12-1. In the eye, an initial burning feeling or irritation progresses to pain accompanied by blepharospasm, lacrimation, and conjunctival injection. The intense blepharospasm causes the eyes to close. Photophobia is often present and may linger for an hour. The mucous membranes of the mouth, including the tongue and palate, have a sensation of discomfort or burning, with excess salivation. Rhinorrhea is accompanied by pain inside the nose and perhaps around the external nares. When inhaled, these compounds cause a burning sensation or a feeling of tightness in the chest, with coughing, sneezing, and increased secretions. On unprotected skin, especially if the air is warm and moist (see skin effects of CS), these agents cause tingling or burning; within a few minutes, erythema may develop at the exposed sites.

Tolerance to Exposure

Typically, effects appear within seconds of exposure to an aerosolized compound and worsen as long as one remains in the cloud. Most effects slowly dissipate, starting within a few minutes after one leaves the contaminated area. By 30 minutes, most effects have completely abated, although the usually mild erythema may persist for 1 to 2 hours. If one does not leave shortly after the onset of irritation, the effects might become more severe, with marked coughing, gagging, retching, and vomiting.

Most individuals note marked harassment at a concentration of 3 to 5 mg/m^3 and leave the area

EXHIBIT 12-1

CLINICAL EFFECTS OF RIOT CONTROL AGENTS

Eye
- Burning, irritation
- Conjunctival injection
- Tearing
- Blepharospasm
- Photophobia

Skin
- Burning
- Erythema

Gastrointestinal Tract
- Gagging
- Retching
- Vomiting

Airways
- Sneezing
- Coughing
- Tightness in the chest
- Irritation
- Secretions

Nose
- Rhinorrhea
- Burning pain

Mouth
- Burning of mucous membranes
- Salivation

as soon as possible.[4] Tolerance develops, however, in those who have been in close contact with CS for a period of time, such as production or laboratory workers. Those who have developed tolerance can stay in their accustomed concentration of CS and the discomfort does not increase, and, in fact, may decrease. Those who work in a CS environment and get CS on their clothing often become so accustomed to its effects that they wear the clothing out of the area without remembering, only to have others complain.

Tolerance was examined experimentally in an early study[5] in which men were placed in a concentration of 0.43 mg/m^3; the concentration was slowly increased to 2.0 mg/m^3 over 60 minutes. If the men were able to withstand the initial effects, they could remain at the higher concentration. During this time, some subjects played cards and two attempted to read.

In a similar study,[4] when four subjects were exposed to a low concentration that was increased to 6 mg/m^3 over 10 minutes, three subjects left before the time was up. In contrast, when the same subjects were exposed to the same low concentration that was slowly increased to 6 mg/m^3 over a 30-minute period, three remained for 30 more minutes (the fourth subject left after 2 min because of coughing, but voluntarily returned for the remainder of the period). Individuals did not develop tolerance to the compound after ten exposures of 1 to 13 mg/m^3 over a 2-week period.

Duration of tolerance was reduced in exercising individuals, presumably because of deeper breathing and deeper penetration of the particles into the lung, and chest symptoms were more pronounced than when the subjects were exposed while resting. An increase in tolerance was noted when the temperature was low (–18°C; 0°F); a slight decrease in tolerance occurred in a hot environment (36°C). Skin symptoms (such as a burning sensation) were more prominent at the hot temperature than at moderate (20°C–32°C) temperatures.[4]

One might expect that personality and mental set could determine tolerance to CS; a dedicated hijacker, for example, might be able to resist its effects. To test for a correlation between personality and tolerance to an irritant compound, a group of men were exposed to CS, then tested on the Minnesota Multiphasic Personality Inventory (MMPI).[6] Those individuals with less tolerance to CS were characterized by the MMPI by greater use of denial, repression, and somatic complaints than the more tolerant group. Furthermore, the more tolerant group had a higher mean general intelligence score (127 compared with 100 for the less tolerant group).

In a similar study,[7] subjects with high scores classified as abnormal on certain MMPI scales tolerated less CS than did subjects with normal scores. After the administration of diazepam, the tolerance to CS was significantly increased in the group with abnormal scores, but not in the group with normal scores. This result suggests that anxiety, which was reduced more by diazepam in the group with abnormal scores, plays a role in tolerance.

Respiratory Tract Effects

Inasmuch as CS is usually disseminated as an aerosol (powder or in solution), the most common route of absorption is by inhalation. In an LCt_{50} study,[8] four species (rat, rabbit, guinea pig, and mouse) were exposed to aerosolized CS powder for 5 to 60 minutes. The LCt_{50} values (based on mortality within 14 d) ranged from 50,010 mg•min/m^3 (in the mouse) to 88,480 mg•min/m^3 (in the rat). No animal died during exposure; most of those that died afterwards did so within 2 days. The lungs of those dying were congested and edematous, and many had hemorrhages. The trachea was congested with moderate amounts of mucus. On microscopical examination, moderate to marked congestion of

alveolar capillaries and intrapulmonary veins, inter- and intraalveolar hemorrhages, and excess secretions in the smaller airways were seen. Animals that died after 48 hours also had evidence of early bronchopneumonia. Those that survived for 14 days had normal lungs on gross and microscopic examination.

Pyrotechnically dispersed smoke from a CS grenade was used in a similar study design with the same four species.[9] At high concentrations and exposure times of 5 to 20 minutes, the LCt_{50} values (based on mortality within 14 days) ranged from 35,000 mg•min/m^3 (in the guinea pig) to 76,000 mg•min/m^3 (in the mouse). No animal died during exposure, and only two died within 12 hours of removal from the chamber. With concentrations ranging from 31.9 to 56.4 mg/m^3 and a 5-hour per day exposure for 1 to 7 days, the LCt_{50} values (14-day mortality) were from 25,000 mg•min/m^3 (rat) to 54,000 mg•min/m^3 (rabbit).

