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Major chemical disasters

Britain's health services are poorly prepared

Industrial progress depends not only on the design and building of safe plants but also on having the means of dealing efficiently with major accidents and limiting their health and environmental consequences.[1] Some parts of the United Kingdom's emergency services are well prepared for dealing with chemical accidents, but a recent symposium on the medical aspects of chemical accidents heard that the health services' ability to cope is often inadequate.[2]

The main hazards posed by the chemical industry are large vapour or flammable gas explosions, fires, and toxic releases. Two of the worst examples occurred in 1984: in Mexico City about 500 people were killed and 5000 injured when a liquid petroleum gas plant exploded, and over 2500 people died in Bhopal after 40 tonnes of methylisocyanate were discharged over the city at night. The accidental release of chemicals during their distribution by pipeline, water, rail, or road can also have severe consequences, though in the United Kingdom the industry has managed to avoid a major disaster despite the 80 million tonnes distributed by road annually.[3]

In air releases toxic exposures can occur by skin absorption and ingestion as well as inhalation, and urgent measures to prevent or reduce exposure in individuals or populations, such as evacuation or decontamination, may need to be taken, particularly if environmental contamination has occurred. The chemical or chemicals involved need to be rapidly identified but identification may be delayed or impossible in releases from uncontrolled chemical reactions or fires. Even if the identity of the agent is known, knowledge about its human toxicity may be sparse, as was the case with methylisocyanate at Bhopal, for adequate assessments exist for only a few per cent of the over 70 000 chemicals in regular commercial use. The information that hospitals can obtain from poisons centres may therefore be limited, and antidotes are available for only a handful of chemicals and have no role in most incidents.

The risk of long term effects from chemical injury, such as carcinogenicity, teratogenicity, and other target organ damage, must also be considered from the outset. Such long term effects can occur even in the absence of severe acute effects in the exposed population, as was feared after the release of dioxin at Seveso in 1976.[4] Although their presentations may be different, toxic releases into water, food, and drink pose similar problems,[5] the most serious recent example being the toxic oil syndrome in Spain in 1981, caused by an unidentified chemical contaminant in rapeseed oil.[6]

In the United Kingdom the most potentially hazardous industrial sites come under the Control of Major Accident Hazard Regulations 1984 (CIMAH), which include a requirement for emergency planning to be undertaken for on site and off site air releases.[7] The implementation of these regulations has provided the opportunity for specialists in accident and emergency medicine and occupational medicine to collaborate with the emergency services in planning for local major incidents. Chemicals that often feature in such releases are chlorine, ammonia, sulphuric acid, hydrogen chloride, phosgene, hydrogen sulphide, and nitrous fumes. Chlorine, the commonest toxic gas for which emergency planning has to be undertaken, is an example of a dense, highly irritant gas that can cause rapid death from bronchospasm, laryngeal oedema, or toxic pulmonary oedema. Computer models of the dispersion of dense gases can be applied to estimate broadly the numbers of deaths and acute casualties for serious but reasonably foreseeable accidental releases, such as a fracture of a liquid gas pipe or failure of a road tanker delivery coupling.[8] As planning of land use to take account of major hazard risks is only recent,[9] some sites are located in densely populated areas containing schools, hospitals, and other establishments, and arrangements for the triage of casualties should reflect this.[8] Hospital protocols should include managing chemical burns of the eyes and skin and inhalational injuries due to irritant gases from toxic releases and fires.

Emergency planning under the major accident hazard regulations includes advising the local population on the hazards of a release, on the warning that people would receive, and on the protective measures that they should adopt. The protection afforded by buildings with their doors, windows, and ventilation systems closed will reduce exposure to half that in the open, provided that the occupants are alerted to go outdoors as soon as the plume has passed.[10] Evacuation may need to be considered for populations further downwind if time permits. In the United States train or truck spills commonly lead to evacuation,[11] but the decision to evacuate is often made hastily using inadequate information; in short releases that leave no residual contamination evacuation can be unnecessary or lead to more injuries, deaths, and psychological trauma than if people stay indoors.[12]

The emergency services have standard procedures for dealing with chemical incidents. The fire brigade is responsible at the scene for containing the hazard and making it safe, while the police have the overall coordinating role. Chemicals being transported may be identified from transport emergency (TREM) cards held by the driver or HAZCHEM codes displayed on the vehicle. The fire brigade has its own chemical advisers and access to computerised databases such as

Chemdata or the Imperial Chemical Industries' Information Register for information on health hazards, fire fighting techniques, and clean up procedures for spills. All staff should ensure that they do not put themselves at risk, and when appropriate the fire brigade will decontaminate casualties before they receive other than immediate lifesaving treatment.

