9 Biological Effects in Humans, Initial Management and Role and Responsibility of Poison Centres in Chemical Accidents

Per Kulling

9.1 INTRODUCTION

Acute exposure on a large scale to hazardous chemicals might happen in accidents at plants or during transport on vehicles, trains and ships. Apart from obvious direct acute effects one must always have in mind the possibilities of symptoms developing days, weeks, months or perhaps even years after the acute exposure. An example of a major accident where acute symptoms were relatively minor is the release of chlorinated phenols from a plant in Seveso in Italy in 1976 (Anonymous, 1981; Coulson and Pocchiari, 1983). Two to four days after the release small animals like chickens and rabbits died, after 3–7 days children who had been playing outdoors in the gas cloud developed mild chemical skin burns, and after 2 weeks to 2 months several people presented with typical skin lesions, chloracne, and slight liver damage. Suspicion was raised of risk for teratogenicity and cancerogenicity; however, despite advanced plans for close follow-up studies concerning those possible effects, no definite conclusion could be drawn. It must be emphasized that these types of studies must always be undertaken to reveal possible late effects or sequelae of an accident.

In the following, mainly acute effects of a chemical accident will be discussed. Repeated low dose occupational exposure will not be discussed. Aspects of the initial management will be given, especially initial measures of importance in minimizing toxic effects on man at the site of the accident. Finally, some comments will be given on different activities that might improve preparedness in cases of accidents with special reference to the role and responsibility of poison centres.

Much of the contents in the following is based upon extensive documentation. Instead of giving all the references to generally agreed principles in clinical toxicology, the list of references includes some major toxicology textbooks.
Table 9.1 Concentration–effect relationships of gaseous ammonia

<table>
<thead>
<tr>
<th>Concentration (ppm)</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Odour threshold</td>
</tr>
<tr>
<td>25</td>
<td>TLV (Sweden)</td>
</tr>
<tr>
<td>50</td>
<td>TLV (ceiling, Sweden)</td>
</tr>
<tr>
<td>100</td>
<td>Irritation of eyes, nose</td>
</tr>
<tr>
<td>500</td>
<td>Strong irritation, severe eye injury</td>
</tr>
<tr>
<td>700</td>
<td>Severe respiratory tract damage, intense cough</td>
</tr>
<tr>
<td>1500</td>
<td>Blindness</td>
</tr>
<tr>
<td>3000</td>
<td>Lethal injuries possible, skin damage</td>
</tr>
<tr>
<td>5000</td>
<td>Pulmonary oedema</td>
</tr>
<tr>
<td>10000</td>
<td>Immediately lethal</td>
</tr>
<tr>
<td>40000</td>
<td>The gas is visible</td>
</tr>
</tbody>
</table>

9.2 DOSE–RESPONSE

Dose–response data are of great importance in toxicity evaluation and threshold limit estimations. For many chemicals these data are well established. For example, documentation concerning ammonia is very good, and is seen in Table 9.1. There is detailed information available on air concentrations, including odour threshold, toxic and lethal concentrations. This information is compiled from experimental data and from experiences at occupational exposure and at minor and major accidents, as ammonia is widely used and much experience from ammonia exposure has been gained. This type of information is also available for many toxic substances frequently used in industries, at working places, and in laboratories.

For less frequently used toxic substances no data or possibly only experimental data exist. This was the fact for methyl isocyanate (MIC) that was involved in the Bhopal tragedy. But from this accident valuable new information has been gathered. For example, a group of scientists from the National Defence Research Institute in Sweden has estimated the toxic effects of MIC from short exposure in man (Table 9.2.) (Karlsson et al., 1985). These figures are based upon dispersion calculations of gas plumes, taking into consideration the amount of released MIC, the time of the release, and weather conditions together with the toxic effects on man in exposed areas.

In almost all situations there is a close correlation between concentration of the toxic substance and observed toxic effects. However, for some substances, like phenol, a diluted solution is more prone to be absorbed through the skin than a concentrated solution, thus the dilute solution may be more toxic in this respect.