The lungs of animals that died before 14 days were edematous and congested, with areas of hemorrhage and excessive amounts of mucus in the trachea and bronchi. The alveolar capillaries and intrapulmonary veins were congested, with areas of alveolar hemorrhages and hemorrhagic atelectasis. A few had edema, but no inflammatory cell infiltration was noted. In addition, most animals had evidence of circulatory failure, with dilated right ventricles and enlarged livers, kidneys, and spleens.[9]

Animals that survived 14 days had no abnormalities on pathological examination. The investigators pointed out that the presence of pulmonary edema and hemorrhages in the absence of inflammatory cell infiltration suggests that the smoke caused direct injury to the pulmonary capillary endothelium and that the main cause of death was pulmonary damage. They also commented that, because of the agglomeration of the smoke particles and subsequent precipitation of the compound, concentrations as high as those used could not be maintained under operational conditions.[9]

Two hundred sixty-four rats and 250 hamsters were exposed to CS concentrations of 750, 480, or 150 mg/m^3 for 30, 60, or 120 minutes, respectively (the calculated Ct values were 22,500, 28,800, and 18,000 mg•min/m^3, respectively). Only one animal died in the first 6 hours after exposure; 33 died within 48 hours, and 31 of these were in the 480-mg/m^3 (60-min) group. Those dying within 48 hours had moderately severe congestion in the lungs, with alveolar hemorrhage and edema in some. Acute tubular necrosis was present in some of the animals. In contrast, no deaths occurred (in 48 animals) within 48 hours in the 750-mg/m^3 group, and only two deaths (in 240 animals) occurred in the 150-mg/m^3 group. In these animals and in those sacrificed at 24 hours and onward, minimal abnormalities were found.[9]

In a continuation of this study,[9] rats were exposed to CS at 1,000 to 2,000 mg/m^3 for 5 minutes per day for 5 days. None of the rats died. Minimal pathological changes were found on sacrifice of the animals, but 5 of 56 had bronchopneumonia. A group of 50 rats was exposed to a concentration of 12 to 15 mg/m^3 for 80 minutes daily for 9 days.[9] Five rats died from bronchopneumonia and on sacrifice, 5 of the remaining 45 rats were found to have bronchopneumonia.

In a long-term study,[10] mice were exposed to 3 or 30 mg/m^3 of CS for 60 minutes per day for 55 exposures and then observed for 6 months longer. A daily exposure of 192 mg/m^3 for 60 minutes per day was stopped after three exposures because of deaths. Rats and mice were also exposed to these doses daily for 120 days; daily exposure at 236 mg/m^3 was stopped after 5 days. At the two low concentrations (3 and 30 mg/m^3), the number of deaths over the year of study did not exceed the number of deaths in control groups, which were exposed to air in the exposure chamber daily. After a year, mice receiving 30 mg/m^3 had a statistically significant increase in chronic laryngitis and tracheitis, but otherwise the pathological findings for these animals were not different from those of the control group. In particular, no relationship was found between specific tumors and the total dose of CS.

Dermatological Effects

CS is a primary irritant to the skin. In addition, individuals may develop allergic contact dermatitis after an initial, uneventful exposure to it.

Typically, several minutes after an acute exposure to a low concentration of CS, a prickly feeling or burning is felt in exposed areas of skin. This sensation is more noticeable if the skin is wet or freshly abraded (eg, after shaving). The sensation may be accompanied or followed by erythema, which usually persists for an hour or less. Under certain circumstances—involving the amount of CS, the temperature, and the humidity—a more intense erythema may follow about 2 hours later. If the amount of CS, the temperature, and the humidity are all high, the erythema becomes even more severe, and edema and vesication appear hours later. The time course is the same as that for the skin damage after exposure to mustard.

To test the effects of CS on human skin, the arms of volunteers were exposed to high concentrations of CS thermally generated from an M7 grenade.[11] The exposure was at a temperature of 36°C and humidity of 100%; the average concentration was 300 mg/m^3, and exposure times ranged from 15 to 60 minutes. All subjects noted stinging about 5 minutes after onset of exposure. After being withdrawn from the apparatus, the arms were rinsed with cold, running water to remove the powder that clung to hairs; this procedure caused the stinging to increase. C*t* values of 4,440 and 9,480 mg•min/m^3 caused an immediate skin response: a patchy, vascular erythema, which subsided after 30 minutes with no further reaction.

C*t* values of 14,040 and 17,700 mg•min/m^3 caused a more severe initial dermal response, which required 3 hours to disappear. After 12 to 24 hours, a delayed reaction, consisting of first- and second-degree burns, appeared. Blistering occurred in four of the eight subjects (Figure 12-1). With treatment (discussed below), these lesions resolved in 10 to 14 days; by 6 weeks later, a small amount of post-inflammatory pigmentation remained.[11]

By means of a sleeve with removable patches, arms of volunteers in another study[12] were exposed to CS thermally generated from an M7 grenade. The patches were removed at appropriate times to give C*t* exposures of 1,550 to 33,120 mg•min/m^3 at tropical conditions (37°C; 98% relative humidity) or at one of three temperate conditions (14°C and 41% relative humidity; 20°C and 95% relative humidity; 22°C and 72% relative humidity). No subjects at 14°C or 22°C had the delayed erythema at C*t* values of up to 25,560 mg•min/m^3. At 20°C (95% relative humidity), all four subjects had minimal delayed erythema at C*t* values of 26,025 or 30,240 mg•min/m^3. In contrast, at the tropical conditions, the effective C*t* for producing delayed erythema was 3,500 mg•min/m^3.

