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CASE REPORT

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Death Resulting from Lacrimatory Agents

Currently used riot control agents are primarily intended to incapacitate an individual without causing him illness or permanent bodily harm. The compounds most commonly employed as lacrimatory agents or "tear gases" are chloroacetophenone (CN) and *ortho*-chlorobenzalmalononitrile (CS). Some studies indicate that CS is less likely to produce harmful effects than CN; CN has been implicated in at least five deaths resulting from its use. The case reported is one of a fatality following the apparently indiscriminate use of CN and CS.

Case Report

A disturbance in the maximum security building at the Oklahoma State Penitentiary at McAlester resulted in the prolonged gassing of the inmates who were confined to individual cells. The ventilating fans of the building were turned off and the windows and doors were closed during the incident. For various sociopolitical reasons not germane to this report, the precise amount of CN and CS used, the duration of gassing, and many other details of the situation are either uncertain or unascertainable. The most reasonable reconstruction of events from subsequent courtroom testimony and available records reveals that the actual gassing lasted approximately 110 min. A minimum of six heat-type grenades (size and concentration unknown) of CN, fourteen 37-mm, 100-g projectiles of CN, and in excess of 0.4 litres (one pint) of 8% CS gas in two 4- to 5-min bursts via "pepper-fogger" were discharged during this period. Additional smaller amounts of these agents were dispersed in the same area the following day. There were conflicting statements in all particulars of this incident.

One prisoner in the area, a 33-year-old white male, was found dead under his bunk approximately 46 h after the initial gassing. There are no official records indicating anything unusual in his appearance or actions in the intervening time. Prisoners who were present, however, report that the deceased had "red eyes" and vomited material they considered "bloody." He was not seen by a physician after the gassing, although he is reported by fellow inmates to have requested medical attention on several occasions. Other prisoners in the area are reported to have developed coughing with varying degrees of illness. At least three were treated for their symptoms. There is no certainty of any of the details since reconstruction of the events by the penal authorities was more political than scientific.

Necropsy examination revealed a normally developed white male with complete rigor mortis and legs fixed in a "kneeling" position. Cyanosis of the face and head and an esti-

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mated two-day growth of beard were present. There was no evidence of physical injury, either internally or externally.

Subpleural petechiae were present in both lungs (right, 780 g and left, 850 g), which were hyperemic with mild edema. The entire larynx and tracheobronchial tree were covered with gray-white material, characteristic of exudate with pseudomembrane formation. Patchy, ill-defined small areas of consolidation were evident in the lungs.

There was no evidence of gastrointestinal hemorrhage. A 2- by 2-mm subendocardial hemorrhage was present on the left wall of the interventricular septum. All organs were characterized by passive hyperemia.

Microscopic examination (Fig. 1) revealed extensive necrosis and ulceration of the mucosal epithelium of larynx, trachea, and bronchi and its replacement by a pseudomembrane of fibrin-rich exudate containing polymorphonuclear leukocytes and their degenerating forms. Scattered areas of bronchopneumonia were present, all clustered about exudate-filled bronchioles. Edema and minimal intraalveolar hemorrhage were observed.

The only other significant findings were a mild degree of hepatic fatty metamorphosis, cerebral edema, and degeneration of neurons in Ammon's horn. Laboratory analyses were negative for CN, ethanol, heavy metals, and multiple other drugs and poisons. The tissues and materials analyzed included gastric wall and content, blood, and liver.

The cause of death was deemed acute necrotizing laryngotracheobronchitis, chemical, resulting from exposure to lacrimatory agents. The failure to demonstrate CN in the autopsy specimens was expected because of the period of survival after exposure.

The opinion concerning cause of death was not unanimous. At a subsequent trial in the U.S. Federal Court in Muskogee the prison physician, a nonpathologist who was not a party to the necropsy, gave testimony which indicated the deceased had been suffocated by person(s) unknown and that the death was unrelated to the gassing.[3]

Discussion

The chemical nature, pathophysiology, and metabolism of CN and CS are well recognized and documented [*1-7*]. Clinical manifestations resulting from the application of these compounds in the suggested concentrations for incapacitation include lacrimation, blepharospasm, transient conjunctivitis, rhinorrhea, sternutation, dyspnea, coughing, nausea, vomiting, diarrhea [*6*], and dermal irritation [*2,8*]. Less frequently recorded effects are cephalgia, altered taste preferences, eructation, dysuria, and lethargy [*6*].

Five fatalities occurring from 4½ h up to 4 days after exposure to CN have been reported [*9*]. Pertinent postmortem findings they had in common were intense cervicofacial cyanosis, pulmonary edema, focal intraalveolar hemorrhage, necrosis of the respiratory mucosa with pseudomembrane formation, early bronchopneumonia, serosal petechiae, cerebral edema, and hepatic fatty metamorphosis.

It is well accepted that with almost any chemical agent the effects produced are strongly dependent on the concentration of the compound and the duration of exposure. The dosage of these compounds may be calculated by a formula used by Cucinell et al [*4*], in which *ct* (dosage in $mg \cdot min/m^3$) equals gas concentration (mg/m^3) multiplied by length of exposure (in minutes). For example, a *ct* value of approximately 8 for CS gas is considered intolerable for man [*4*].

The dimensions of the building in this instance were approximately 14 by 5 by 49 m, roughly equal to a volume of 3000 m^3. If one considers only the 100-g projectiles of CN that were discharged and assumes that there was uniform distribution solely of this agent throughout the building, one may calculate a *ct* of 41 000 employing the formula cited above. The actual *ct* would, obviously, have been much higher than this if the total amount

[3]William Gardner, U.S. Department of Justice, Washington, D.C., personal communication.