Although very good information on dose–response relationships is available,
Table 9.2  Suggested toxic effects of MIC at short exposure times in man

<table>
<thead>
<tr>
<th>Concentrations of MIC (mg/m³)</th>
<th>Effects in man</th>
</tr>
</thead>
<tbody>
<tr>
<td>---</td>
<td>Irritation</td>
</tr>
<tr>
<td>23</td>
<td>Risk of severe injuries</td>
</tr>
<tr>
<td>68</td>
<td>Severe injuries and risk of fatalities</td>
</tr>
<tr>
<td>225</td>
<td>High proportion of fatal injuries</td>
</tr>
<tr>
<td>675</td>
<td></td>
</tr>
</tbody>
</table>

From Karlsson et al., 1985.

This information might be of less importance in the very acute situation as, initially at least, the dose is most often unknown. Several hours may pass before one has a chance or time for more sophisticated determinations with appropriate equipment. In the acute situation one has to trust the observed or expected symptomatology to help define parameters. However, in planning activities the dose-response relationships for various chemicals are of great value for risk assessment.

9.3 ACUTE EXPOSURE

The most common route of exposure in major accidents is inhalation of gases that were stored under pressure. These will spread rapidly and over a wide area. Other likely routes of exposure in this context are the eye and skin. Ingestion of the toxin is less likely to occur unless drinking water has been contaminated, either by accident or sabotage. Ingestion exposure will not be dealt with in this context, nor will problems with chemical warfare agents.

9.3.1 Eye Exposure

Accidental splashing or squirting of liquid substances into the eyes is the most common cause of toxic eye injury. Some substances produce severe deep injury almost immediately, while others produce superficial reversible damage. However, the injury from some substances may appear unimpressive at first but later becomes progressively worse after a variable latent period. Gases, vapours and dusts also cause eye damage by lachrymatory action, corneal epithelial injury, or keratitis. These effects might be immediate or delayed.

Liquid Substances

The most typical caustic injuries after contact with liquids are produced by alkalis and acids inducing rapid deep penetrating injuries of the cornea,
conjunctiva, sclerae and even the lens and iris. This type of injury is also seen after exposure to oxidizing agents and tissue fixatives. Alkalis and related substances have a tendency to penetrate the deep layers of the cornea by saponification of the fatty components of the cells, while acids and related compounds cause a coagulation necrosis of the epithelial surface thus preventing deep penetration. However, very strong acid solutions produce a direct penetration injury of the corneal epithelium.

Delayed or latent action injury may develop without any initial signs of damage. For example, alkylating agents such as mustard gas and dimethyl sulphate, and tissue fixatives such as formaldehyde, bind to or react chemically with tissues so that they interfere with biological functions. The tissue changes may not be clinically evident immediately, but within hours the changes lead to biological breakdown which results in oedema and necrosis of cells and secondary inflammatory reactions.

Gases, Vapours and Dusts

Most of the substances that induce lachrymation, whether by physiological or specific chemical mechanisms, can injure the cornea when their concentrations are high. In general, these substances for which one might postulate a physical action by liquid contact tend, at high vapour concentrations, to produce epithelial injuries. Some gases, vapours or dusts may also cause a latent disturbance of the corneal epithelium with loss of epithelial cells and considerable associated discomfort, but the injury and discomfort do not appear until several hours after exposure. The subsequent course of this type of damage is variable depending on the severity of exposure. In the mildest cases the epithelium heals spontaneously, returning to normal in a day or two. In more severe cases the epithelial damage may progress, and the whole epithelium may eventually be lost, requiring a somewhat longer period to regenerate completely. In the most extreme cases, the damage can involve the corneal stroma as well as the epithelium. Such extreme reactions are rare.