The authors of the study pointed out that many variables make it difficult to predict which individuals might be more sensitive than others. Among these variables are skin pigmentation, eye color, complexion, and susceptibility to sunburn.[12]

Although the conditions of these studies were severe, serious skin reactions can occur under milder, more common conditions. First- and second-degree burns were produced in a group of U.S. Army Chemical Corps officers on a field exercise.[13] Temperature and humidity were high, it had been raining heavily, and their uniforms were soaked through. The officers, who were wearing fatigues, ponchos, and M17 protective masks, were hit with a cloud of micropulverized CS1 from a disperser; soon afterwards, they noted burning of their unprotected skin. About 2 hours later, some of the men hosed off and some changed clothes, but most did neither. About 14 to 16 hours after exposure, blistering began, and all of the men who had not hosed off or changed clothes eventually developed vesication.

Firemen in Washington, D. C., were frequently exposed to CS during the riots of April 1968; in addition, they were exposed to CS as they entered buildings in which CS had been disseminated. The CS on floors or furniture was reaerosolized both by their movement and by the force of water from their hoses. They later developed erythema and edema of periorbital skin and other exposed areas.[14]

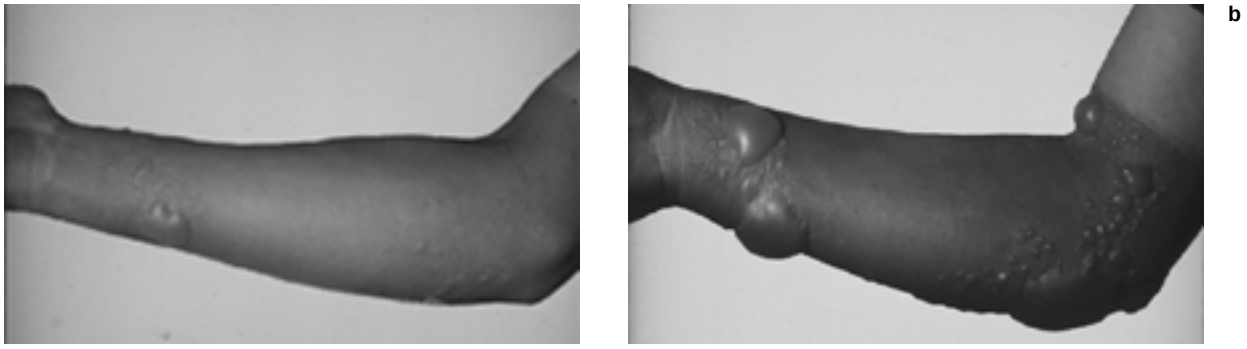


Fig. 12-1. (**a**) Erythema 25 hours after exposure to a high C*t* (the product of concentration of vapor or aerosol • time of exposure; in this instance, 14,040 mg•min/m^3) of CS at 97°F and 100% humidity. (**b**) The same skin lesions at 45 hours, with vesication. Reprinted from Hellreich A, Goldman RH, Bottiglieri NG, Weimer JT. *The Effects of Thermally-Generated CS Aerosols on Human Skin.* Edgewood Arsenal, Md: Medical Research Laboratories; 1967: 19. Technical Report 4075.

Earlier investigators reported vesication after CS patch testing.[4] They also mixed CS with sodium hypochlorite (household bleach) and found that in all subjects tested, the product caused a reaction that was much more severe than that produced by CS alone. For that reason, hypochlorite is not recommended for decontamination of CS on skin. (A hypochlorite is successfully used as a decontaminant for most other chemical agents.)

CS is a primary irritant and causes contact dermatitis, typically in workers in CS-manufacturing or -packing plants. A reaction is more common in warm weather and high humidity or in sweating subjects. The lesion begins some hours after exposure as an erythema, with burning and stinging; the area becomes edematous at about 24 hours, then vesicles or bullae may appear. Common sites are those of partial occlusion, such as the areas under the cuff or glove and under the shirt collar.

CS is also a sensitizer and can cause allergic contact dermatitis, which is the result of a delayed hypersensitivity reaction. An initial exposure may not cause a reaction, but a later exposure to even a small amount produces an often severe dermatitis, with erythema, edema, vesication, and, in severe instances, purpura and necrosis.

Differentiation of the two reactions—primary irritant dermatitis and allergic contact dermatitis—is often difficult clinically and usually requires patch testing.

Ophthalmological Effects

The eye is a sensitive target organ of riot control agents. In studies[14,15] on humans, CS (0.1% or 0.25% CS in water; 1.0% CS in trioctyl phosphate), when placed or sprayed into the eyes, caused inability to open the eyes for 10 to 135 seconds. A transient conjunctivitis but no corneal damage as assessed by slitlamp biomicroscopy resulted.

In another study,[16] subjects were exposed to CS2 (powder dispersal) or CS powder (thermally disseminated) at 0.1 to 6.7 mg•min/m^3 for 20 seconds to 10 minutes. Their visual acuity was tested at intervals during and after the exposure. Subjects who could keep their eyes open during the exposure to read the chart had minimally impaired visual acuity, and no appreciable change in acuity from preexposure readings was found.

In an investigation of the ophthalmic toxicity of CS,[17] rabbit eyes were contaminated with CS in solution (0.5%–10% in polyethylene glycol), as a solid, and as a pyrotechnically generated smoke (15 minutes at 6,000 mg/m^3). The effects were most severe with the solution and least severe with the smoke. After exposure to the smoke, the eyes had a transient, slight excess of lacrimation and congestion of conjunctival vessels lasting 24 hours; the tissues were normal when examined 7 days later.

The solid (0.5–5.0 mg) caused lacrimation at all doses, blepharitis that increased with dose and lasted up to a week, and chemosis at 5 mg, which was mild and lasted 3 days. Minimal iritis and keratitis, of 24 hours' duration, were seen in two of five animals receiving 5 mg. At concentrations of 1% and higher, CS in solution caused conjunctivitis and iritis, chemosis, keratitis, and corneal vascularization; the lesion was more severe and lasted longer with the higher doses. Histological examination indicated patchy denudation of corneal epithelium and a neutrophilic infiltration of the cornea.[17]

Reports of severe eye injuries from riot control agents have involved the agent CN. They are discussed below in the CN section.