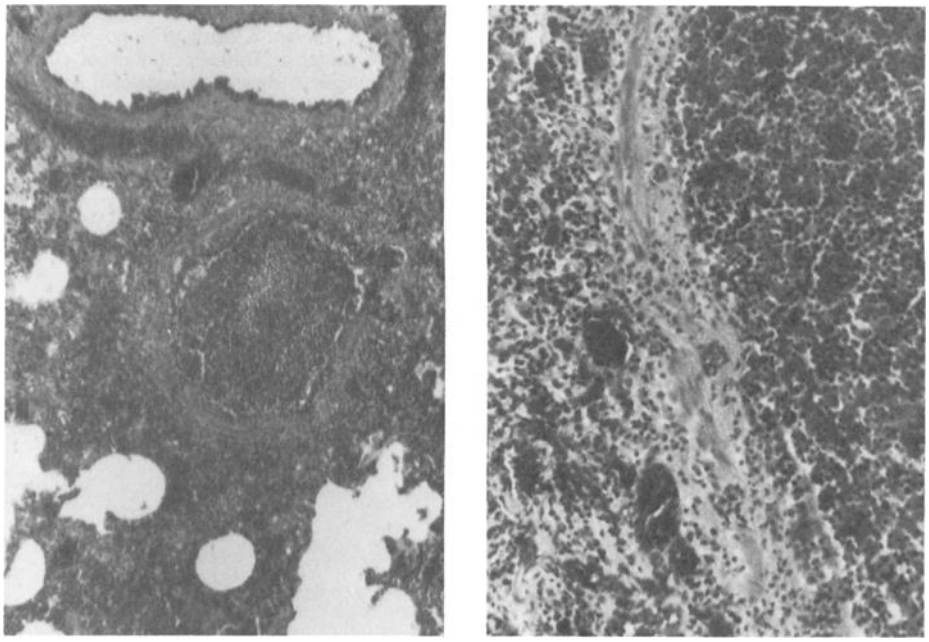

FIG. 1—(left) *A small bronchus is occluded by exudate and bronchopneumonia is developing.* (right) *A fibrin-rich exudate of polymorphonuclear leukocytes and their degenerating forms replaces the bronchial epithelium.*

of CN released could be determined and taken into account. The distribution about the building would probably not have been uniform because of the specific gravity of the gas. It may be assumed, therefore, that some of the inmates received much higher concentrations than others.

In view of the experience of others, as cited above, this death most likely was the result of exposure to CN. The role of CS in the production of the pathologic changes is problematic. It may have potentiated the effects of the CN or played no role at all. It would appear improbable that CS alone was responsible for the changes observed in this case, although the authors see no reason to believe that it alone could *not* produce similar changes if an individual was exposed to a high concentration of the material for a sufficient time.

Summary

A case of death resulting from the indiscriminate use of the tear gas compounds CN and CS has been presented and briefly discussed.

Acknowledgment

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References

[*1*] Ballantyne, B. and Callaway, S., "Inhalation Toxicology and Pathology of Animals Exposed to *O*-Chlorobenzylidene Malononitrile (CS)," *Medicine, Science and the Law,* Vol. 12, No. 1, Jan. 1972, pp. 43-64.

[2] Ballantyne, B. and Johnston, W. G., "*O*-Chlorobenzylidene Malononitrile (CS) and the Healing of Cutaneous Injuries," *Medicine, Science and the Law,* Vol. 14, No. 2, April 1974, pp. 93-97.
[3] Ballantyne, B. and Swanston, D. W., "The Irritant Potential of Dilute Solutions of *Ortho*-Chlorobenzylidene Malononitrile (CS) on the Eye and Tongue," *Acta Pharmacologia et Toxicologica,* Vol. 32, No. 3, 1973, pp. 266-277.
[4] Cucinell, S. A., Swentzel, K. C., Biskup, R., Snodgrass, H., Lovre, S., and Stark, W., "Biochemical Interactions and Metabolic Fate of Riot Control Agents," *Federation Proceedings. Federation of American Societies for Experimental Biology,* Vol. 30, No. 1, Jan.-Feb. 1971, pp. 86-91.
[5] Gaskins, J. R., Hehir, R. M., and McCaulley, D. F., "Lacriminating Agents (CS and CN) in Rats and Rabbits," *Archives of Environmental Health,* Vol. 24, No. 6, June 1972, pp. 449-453.
[6] Punte, C. L., Owens, E. J., and Gutentag, P. J., "Exposures to *Ortho*-Chlorobenzylidene Malononitrile," *Archives of Environmental Health,* Vol. 6, March 1963, pp. 72-80.
[7] Punte, C. L., Weimer, J. T., and Ballard, T. A., "Toxicologic Studies on *O*-Chlorobenzylidene Malononitrile," *Toxicology and Applied Pharmacology,* Vol. 4, Sept. 1962, pp. 656-662.
[8] Shmunes, E. and Taylor, J. S., "Industrial Contact Dermatitis," *Archives of Dermatology,* Vol. 107, No. 2, Feb. 1973, pp. 212-216.
[9] Stein, A. A. and Kirwan, W. E., "Chloracetophenone (Tear Gas) Poisoning: A Clinico-Pathologic Report," *Journal of Forensic Sciences,* Vol. 9, No. 3, July 1964, pp. 374-382.

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