Most likely to cause accidents involving eye injury to many persons are gases, vapours or dusts, but splashes of liquids into the eye may occur in an industrial accident or at a road or railway accident where the toxic substance is splashed or squirted widely. Eye injury in these situations will often be combined with skin lesions, respiratory tract injuries or systemic poisoning.

9.3.2 Skin Contact

Skin exposure to toxic agents may cause local damage alone or local damage in combination with systemic poisoning. Systemic poisoning may even be the only feature as a result of skin absorption through the intact skin. Although the skin
is usually an effective barrier to toxic chemical absorption, the intact skin behaves qualitatively like other cellular membranes. Toxic agents penetrate the skin at rates determined by their lipid solubility. Lipid-soluble substances are readily absorbed through the skin. Organic solvents present as vehicles for some industrial chemicals, may also enhance skin absorption. Inflammation, rubbing or other causes of increased skin blood flow will further increase chemical absorption.

The skin damage that is seen, for example, in corrosive burns may destroy the natural barrier of the skin. Non-soluble lipid substances may in this way be absorbed and cause systemic poisoning. Even a first degree burn may destroy the barrier of the skin.

The local damage from skin chemical contact is usually a chemical or corrosive burn and may be classified according to the same guidelines as thermal burns. Typical lesions are seen after acid or alkali burns. In mild cases of these chemical burns, only the superficial layers of skin are affected, while in severe cases all skin layers and possibly underlying tissues are damaged.

The local damage is most often seen directly after skin exposure to the toxic agent, but in some situations the initial signs of local damage may be lacking, although advanced local damage will appear later. For example, phenol and phenol-like compounds anaesthetize the skin initially, thus masking the typical signs of local damage and pain. After skin contact with low concentrations of hydrofluoric acid solutions initial signs of skin damage (or systemic poisoning) are lacking, but several hours later signs of skin damage and subcutaneous tissue damage will appear. The fluoride ion penetrates the skin and interferes with the cellular membrane calcium ion causing cellular necrosis. However, strong hydrofluoric acid solutions will produce an immediate local skin burn.

Systemic poisoning after skin absorption through intact skin is most likely to occur after contact with lipid-soluble substances, such as the organophosphates.

After skin absorption, symptoms usually appear after a symptom-free interval of minutes, hours or even days depending on the type of damage and target organs. Symptoms of central nervous system effects as excitation, convulsions, CNS depression and coma appear soon after the exposure, as do symptoms from the circulatory system. The signs of renal and hepatic damage usually are not present until one, two, or several days after exposure.

After skin exposure, it is of the utmost importance to find out what substance or substances are involved so that correct initial therapeutic measures can be undertaken to avoid unnecessary damage. This is, of course, true for all routes of exposure, but it is especially important after skin contact that correct and prompt initial measures be employed to effectively avoid or minimize local damage or systemic poisoning.
Inhalation

Toxic inhalants (gases, vapours and dusts) are characterized by their physical properties and induced pathophysiology, which in turn determine the clinical findings on presentation. Five categories of noxious agents are recognized: irritants, systemic poisons, combined irritants and systemic poisons, inert gases, and hot gases.

Irritants

Irritant inhalants produce toxicity by the way they cause mucosal damage of the respiratory tract. The location and severity of the injury depend upon the reactivity, concentration and water solubility of the substance and on the duration of exposure. Previously underlying diseases, especially of the respiratory tract, play an important role in this context.

Inhalants that are highly water soluble (like ammonia) dissolve in the water phase of the upper respiratory tract mucous membranes and will not reach the lower respiratory tract unless exposure is prolonged or high concentrations of gas are inhaled. These gases usually produce immediate symptoms, like watering of the eyes, rhinitis, pharyngitis, cough, and in severe cases laryngeal oedema. After exposure to high gas, vapour or dust concentrations, reflexogenic circulatory or respiratory arrest may occur. However, in prolonged exposure damage to the lower respiratory tract may supervene. This is even more likely to occur with the chemicals that are intermediate in water solubility, such as the halogen gases, hydrogen halides and sulphur dioxides.