Gastrointestinal Tract Disturbances

A handful of instances in which an individual ate CS are known. In all but two cases, children were the victims. Typically, they were playing in an old impact area on a military installation and came across some shells containing a powdery substance, which they ate. One adult ingestion was an attempt at suicide by an otherwise healthy young man; the other was an individual who ate a CS pellet (820 mg) after a friend told him it was a vitamin pill.[18]

The oral LD_{50} (dose that is lethal to 50% of the exposed population) of CS was found to be 143 mg/kg in the female rabbit, the most sensitive of three species studied (the rat, about 1,300 mg/kg; the guinea pig, 212 mg/kg; and the male rabbit, 231 mg/kg).[8] The animals that died had multiple, extensive hemorrhagic erosions of the gastric mucosa, with perforation of the wall, and a few had increased peritoneal fluid. In those surviving for several days, intraabdominal adhesions were found. After male rats and female guinea pigs received 0.5 LD_{50} of CS by stomach tube, and male rabbits received 0.3 LD_{50} by this route, the incidence of wet or runny stools was no greater than that for the control vehicle, polyethylene glycol 300 (PEG300).[19] The investigators concluded that diarrhea is not an effect of ingested CS. They also suggested that rioters would not have diarrhea from CS exposure, since they would be unlikely to swallow this much, but that an intensely emotional experience such as being in a riot may itself be a cause of disturbed bowel function. In another study,[20] the oral LD_{50}

varied widely in rats (178–358 mg/kg), depending on the solvent used. After death, moderate to severe gastroenteritis was noted on gross examination.

No deaths or severe complications in humans from ingestion of CS are known. The young man mentioned above who had attempted suicide by CS ingestion was given large amounts of what were described as "saline cathartics" and over the next 24 hours had repeated episodes of severe abdominal cramps and diarrhea; whether these symptoms were due to the illness or the treatment is unknown. A surgical team examined the patient early and stood by during the acute phase. The patient recovered uneventfully. The adult who ate a CS pellet was given liquid antiacid and viscous lidocaine orally and droperidol intravenously. He vomited twice, had six voluminous watery bowel movements without blood, and otherwise recovered uneventfully. Blood cyanide was less than 1 μg/dL 18 hours after ingestion (see section on metabolism).[18]

Metabolic Effects

Both in vivo and, in water, in vitro, CS (*o*-chlorobenzylidene malononitrile) is hydrolyzed to 2-chlorobenzaldehyde and malononitrile. Malononitrile contains two cyanide moieties, and it is thought that at least one of these is liberated and attaches to sulfur via the enzyme rhodanese to form thiocyanate, which is excreted in the urine.

Some authors have suggested that cyanide contributes to mortality in CS-caused deaths.[21,22] In dogs given CS by the aerosol or intravenous routes, the plasma concentrations of thiocyanate increased over the following 24 to 48 hours, presumably because of transformation of the liberated cyanide to thiocyanate by combination with endogenous sulfur.[21] After CS was given intraperitoneally, the mortality was markedly decreased by the intravenous administration of thiosulfate, which may have provided additional sulfur for the transformation of cyanide to thiocyanate.[21] Also, after intravenous administration of CS or malononitrile, the signs and the times to death were similar (15–60 min), suggesting that both caused effects by the same mechanism.[22] In this report, the authors also noted the similarities of signs and times of death for these two compounds, compared with cyanide administered intravenously.

One author of the latter report, however, clearly notes in a later communication[23] that the mode and time of death differ depending on whether CS is administered by the intravenous route or by aerosol. As noted earlier in the discussion of respiratory effects for CS, animals exposed to far greater than the lethal C*t* do not die during exposure or immediately afterwards, but many die hours later, in contrast to the usually rapid death caused by cyanide. Moreover, the lung damage found on pathological examination is adequate to explain death.[8,9]

In addressing this issue, a British report[3] suggests that whereas cyanide might be a causative factor in the rapid deaths occurring after intravenous administration, it is not a factor in death after aerosol administration. If one were to absorb completely all the CS during a 1-minute exposure at 10 mg/m^3, and if both cyanides on the molecule were liberated—and evidence suggests that only one is liberated—the total amount of cyanide received would be equivalent to that received from two puffs of a cigarette.

Other Physiological Responses

When subjects were exposed to CS concentrations of 1 to 13 mg/m^3 daily for 10 days, their airway resistances, measured 2 to 4 minutes after the fourth and tenth exposures, were unchanged from the preexposure values.[4] Tidal volume, vital capacity, and peak flow in 36 subjects also were unchanged when they were measured immediately and 24 hours after exposure to CS.[5]

Heart rates of subjects were lower immediately after exposure compared with preexposure values.[4] Subjects entered a chamber of CS with masks on; immediately on removing their masks, their mean blood pressure increased by 20 mm Hg systolic and 11 mm Hg diastolic. After they had remained in the CS for 20 minutes, however, their blood pressures were comparable to the preexposure values.[4] The blood pressures of subjects drenched with dilute solutions of CS were transiently elevated to about 150/90 mm Hg.[24]

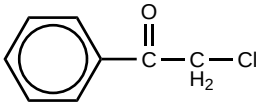
After daily exposures to CS for 10 days, seven subjects had no alterations in blood sodium, potassium, alkaline phosphatase, or bromsulfophthalein; one of the seven had an increase in thymol turbidity. No chest radiograph or urinary changes were seen.[4] In another study,[5] although significant changes were seen in some blood chemistries after exposure, all values were within the normal range.