Health professionals are more used to disaster planning for major trauma than for mass chemical exposures and the medical management problems these pose.[5] As a result the level of preparedness of accident and emergency departments in the United Kingdom is variable with regard to training, the use of chemical incident protocols, the provision of protective clothing and antidotes, and the availability of facilities for decontaminating seriously ill casualties.[2] In this issue Thanabalasingham *et al* illustrate such deficiencies in an actual incident and highlight the risks that emergency teams may face as a consequence (p 101).[13]

There is always the risk that the medical response to the uncommon but major incident might be delayed or mismanaged because the epidemiological, laboratory, and toxicological skills needed rapidly to evaluate and advise on the hazard are not available locally.[5] A team of experts may need to be deployed to make an urgent clinical and epidemiological assessment of the health impact and to ascertain the sources and extent of a toxic exposure in the population. This also applies to outbreaks of toxic illness from ingesting contaminated food or water, which might first come to the attention of general practitioners or specialists in public health medicine[14]; a recent example was the Lowermoor incident in 1988, when water was contaminated with aluminium sulphate. Until now there has been no provision for deploying such back up for local emergency services in the United Kingdom and no central government responsibility for coordinating major chemical incidents in peacetime,[15] but the Lowermoor incident has prompted the government to take some action. The Department of Health has recently formed an independent health advisory group to provide advice to health officials—urgently and on request—confronted with serious chemical contamination of a water supply. Local and national medical planning for chemical incidents needed strengthening. The many agencies and professional groups concerned need to develop much closer links and identify nationally the sources of expertise available in an emergency.

PETER J BAXTER

Consultant Occupational Physician,
University Department of Community Medicine,
Addenbrooke's Hospital,
Cambridge CB2 2QQ

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Transient global amnesia

Recurrences are rare and patients may drive

The syndrome of transient global amnesia first became clearly recognised with the description of 17 cases by Fisher and Adams in 1964.[1] Some two years later the late Lord Brain described a series of cases at a meeting at the Association of British Neurologists, and it was clear from the discussion that most neurologists had seen examples of the condition. Transient global amnesia is now a well established clinical entity with extensive reports describing more than 1000 cases.[2]

Transient global amnesia typically occurs in a middle aged or elderly person, who suddenly develops a disorder of memory, often regarded as confusion, which lasts for some hours. During this time the registration and recall of current events are impaired and afterwards the victim cannot remember any of the events during the confused period. During the attack the patient seems healthy, though is often distressed and not aware of what is wrong. There is no loss of personal identity and complex functions such as driving may be performed without difficulty. Recovery is complete and recurrences are unusual.

The pattern of memory disturbance has been the subject of much interest for several years, though neuropsychological function during attacks has rarely been examined. Hodges and Ward managed to examine five patients during an attack and were able to show a characteristic neuropsychological deficit.[3] Personality, problem solving, language, and visuospatial function remained intact. Immediate memory was preserved, but longer term verbal and non-verbal memory was severely disrupted. Retrograde amnesia was commonly present, though this tended to diminish during recovery, leaving a short retrograde gap in all cases. These observations confirm that transient global amnesia is primarily a disorder of memory and are compatible with the widely held view that the cause of the trouble lies within the temporal lobes.

Aetiologically transient global amnesia remains an enigma, though the most widely accepted view is that it is due to thromboembolic cerebrovascular disease with ischaemia in the territory of the posterior cerebral arteries, which supply the medial temporal lobes[4]—in other words, a transient ischaemic attack within the vertebrobasilar system. Other views are that the syndrome is caused by epilepsy[5] or migraine.[6] Evidence against the transient ischaemic attack theory is that the attacks last longer than an ordinary transient ischaemic attack, repeated attacks are rare, and strokes within the appropriate vascular territory, producing permanent memory loss, are exceptional.[3]

A new study has provided some important information about this disorder and its possible cause. Hodges and