Otherwise, it is mainly the inhalant chemicals of low water solubility in low concentrations and at short exposure that will give rise to damage to the lower respiratory tract causing pneumonitis, alveolitis and pulmonary oedema, often without any significant effects on the upper respiratory tract or eyes. This group includes chlorine, isocyanates, nitrous oxides and phosgene. Usually initial symptoms of the respiratory tract are prominent giving a guideline for the severity of exposure. However, attention should be paid to the fact that the initial signs of respiratory tract damage may be lacking with nitrous oxides and phosgene. However, following the onset of initial symptoms, there is usually a latent period during which the patient suffers little discomfort. This period may last between 30 minutes and 24–48 hours, and rarely 72 hours. This latent period is followed by the development of respiratory tract symptoms and pulmonary oedema due to increased capillary permeability. In severe cases, pulmonary oedema may appear during or shortly after the chemical exposure.

Apart from chemical irritation, other effects on the respiratory tract appear. Isocyanates, for example, also caused an asthma-like syndrome (Adams, 1975; Chailleux et al., 1982; NIOSH, 1973). This asthma-like syndrome may be caused by two different mechanisms. One mechanism is attributed to the fact that isocyanates are sensitizers of the respiratory tract giving asthmatic
Biological Effects in Humans

Symptoms, particularly after repeated exposure to low concentrations of the isocyanate. The other mechanism is that isocyanates may alter the biological response to beta-adrenergic stimulation or may induce local histamine release, thus causing bronchoconstriction that does not appear until after a latent period of several hours.

Systemic Poisons

Chemicals that are inhaled may also cause systemic poisoning without any respiratory tract symptoms. Symptoms of systemic poisoning vary according to the toxic substance and its target organs. Almost all types of toxic damage may be seen, and symptomatology may be immediate or delayed. The most prominent immediate symptoms are those from the central nervous system and circulatory system. Hydrogen cyanide and hydrogen sulphide, for example, block the cellular utilization of oxygen thus giving rise to cellular asphyxia and symptoms from the central nervous and circulatory systems almost immediately. This is true also for carbon monoxide, but the main toxic action of carbon monoxide is preventing delivery of oxygen to the cell by blocking haemoglobin oxygen-transport capacity. Organophosphates are powerful inhibitors of cholinesterases, resulting in accumulation of acetylcholine at synapses in the nervous system and at myoneural junctions, giving rise to cholinergic poisoning. Volatile hydrocarbons are narcotics causing central nervous system depression. However, halogenated hydrocarbons also sensitize the myocardium to endogenous and exogenous catecholamines, causing arrhythmias, predominant ventricular arrhythmias, and sudden death due to ventricular fibrillation. Inhalation of oxidizing agents (nitrites, and nitrobenzene) will cause methaemoglobinemia.

Irritants and Systemic Poisons

Toxic inhalants that are both irritants and systemic poisons also exist, for example, hydrogen sulphide, ozone, acetylene, and some metal fumes.

Inert Gases

Biologically inert gases are not toxic in themselves, but in high concentrations and in poorly ventilated rooms they will displace oxygen from the air and thus cause hypoxia. Gases of this type are hydrogen, carbon dioxide, methane and liquid gas.

Hot Gases

Inhalation of hot gases may cause thermal burns to the mucous membranes of the entire respiratory tract.
Treatments for Assessing and Reducing Injury from Chemical Accidents

9.4 INITIAL MANAGEMENT

Treatment of acute poisoning is based on four main principles that may be utilized to varying degrees, depending on the circumstances of exposure and the characteristics of the toxic agent. These principles are:

1. Removal of the toxic agent to prevent further local damage or absorption into the body.
2. Symptomatic therapy.
3. Antidotal therapy.

Removal of the toxic agent to prevent further local damage or absorption into the body is of crucial importance in the initial management of victims on the accident site after a chemical accident.