Pregnant rats and rabbits were exposed to CS aerosols at concentrations of 6, 20, or 60 mg/m^3 for 5 minutes on days 6 to 15 and 6 to 18 of gestation, respectively. In addition, rats were given CS (20 mg/kg) intraperitoneally on days 6, 8, 10, 12, and 14 of gestation. No embryolethality or teratogenicity was evident.[25]

CS and some of its metabolites were found not to have mutagenic effects in the Ames *Salmonella*

typhimurium assay with microsome supplementation.[26] In addition, no mutagenic effects were found in assays for reverse mutations in *S typhimurium* after exposure to CS, in assays for sex-linked, recessive lethal mutations in sperm cells after *Drosophila* were fed CS, or in chromosomes of bone marrow erythrocytes of mice exposed to CS.[27] The authors of another study[28] of rats and *Salmonella* concluded that CS did not induce point mutations or carcinogenic processes mediated by DNA binding. However, CS did give a positive response in the forward mutation assay in mouse lymphoma cells.[29]

CN (1-CHLOROACETOPHENONE)



CN

Physical Characteristics

Like CS, the riot control agent known as CN (1-chloroacetophenone) is a solid or powder and can be disseminated as a smoke generated from a grenade or other device, or in powder or liquid formulations. Under the trade name Mace, it is in most devices sold for self-protection, although today it is commonly mixed with or is being replaced by capsaicin (pepper spray).

CN was first synthesized by Graebe in 1871 and was used in World War I. Before the late 1950s, it was the standard tear gas used by the military and law enforcement agencies.

The harassing concentration for CN is about 10 mg/m^3, compared with about 4 mg/m^3 for CS. It is more toxic than CS, and the human LCt_{50} (median lethal C*t*) has been estimated to be 7,000 $mg \cdot min/m^3$ for pure aerosol and 14,000 $mg \cdot min/m^3$ for a commercial grenade.[30]

Clinical Effects

In general, the clinical effects caused by CN are the same as those caused by CS. The harassing dose is higher and CN is more toxic and more likely to cause serious effects, particularly in skin and eyes (see below). Most effects from exposures to a low concentration will disappear within 20 to 30 minutes.

Respiratory Tract Effects

In studies parallel to those described above for CS, CN was found to be 3- to 10-fold more toxic (lower LCt_{50}) than CS in rats, rabbits, guinea pigs, and mice.[8] In addition, the pathological findings in the lungs were more severe, with more edema; patchy acute inflammatory cell infiltration of the trachea, bronchi, and bronchioles; and more evidence of early bronchopneumonia.

Dermatological Effects

A textbook published in 1925 states that CN in field concentrations does not damage human skin; however, the powder might produce burning: "slight rubefaction, and sometimes small vesicles appear."[31(p171)] Early cases of CN dermatitis—one of primary irritant dermatitis in a soldier and three in civilian employees who probably had allergic dermatitis from working around CN for years—were described several years later.[32]

A severe allergic reaction to CN developed after a 43-year-old military recruit went through the CN training chamber routine (ie, an individual spends 5 min in the chamber masked, then removes the mask and exits the chamber). Within 5 minutes after exiting, the patient complained of generalized itching, which became progressively worse over the following hours. Four hours after exiting, he had a diffuse and intense erythema over his entire body except his feet and the portion of his face covered by the mask. His temperature was 38.9°C (102°F) and rose to 39.4°C (103°F) the next day. By 48 hours after exposure, he had vesication and later developed severe subcutaneous edema that "strikingly altered the appearance of the face"[33(p1879)] and severe generalized itching. Over the next 4 days, the signs subsided, and desquamation, which was profuse at day 6, gradually decreased. The patient had developed itching during a tear gas exercise 17 years previously but had not been exposed in the interim.[33]

A police officer received an initial exposure to CN and 5 years later, on repeated exposure, developed recurrent attacks of what was probably allergic contact dermatitis. The source of the repeated exposures was unrecognized until he realized that he had been using outdated CN bombs for eradication of rodents on his property.[34]

CN (0.5 mg), when left in place for 60 minutes, caused irritation and erythema on the skin of all

humans tested in one study,[35] whereas CS caused no effects in amounts less than 20 mg. When the CN was moist, 0.5 mg caused vesication in most subjects, whereas vesication was not seen after exposure to 30 mg or less of CS.

In addition to being a more potent primary irritant on the skin than CS, CN is also a more potent skin sensitizer.[36] Several people developed allergic contact sensitivity to CN after patch testing.[37] Because of the high incidence of sensitization in test subjects, CN should be considered a potent allergic sensitizer, and those who are frequently exposed should be aware of the high likelihood of developing allergic dermatitis.[38]

Ophthalmological Effects

The irritation caused by CN in the eye signals avoidance and, by causing lacrimation and blepharospasm, initiates a defense mechanism. High concentrations of CN sprayed into the eyes from a distance have caused edema of the corneal epithelium and conjunctiva and many minute epithelial defects in the cornea.[39] Healing was rapid, however.

More lasting or permanent effects may occur when CN is released at close range (within a few meters), particularly if it is from a forceful blast from a cartridge, bomb, pistol, or spray. One study[40] based on case records from the files of the Armed Forces Institute of Pathology in Washington, D. C., reviewed eye injuries from tear gas; unfortunately, many of the histories were incomplete. In about half the cases, the injuries were self-inflicted and accidental; in the other half, the injuries were caused by a second person firing a weapon from close range with intent to injure the patient. In some instances, particles of agglomerated agent were driven into the eye tissues by the force of the blast; the authors of the study suggested that a chemical reaction caused damage over months or years. In other instances, the injury was probably caused by the blast or other foreign particles rather than by CN. The authors carefully pointed out that features of the weapon, such as the blast force, the propellant charge, the wadding, and the age of the cartridge (in older cartridges, the powder agglomerates and forms larger particles) should be considered in evaluating eye damage due to CN.

The author of another review[41] came to the same conclusion: the traumatic effect of the blast is a considerable factor, and one cannot always be sure that CN per se is the cause of permanent injury.

In a study[20] comparing the effects of CN and CS in the eyes of rabbits, CN at a concentration of 10% (wt/vol) caused iritis and conjunctivitis lasting longer than 7 days and corneal opacity lasting longer than 55 days. In contrast, CS, at the same concentration, caused moderate conjunctivitis but no iritis or corneal opacities; all eyes were normal at 7 days. Other evidence[30] indicates that when CN is applied directly to the eye in powder form or is sprayed at close range, a more severe reaction than that seen with CS may result.

