The Prehospital Level: The doctrine, Standing Operating Procedures (SOP), training and equipment at the prehospital level, has been finalized and joint exercises are being conducted regularly with MDA ambulance teams, police forces, and fire services.

MDA HAZMAT Response Teams: These teams are based on MDA's regular ambulance teams (MICU and regular ambulances) in 10 out of 40 MDA stations throughout the country. These HAZMAT response stations were chosen in locations in which the main chemical industry or storage and shipment facilities are located. The stations have been equipped with two sets of personal protective suits each, of the "RESPONDER 0011" type. We are now in the process of replacing these suits with the more heavy "BUTYLIC" suit, produced by the TRALLEBORG Company. Each suit includes a SCUBA respirator, BD-88. In addition, five light, disposable suits are available in each station for decontamination teams, with gas masks and filters. The medical equipment contained in a first-aid kit includes an "Ambu" manual respirator with filter, an aspirator (foot-pump type), dressing material, a rubber tourniquet, and a cervical collar. All MDA personnel in the HAZMAT stations, including paramedics, ambulance drivers, and adult volunteers, undergo an annual refresher course and an exercise with police and fire services.

Hospitalization: All HAZMAT victims will be hospitalized for observation and medical treatment. Depending on the distance from the incident site to the hospital, the number of casualties and available means for evacuations, an undefined number of victims may bypass the decontamination station and MDA's first-aid points on the scene, and reach the hospitals without decontamination or receiving first aid on-site. Hospitals have to be prepared (as they were during the Gulf War) to receive HAZMAT casualties on very short notice, or without any advance warning. This is a very demanding situation for any hospital and emergency department, even in conventional mass-casualty situations. It requires planning, preparation of a decontamination facility at the entrance to the emergency department, specific medical equipment, drugs, and trained medical teams.

III.4
Decontamination of Victims

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Individuals exposed to liquid toxic chemicals must be decontaminated as soon as possible to: 1) save the life of the exposed victim; 2) make transportation and treatment possible; 3) avoid secondary contamination of rescue services and medical personnel; and 4) avoid secondary contamination of transportation vehicles and hospital.

It is extremely important to stop the exposure of the victims to the toxic chemicals and to undress the victim. Decontamination then can be performed by means of: a) dry methods, or b) wet methods.

In peacetime chemical disasters, where resources and supplies are plentiful enough to meet the requirements, the situation is more favorable than in wartime chemical disasters. Mass casualties, lack of resources, use of chemical warfare agents, combined chemical lesions and trauma, and the need for specific decontaminating agents are some of the problems in war. Rapid and accurate detection of chemical exposure has yet to be developed.

Therefore, safe and effective routines for decontamination and treatment have to be developed to save the patient without contaminating medical personnel or the hospital.

III.5
Chemical Disasters: Lung Injuries

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Inhalation or aspiration of various chemical compounds can cause severe lung damage—either acute and fullminant or more protracted—sometimes characterized by a biphasic progress with a period of apparent recovery. The distribution of the damage within the lung is dependent on the solubility in water of the compound; the more water soluble it is (e.g., formaldehyde, ammonia), the higher up in the airway tree (larynx, trachea) the damage will be located. If the compound is less soluble in water (e.g., chlorine, isocyanates), small airways and alveoli may be damaged.

In upper-airway involvement, the major findings are burns, edema, coughs, bronchospasms, and breathlessness. With severe exposure, laryngeal and epiglottic edema may develop, and, in the worse cases, laryngospasm. Respiratory and circulatory collapse may occur by reflex mechanisms.

Damage to the peripheral airways and the alveoli causes capillary leakage and pulmonary edema. The chemical irritation also may cause a pneumonitis with leaking airways and activation of inflammatory cells. Finally, many inhaled gases and other compounds can cause systemic effects with little direct damage to the lung tissue (e.g., carbon monoxide, alcohol).

III.6
Ventilatory Support in Acute Severe Lung Injury

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The acute pathological response of the respiratory tract to chemical irritants is characterized by inflammatory changes in the airway and by alveolar edema. The pathophysiologic correlate is an imbalance of ventilation to perfusion (V_A/Q mismatch). Clinically, cough, stridor, hypoxemia, and respiratory distress are common symptoms.

Treatment should be directed against the airway inflammatory response with endothelial injury and airway obstruction through the administration of appropriate pharmacological agents. Mild to moderate hypoxemia may be counteracted with supplemental oxygen administered via nasal prongs or by face mask. More pronounced hypoxemia usually requires measures that counteract

the V_A/Q inequalities. Continuous positive airway pressure delivered through a face mask increases end-expiratory lung volume. Gas delivery to well-perfused, but atelectatic or poorly ventilated lung areas, is improved, and arterial oxygen saturation increases. However, venous return to the heart may be impaired, and cardiac output may fall, as a result of the increase in intrathoracic pressure.