Symptomatic therapy is always applicable in treating poisoned patients. In most cases this is the only necessary principle to be used in managing the poisoned patient to full recovery. Maintenance of vital functions (like avoiding airway obstruction, assisting ventilation, replacing fluid losses) is of vital importance and self-evident.

Antidote therapy is most effective in reducing morbidity and mortality in a certain, limited number of poisonings. A universal antidote does not exist, and antidote therapy should only be used for specific indications. Details on recommended antidotes and indications for therapy will soon be presented in an IPCS publication (International Programme on Chemical Safety, in press). However, in this chapter, such antidote therapy is discussed that could be administered on the accident site.

Enhancement of poison elimination is a special measure performed at hospitals and will not be considered here as it falls outside the scope of this presentation.

In the following, initial patient management will be discussed with special reference to measures on the accident site that will minimize toxic effects on man.

9.4.1 Eye Exposure

Immediate or 'first-aid' decontamination should be carried out with the utmost speed possible by flooding the exposed eye with water to reduce damage from surface exposure to chemicals. For continuing irrigation, ordinary tap water or physiologic saline solutions are the first choices. Time should not be wasted looking for special irrigation fluid.

Dilution and flushing begun as soon as possible after injury is especially critical following caustic exposure. Transportation to a hospital should not be considered more important than thorough on-site irrigation.

As eye pain causes blepharospasm, the victim needs assistance in keeping
the eye lids open. A topical anaesthetic will facilitate adequate eye irrigation and make the patient more comfortable.

Common practice is to irrigate for 15–30 minutes to be sure of thorough cleansing. However, if the nature of the chemical contaminant is definitely known, the irrigation used should be adjusted accordingly. For severe alkali burns, irrigation should be continued for a long period, initially for at least 15–30 minutes and thereafter repeatedly for several hours. For acid burns, irrigation should be performed for 15 minutes, and for minor irritants, irrigation for a few minutes is sufficient.

Despite the theoretical advantage of using special agents for neutralizing certain chemicals, this type of treatment has seldom provided a significant improvement over immediate irrigation with water or saline, both of which are also usually much more readily available for first-aid treatment.

All corrosive burns of the eye should be followed up by an ophthalmologist’s examination for possible more sophisticated and specialized therapy.

### 9.4.2 Skin Contact

After exposure of the skin to toxic chemicals, flushing with copious amounts of water should be started as soon as possible, and with corrosives it should even be accomplished under clothes. Contaminated clothing, shoes, wrist watches, and jewellery should be removed to facilitate additional adequate flushing. Note that copious amounts of water should be used, especially if heat production will be pronounced when water is applied as with exposure to strong acids like sulphuric acid. After exposure to yellow phosphorus, the contaminated part of the body should be kept under water or dressed with wet dressings, as yellow phosphorus ignites in the air. In some occasions special solutions should be used. For phenol, for example, polyethylene glycol is used as a cleansing solvent as phenol is poorly soluble in water.

After adequate flushing, the skin should be washed thoroughly with soap and water, especially if there is a risk of absorption through the skin.

After skin exposure to corrosives, the risk of severe fluid loss should be considered, and the victim should be given intravenous fluids at an early stage.

In some cases application of an antidote on the skin is of crucial importance. For hydrofluoric acid, for example, calcium gluconate jelly is applied. The fluoride ion is then bound to the calcium in a stable, inert complex, thus preventing the fluoride ion from penetrating the skin causing severe tissue damage and possible systemic poisoning.

### 9.4.3 Inhalation Exposure

The victim should be taken from the toxic area as soon as possible. Note that the rescuer may need protective clothing and a protective breathing mask.
Methods for Assessing and Reducing Injury from Chemical Accidents

**Irritant Gases**

After exposure to irritant gases the victim should rest, if possible, in a half-sitting position to avoid the developing of pulmonary oedema. Oxygen should be administered as soon as possible. Physical activity and hypoxia increase the risk of pulmonary oedema occurring.