Although permanent eye damage has been reported from the use of CN weapons at close range, separating the effects of the weapon from those of the compound is difficult. There is no evidence that CN at harassing or normal field concentrations causes permanent damage to the eye.

SEVERE MEDICAL COMPLICATIONS FROM THE USE OF CS AND CN

The indiscriminate use of large amounts of CN in confined spaces has caused injuries requiring medical attention and death. An incident of injury to an infant from CS has also been reported.

A 4-month-old infant was in a house into which police fired CS tear gas canisters for 2 to 3 hours to subdue a disturbed adult. Immediately on being removed from the house, the infant was taken to a hospital, where he was observed to have copious secretions of the nose and mouth and frequent sneezing and coughing. He required frequent suctioning to relieve upper airway obstruction. Physical examination was unremarkable except for the secretions, slight conjunctival injection, and rapid heart rate and respirations. On the second day, he had an episode of cyanosis, which cleared with suctioning. On examination, he was in respiratory distress with suprasternal retraction, wheezes, and rales bilaterally. The chest radiograph was clear. Antibiotics, high-dose steroids, and positive-pressure breathing were started. He slowly improved until the seventh hospital day, when his temperature rose to 40.4°C (104.4°F) and coughing increased. An infiltrate was noted on the chest radiograph. Physical findings were unremarkable except for coarse breath sounds throughout the lungs. He improved with further antibiotic and ventilatory therapy and was discharged on day 12, only to be readmitted on day 13 with an increasing cough and a progression of the infiltrate. With more antibiot-

ics and other therapy, he gradually recovered and was discharged after 28 days in the hospital.[42]

In a prison incident, 44 inmates were in a cell block sprayed with CN; 28 inmates later sought medical attention, and 8 were hospitalized. All eight complained of malaise, lethargy, and anorexia. Five had pharyngitis, three of whom developed pseudomembranous exudates several days later. Three also developed tracheobronchitis with purulent sputum, but no infiltrates on chest radiograph. Four patients had facial burns, and three had bullae on the legs; the most severely affected had first- and second-degree burns over 25% of his body. One patient was admitted 5 days after the incident with a papulovesicular rash of his face, scalp, and trunk, which had appeared 2 days earlier. Ten prisoners were treated as outpatients for first- and second-degree burns, and six had localized papulovesicular rashes. Ten had conjunctivitis with edema of the conjunctiva, and in some the eyelids were closed by the swelling, but no patient had corneal injuries or permanent eye damage. The patients with laryngotracheobronchitis were given bronchodilators, postural drainage, and positive-pressure exercises. Two were given short-term, high-dose steroids, but none received antibiotics. One required bronchodilator therapy 3 months later, but the others made prompt recoveries.[43]

The skin lesions were treated with debridement and applications of silver sulfadiazine and, in some cases, with topical steroids and antihistamines. Skin color was almost normal 3 months later. Topical steroids caused the conjunctival edema to begin to resolve in 48 hours. The only estimate of the amount of CN used was obtained from the prisoners, each of whom claimed to have been sprayed multiple times. Although the first- and third-floor windows were open, the exhaust system was off during the incident.[43]

In another prison incident, the windows and doors were closed and ventilation was off during what was described as a "prolonged gassing" of inmates confined to individual cells. It was later estimated that the incident lasted 110 minutes. Among the dispensers used were at least six thermal grenades of CN, fourteen 100-g projectiles of CN, and more than 500 mL of an 8% CS solution. Using only the amount in the CN projectiles, the authors of the report calculated that the prisoners were exposed to a Ct of 41,000 mg•min/m^3. The total number of prisoners exposed was not noted. Afterward, some had coughing with varying degrees of illness, and at least three received medical treatment (the authors carefully pointed out that they were unable to obtain details).[44]

A prisoner was found dead under his bunk 46 hours later. Other prisoners reported that he had had "red eyes," had vomited "bloody" material, and had sought medical attention on several occasions. On autopsy, he was noted to have rigor mortis, cyanosis of the face and head, and no evidence of physical injury. His lungs had subpleural petechiae, hyperemia, mild edema, and patchy areas of consolidation; microscopic examination showed bronchopneumonia clustered around exudate-filled bronchioles. His larynx and tracheobronchial tree were lined with an exudative pseudomembrane; microscopic examination showed this was a fibrin-rich exudate containing polymorphonuclear leukocytes and their degenerating forms. There was no evidence of gastrointestinal hemorrhage; other organs had passive hyperemia.[44]

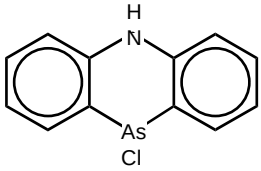
Another individual had an altercation with the police and locked himself into a room in his house. A single CN grenade (128 g) was thrown into the room (approximately 27 m^3), where the patient remained for 30 more minutes (128,000 mg • 30 min ÷ 27 m^3 provides an estimated Ct of about 142,500 mg•min/m^3, or an exposure 10-fold higher than the estimated lethal Ct_{50}).[45]

On admission to the hospital, his respirations were 24 per minute, his conjunctiva were suffused, his pupils were small and unreactive, mucoid discharge from his nose and mouth was abundant, his lungs were clear, and an occasional premature ventricular contraction was evident on the electrocardiogram. He remained "in a semicomatose condition for approximately 12 hours and then suddenly developed pulmonary edema and died."[45(p375)] Relevant findings on autopsy included cyanosis, frothy fluid in the mouth and nose, acute necrosis of the mucosa of the respiratory tree with pseudomembrane formation, desquamation of the lining of the bronchioles with edema and inflammation of the walls, and a protein-rich fluid in most of the alveolar spaces. Foci of early bronchopneumonia were present.[45]

Information on three other cases of death from CN, which the authors obtained from other medical examiners, are summarized in the same report.[45] Details were scanty, but the autopsy findings were similar; in each case, the individual was confined in a relatively small space. Exposure was for 10 minutes in one instance and for hours in the others (details of exposure were unknown).