Endotracheal intubation and mechanical ventilation is indicated to protect the airway when consciousness is decreased or when severe respiratory distress or acute respiratory acidosis is present. Controlled studies that specifically compare different ventilatory techniques in ventilatory failure inhalation injury are lacking. Ventilator settings are chosen so that SaO_2 exceeds 90% and so that pH is within an acceptable range. Positive end-expiratory pressure is used to improve oxygenation and reduce the need for fractional concentrations of inspired oxygen (F_IO_2) of more than 0.6. If there is significant airway obstruction, inspiratory time may have to be shortened to allow for complete expiration.

It is important to recognize that mechanical ventilation is a supportive measure, and not curative in itself. Moreover, mechanical ventilation is associated with many side effects. It can be shown experimentally that ventilation with high-tidal volumes or high-peak inspiratory pressure induces lung injury. It is essential to minimize the risks of such complications during mechanical ventilation. Close monitoring of hemodynamic and pulmonary function identifies adverse effects, such as excessive alveolar pressure, dynamic hyperinflation, and compromised cardiac function, and permits prompt action to be taken against these complications.

III.7
Pharmacological Treatment of Lung Injuries After Exposure to Irritant Gases

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Management of victims exposed to irritant gases (e.g., ammonia, chlorine, nitrogen oxides, phosgene, sulfur dioxide) includes symptomatic therapy (including oxygen), assisted or controlled ventilation, and bronchodilatory therapy. B_2-adrenergic agonists should be given primarily via inhalation. Administration of xanthine derivates also may be indicated.

The use of corticosteroids in these situations is controversial. There are animal studies indicating both the positive and negative effects of corticosteroids. However, clinical experience suggests that the use of topical or systemic corticosteroid therapy may be helpful in relieving symptoms of irritation, although controlled clinical trials are lacking. The damage seen after exposure to irritant gases can be described as decimation of the epithelial layer, alveolar damage, endothelial damage, alveolar-capillary congestion, inflammatory reaction, capillary hyperpermeability, and eosinophilic hyaline membrane formation. Some of these effects are, at least theoretically, possible to treat with corticosteroids. Corticosteroids affect every stage of inflammatory and immunological reactivity, influence movement and distribution of lymphocytes, neutrophils, and eosinophils. They also decrease the accumulation of these cells at inflammatory sites, and inhibit the leakage of fluid and cells from capillary beds. Furthermore, they stimulate the synthesis of lipomodulin, which inhibits the activity of phospholipase A_2, and decreases neurophil and macrophage chemotaxis, histamine release from mast cells and basophils, and bronchospasm and inflammatory oedema mediated by leukotrienes. It seems logical that at least topical (inhalation) corticosteroid therapy might be indicated in victims with signs of respiratory tract irritation, or in cases of heavy exposure to phosgene and nitrogen oxides without any immediate symptoms.

In cases with concomitant thermal or corrosive burns, systemic corticosteroid therapy might be contraindicated.

III.8
Do You Know What's Happening Inside?

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An illegal chemical industry poured a cyanide compound into the sewer system. The hydrogen cyanide passed into the collector sewer, which had a pH = 3.1. Because of the value, it formed a hydrogen cyanide bubble.

The victim's house did not have a gas trap connected to the rain-water sewer, and the hydrogen cyanide entered through this route causing the immediate death of the four occupants of the building.

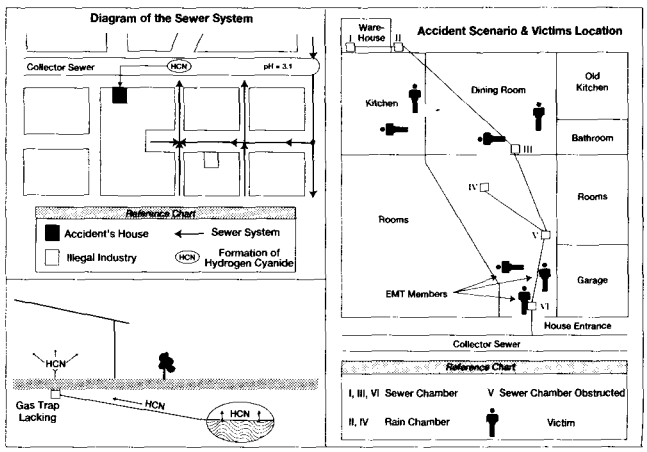
A neighbor who always visited the victim's house in the afternoon notified the emergency medical services because she knew there were people inside, yet nobody responded. A medical provider forced the street door open, and then fell into a coma state. Two minutes later, a fire brigade arrived. A firefighter was poisoned when he tried to revive the victims by mouth-to-mouth resuscitation.

Immediately, the officer of the brigade ordered the isolation of the area until the situation was controlled.

The zonal hospital reported nine cases of intoxication about two hours after the accident had occurred.

Effects on Population:

Deaths	7 people
Poisoned	10 people



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