Apart from optimal symptomatic therapy, corticosteroid administration by inhalation and systematically is recommended as soon as possible to minimize pulmonary damage. This therapy has proved most successful (Lorin and Kulling, 1986).

**Systemic Poisons**

After exposure to gases that produce systemic poisoning, treatment should be guided by the specific inhaled toxicant and the presenting symptomatology.

If the victim is unconscious oxygen should be given. Apart from being a symptomatic therapy, oxygen reduces the toxicity of carbon monoxide, hydrogen cyanide and hydrogen sulphide.

Additional specific treatment, like antidotal therapy, is of critical importance in some poisonings, like hydrogen cyanide, organophosphate or heavy metals and should preferably be given promptly at the place of the accident. For this reason it is important that contingency planning for chemical accidents includes planning for adequate storage and availability of antidotes.

### 9.5 ROLE AND RESPONSIBILITY OF POISON CENTRES IN CHEMICAL ACCIDENTS

Poison centres have important roles in handling and managing chemical accidents where toxic effects in man can be expected. They can promptly give the needed information about the toxic substance including toxicity data, risks, first-aid measures, and hospital treatment required in the acute situation. Other important tasks for the centre in the chemical safety programme are to take an active part in the contingency planning for chemical accidents and in the education of rescue personnel and disaster medicine planners. A poison centre has a central position and can in this way act in a centralized way, for example, by providing and adapting information for the rescue team and giving guidelines on the supplying and the location of antidote stores (See Appendix in Part A of this volume for information on poison control centres and additional resource materials.)

#### 9.5.1 Information

The primary theme of appropriate and effective disaster management is information management. Timely information can change the entire character
of an accident; the poison centre, as an information source, has an important
role in this matter.

When an accident occurs like a gas leak from a factory or a hazardous
material rail or road accident, one early action at the site of the accident should
be to contact the nearest poison centre and inform it about the toxic substance
and/or synonyms. This measure must be included in the disaster checklist at
chemical plants and emergency centres. In this way the poison centre has
adequate time to compile the relevant information about the toxic substance,
even if all the detailed information is not available easily at the centre.

Taking into consideration the large number of toxic substances that are
handled at plants and transported on roads, by rail or ships, and the great
variety of possible toxic effects in human beings, information documenting
toxic effects, physico-chemical properties, first-aid measures and more has to
be compiled and made easily available in case of an accident. It is universally
recognized that it is of vital importance to establish poison control programmes
and protocols for the collection of data on toxic chemical effects on man and the
environment. In this work, poison centres play a key role. Poison centres' main
tasks are compiling information on chemical substances, including toxicity
data, first-aid measures, and more sophisticated therapeutic measures and to
disseminate this information, thereby making it easily and readily available in
an acute exposure situation.

Many chemicals used in household products or chemicals frequently used as
industrial chemicals are well known and detailed information can easily be
given. This information includes general data on the substance (synonyms,
chemical formula and structure, molecular weight, physical data such as
melting and freezing points, density), mode of action, routes of exposure,
kinetics (including absorption, transport and distribution), biotransformation
and elimination. Further, it includes information on toxicity (toxic doses and
concentrations), lethal doses and concentrations, symptoms expected from
different target organs in poisoning, measures for treatment of toxic effects,
and general and specific measures, like antidotal therapy and elimination
techniques.

However, for less frequently used chemicals, information is often scarce or
totally lacking. There are thousands of chemicals that need to be better
documented. This type of activity can be organized in many ways, but
resources must be made available. During a three-year-long period (1977–80),
the Swedish Poison Information Centre received support from the Swedish
Council of Environmental Information (that no longer exists) for two informa-
tion experts, one pharmacy and one chemistry (specialized in organic chemis-
try) for this type of work. One hundred and forty-five documents were
compiled and have been of great importance in the regular work at the centre.
A close collaboration between the Scandinavian centres in this activity has also
existed for a limited period.
A pilot activity has also been underway, sponsored jointly by The World Federation of Clinical Toxicology Centres and Poison Control Centres and the International Programme on Chemical Safety. Six poison centres from different parts of the world have participated in this activity each preparing one document. The conclusions that can be drawn from this pilot activity are, among others, that this type of work can not be continued in a centre’s regular workload unless extra resources are provided to the centres.