OTHER RIOT CONTROL COMPOUNDS

DM (Diphenylaminearsine)



DM

The riot control agent known as DM (diphenylaminearsine) is one of a group of compounds that are known as vomiting agents. The others, which are of much less military importance, are the agents DA (diphenylchlorarsine) and DC (diphenylcyanoarsine). DM was first synthesized by the German chemist Wieland in 1915 and, independently, by the U.S. chemist Adams in 1918. DM is also known as adamsite.

DM is a yellow-green, odorless, crystalline substance that is not very volatile. It is insoluble in water and relatively insoluble in organic solvents. Its primary action is on the upper respiratory tract, causing irritation of the nasal mucosa and nasal sinuses, burning in the throat, tightness and pain in the chest, and uncontrollable coughing and sneezing. It also causes eye irritation and burning, however, with tearing, blepharospasm, and injected conjunctiva.

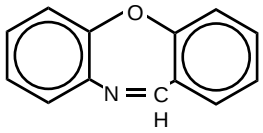
DM is more toxic than other riot control agents; the LCt_{50} for humans has been estimated to be 11,000 mg•min/m^3.[46] The amount that is intolerable for humans has been estimated by some to be 22 mg•min/m^3 and by others to be 150 mg•min/m^3.[46] The threshold for irritation in humans is about 1 mg/m^3, but men have tolerated C*t* exposures of 100 to 150 mg•min/m^3.

Two characteristics make this class of compounds unique among the riot control agents. The first is that the effects do not appear immediately on exposure or seconds afterwards, but several minutes later. In the absence of symptoms, a soldier will not mask immediately; by the time he masks, he will have absorbed a significant amount. The effects may then cause him to unmask.

The second characteristic of these compounds is that there may be more prolonged systemic effects, such as headache, mental depression, chills, nausea, abdominal cramps, vomiting, and diarrhea, which last for several hours after exposure. DM and related compounds are known as vomiting agents, but the incidence of vomiting and the amount of compound necessary to cause it are not known with certainty. In studies dating from 1922 to 1958,[46] humans were exposed to C*t*s ranging from 4.6 to 144 mg•min/m^3; nausea was noted in fewer than 10% of the subjects. Because of the lack of data, the C*t* necessary to cause nausea and vomiting has not been established,[46] but has been estimated to be about 370 mg•min/m^3.[24]

One death has been reported[46] from DM inhalation (the information on this fatality is incomplete). A DM generator was operated in a barrack, exposing 22 sleeping men. The estimated concentration was 1,130 to 2,260 mg/m^3, and the duration of exposure was estimated to be 5 minutes (by one source) or 30 minutes (by a second source). For a 5-minute exposure, the estimated C*t* would be 5,650 to 11,300 mg•min/m^3; for a 30-minute exposure, 33,900 to 67,800 mg•min/m^3. One individual died; the postmortem findings were severe airway and lung damage, similar to those seen after death from CN. Another source[47] reported severe pulmonary injury and death after accidental exposure to high concentrations of DM in confined spaces, but no details were given.

CR (Dibenz*(b,f)*-1:4-oxazepine)



CR

The riot control agent known as CR (dibenz*(b,f)*-1:4-oxazepine) is a relatively new compound, first synthesized in 1962 by Higginbottom and Suschitzkey. CR is more potent and less toxic than CS. Because of the low vapor pressure of CR solution, no respiratory tract effects are anticipated from its use. The LCt_{50} for animals exposed to grenade-generated smokes was found to be 167,500 mg•min/m^3. The estimated LCt_{50} for humans is probably higher than 100,000 mg•min/m^3.[24]

CR is sparingly soluble in water, and a cosolvent (PEG300 is frequently used) is necessary when it is

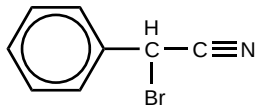
dispersed in solution. Since CR does not degrade in water, it resists weathering and persists in the environment.

In humans, the effects caused by CR are qualitatively similar to those caused by CS, but there is an approximately 5-fold difference in potency. A splash of a solution in the range of 0.01% to 0.1% causes immediate eye pain, blepharospasm, and lacrimation, which persist for 15 to 30 minutes, and conjunctival injection and minimal edema of lid margins, which last for 3 to 6 hours. A solution splashed in the mouth causes burning of the tongue and palate and salivation for 5 to 10 minutes. If a splash enters the nose, it causes irritation and rhinorrhea. Skin exposure causes burning within a few minutes, which persists for 15 to 30 minutes, and an erythema lasting for 1 to 2 hours. A blood pressure increase may accompany the subjective discomfort; this is thought to be caused by the stress of the irritation, since the amount of CR that could be absorbed is much too small to cause a pharmacological effect.[24]

A transient erythema (1–2 h) occurs, but CR does not induce inflammatory cell infiltration, vesication, or contact sensitization, and it does not delay the healing of skin injuries.[24,48] The potential for eye damage is also significantly less than it is from CS or CN.[24] CR was neither teratogenic nor embryolethal in one study[49] when given as an aerosol or by gavage.

Compared with other riot control agents, CR is relatively new; no data from its use exist. Experimental studies indicate that its effects are similar to those of CS except that it causes almost no effects in the lower airways and lungs. It is much more potent than CS—a smaller concentration is needed to cause effects—and it appears to be much safer, as judged from the higher LCt_{50} and the lack of persistent skin and eye effects.

CA (Bromobenzylcyanide)



CA

The riot control agent known as CA (bromobenzylcyanide) was the last irritating agent introduced by the Allies in World War I, and it was the most potent. It corrodes iron and steel, is not chemically stable in storage, and is sensitive to heat, all characteristics that made it unsuitable for storage and use in artillery shells.[50]

CA irritates the eyes and causes lacrimation at concentrations of 0.15 and 0.3 mg/m^3; the LCt_{50} was estimated to be 27,000 $mg \cdot min/m^3$.[50] More recent studies indicate that the estimated LCt_{50} for humans is 11,000 $mg \cdot min/m^3$,[51] indicating that it is among the more toxic riot control agents. The health effects caused by CA are very similar to those caused by CS and CN.

CA is rarely used and is a relatively unimportant agent of this class. The compound is included here primarily because it is discussed in *Treatment of Chemical Agent Casualties and Conventional Military Chemical Casualties*,[52–54] field manuals published by the Department of Defense for use by the U.S. Army, Navy, and Air Force.

MEDICAL CARE

The effects from riot control agents are usually self-limiting, and medical attention is usually not required. Exiting the contaminated area should bring some measure of relief in 15 to 30 minutes or sooner. In rare circumstances, complications may occur on the skin, in the eyes, or in the airways.

Decontamination

The use of water on the skin may result in transient worsening of the burning sensation. Soap and water may be more effective but may also cause a momentary increase in the symptoms. CS rapidly hydrolyzes in an alkaline solution; a solution containing 6% sodium bicarbonate, 3% sodium carbonate, and 1% benzalkonium chloride was found to bring prompt relief of symptoms and to hydrolyze the agent.[13] No form of hypochlorite should be used.

Skin

For dermatitis, a topical steroid preparation (eg, triamcinolone acetonide, fluocinolone acetonide, flurandrenolone, or betamethasone-17-valerate) is the principal therapeutic agent. Oozing lesions should be treated with wet dressings (moistened with fluids such as 1:40 Burow's solution). Appropriate antibiotics should be given for secondary infection, and oral antihistamines for itching.[13] Vesicating lesions have been successfully treated with compresses of a cold silver nitrate solution (1:1,000)

for 1 hour, applied six times daily.[11] One person with severe lesions and marked discomfort was given a short course of an oral steroid. An antibiotic ointment was applied locally, but systemic antibiotics were not used.[11]

Eye

A local anesthetic might be applied once for severe pain, but continued use should be restricted. The eye should be thoroughly flushed to remove any particles of the agent. If the lesion is severe, the patient should be sent to an ophthalmologist.

Respiratory Tract

Usually, the cough, chest discomfort, and mild dyspnea are gone 30 minutes after exposure to clean air. However, both the animal data (detailed in the section on CS) and the clinical experience with the infant exposed to CS suggest that severe respiratory effects may not become manifest until 12 to 24 hours after exposure. An individual who has prolonged dyspnea or objective signs should be hospitalized under careful observation. Further care should be as described in Chapter 9, Toxic Inhalational Injury. Although people with chronic bronchitis have been exposed to riot control agents without untoward effects, any underlying lung disease (eg, asthma, which affects one person in six in the general, or the military, population) might be exacerbated by exposure to CS.[3]

Cardiovascular System

Transient hypertension has been noted after exposure to riot control agents, primarily because of the anxiety or pain of exposure rather than a pharmacological effect of the compound. Whatever the cause, adverse effects may be seen in individuals with hypertension, cardiovascular disease, or an aneurysm.

FUTURE USE

More research is needed to illuminate the full health consequences of riot control agents, as one report[55] has suggested. Information gaps in this chapter indicate areas that might fruitfully be explored, although funding for such research is problematic. The limited resources of the military program in chemical defense are probably more wisely spent on investigating better defense against and medical care for victims of agents that cause more severe consequences and are more likely to be used on a battlefield. Law enforcement agencies generally have few funds for these purposes. Manufacturers probably do not have a large interest in this topic; it is unlikely that their profits from these compounds are large enough to support such an effort. Federal medical funding is generally concerned with more serious diseases affecting larger segments of the population.

Other concerns discussed in the report[55] were the "pattern of use" of these compounds. Are there circumstances in which the use of riot control agents can, or cannot, be condoned? The "pattern of use" might be difficult to regulate, particularly in the areas and under the circumstances in which the use of CS or CN has apparently been abused (eg, the West Bank and the Gaza Strip in the Middle East, and Seoul, South Korea). Public opinion and the Geneva Protocol did not dissuade Iraq from using several types of chemical weapons in the conflict with Iran, or prevent Libya from constructing a large manufacturing facility at Rabta, apparently for the manufacture of chemical weapons. Despite the concern about the loss of innocent lives and injury among innocent bystanders, there is serious doubt that a prohibition of the use of riot control agents would be effective.

While it is true that in some instances dialogue and negotiation should precede the use of riot control agents, one wonders how this suggestion might have been received by the desperate refugees. Although CS allegedly caused injury, the amount of injury was probably small compared to what might have been inflicted if CS had not been available and more extreme measures had been used. Possibly, the use of CS is sometimes the most benign solution in ugly and dangerous circumstances.

SUMMARY

Riot control agents are intended to harass or to cause temporary incapacitation. Their intended target might be the foe in an armed conflict—with the limitations outlined above—or rioters in a civil disturbance.

Much evidence suggests that riot control agents are safe if they are used as intended and if the response is as intended. When they are not used as intended, and the response is not as intended, how-

ever, there may be devastating consequences (eg, the deaths of the Branch Davidians at Waco, Tex.). Almost all of the reported adverse effects have resulted from indiscriminate use of weapons containing riot control agents or from resistance to the effects of the compounds, which increases the amount of exposure. Sometimes injury results from the effects of the delivery system of the weapon rather than from the compound; these two sources of injury should not be confused.

Indiscriminate or uncontrolled use of CS, or any riot control compound, is obviously not desired, nor is it necessary in circumstances in which a better, less drastic solution is possible. But the use of CS or CN might be more benign than the use of more deadly alternatives in desperate circumstances. As the data clearly suggest, CS is a relatively safe compound when used as intended.

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