To find out what substances should be included in this work, it is very important to provide poison centres with information about what substances are commonly stored and used at plants and at working places and what substances are often transported on roads, by rail or ships. Lots of experience from accidents with industrial chemicals are at hand in many plants, experience that is seldom spread outside the plant. It is of vital importance for poison centres to obtain and assimilate this experience.

In these types of activity much information would become available, perhaps even including unforeseen risks that will give guidelines on preventive measures to be used when handling, loading, storing and transporting such substances.

Apart from giving information on the telephone to the general public, doctors, hospitals and rescue teams at a chemical disaster, information must, at an early stage, also be given to the communications media so they can give relevant information to the general public about the toxic substance, how to protect oneself and where to go for safety as soon as possible.

9.5.2 Contingency Planning

Regarding contingency planning for chemical accidents, the poison centre must assume responsibility and act to achieve and maintain effective disaster response systems (Kulling, 1984). Disaster medicine planning must be extended to include chemical accidents. A close collaboration between the disaster medicine planners and poison centres must be established. For example, the centre has to provide the planners with guidelines on appropriate measures for decontamination, first aid, how to supply and how to get access to antidote stores, and the like.

At the request of the Stockholm County Council, the Swedish Poison Information Centre has participated in developing an emergency plan for major chemical accidents. Apart from general information about the management of poisoning, this plan includes detailed information about some selected chemicals, including the major risks and detailed instructions on the management of poisonings due to these chemicals. The information has been adapted so that it will be understood by both non-medical and medical personnel. Guidelines on maintaining antidote stores at four of the Stockholm County Council hospitals have also been given, including recommended packaging, number of packages
and shelf life. Each antidote depot is intended to fulfil the requirements of several seriously poisoned persons for up to three days. A list of necessary antidotes to be stored in mobile units that will be brought directly to the accident location is also provided for in the plan. This plan developed by the poison centre has been considered a good model, and similar emergency plans now exist in many regions in Sweden.

9.5.3 Education

Another very important task is the education of all members of the rescue team. Training programmes and exercises dealing with chemical accidents have to be organized and the poison centre must take an active part in these activities. The rescue team must be provided with information about its main risks, first-aid measures and hospital treatment, and these data must be adapted to disaster situations. To meet this demand in Sweden, a handbook has been produced jointly by the Poison Information Centre, the Organizing Committee for Disaster Medicine, the Fire Fighting Service, and the National Board of Health and Welfare. This handbook deals with the different aspects on rescue work in instances of chemical calamities and is meant to be used by both medical and non-medical personnel.

In the training programmes for medical and non-medical rescue teams, lectures dealing with different aspects of acute poisoning in chemical accidents are included. These lectures are held at different locations throughout the country.

9.5.4 Follow-up Studies

Close follow-up evaluation of major as well as minor chemical accidents will provide much valuable information for the improved handling of such accidents. To visit sites of accidents as an observer in order to gather information and experiences from an ongoing accident is of vital importance. This is needed not only in the acute phase, but also at a later stage for follow-up studies of any possible late sequelae.

9.5.5 Collaboration

A close collaboration between poison centres all over the world and between poison centres and other organizations dealing with disaster medicine planning, like the World Association of Emergency and Disaster Medicine and the International Programme on Chemical Safety, is of vital importance. Close collaboration between poison centres and the chemical industry, both nationally and internationally, is also necessary to improve preparedness and afford prevention of chemical accidents.
REFERENCES AND FURTHER READING


