Methyl bromide intoxication during grain store fumigation

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There have been over 300 cases of methyl bromide poisoning reported in the literature. The first objective of this case report was to bring out an experience with the false belief that work in a closed space is safe when accompanied by the use of a cartridge respirator with activated charcoal. The second objective of this article was to demonstrate the marked toxicity of methyl bromide with the potential to cause long-term neurological damage. Two experienced fumigation workers (equipped with rapidly saturable respiratory cartridges) entered a building where the concentration of methyl bromide was 17g x m\(^{-3}\) instead of the advised 20mg x m\(^{-3}\). They felt rapidly unwell and complained of nausea and shortness of breath, followed for one them by generalized convulsions. Five months later this last man was still bedridden. The other worker had almost no after-effects. The highest bromide level was found in the blood and also in the activated charcoal cartridge of the most injured worker. There was a relationship between methyl bromide level exposure and neurological damage importance.

Received 13 December 1994; accepted in final form 29 September 1995.

Methyl bromide was commonly used in fire extinguishers in aircraft engines and in submarines during the Second World War. Nowadays it is widely used against rodents, insects and mites as well as in a range of pathogenic organisms in soil, compost and timber. It is a highly toxic and easily penetrating fumigant.

Over 300 cases of systemic poisoning and 60 fatalities attributable to methyl bromide, have so far been reported. Particular concern about methyl bromide toxicity is warranted due to the absence of reliable warning properties, the delayed onset of symptoms, the potentially lethal outcome despite medical intervention and the possibility of serious peripheral and central nervous system after-effects.

Our report aims to draw attention to the continuing risk of exposure to methyl bromide, including its regular use by professionals.

The incident reported below documents the danger involved in neglecting elementary safety rules.

CASE REPORT

The disinfection of a grain store by methyl bromide fumigation was necessary. This was carried out by a team of two professional disinfection workers (44 and 39 years old), who worked for an experienced fumigation company. They had worked in this job for 20 and 2 years, respectively. The technique employed was intrinsically standardized. The workers ensured that the seven floor building was gas tight by sealing all windows and openings with plastic sheets. Afterwards they set on the floors gas cylinders containing methyl bromide, and fans which spread the disinfectant through all the rooms. They then turned the gas cylinder taps on, and started the fans. They started on the top floor and finished on the ground thereby closing the last opening. Gas concentration was determined during fumigation by gas chromatographs connected to the outside. It showed concentration from 10 gram per cubic meter (g x m\(^{-3}\)) to 20g x m\(^{-3}\). Having completed this operation, they waited 24 hours and then opened the main openings from the outside. In order to save time however they did not adhere to poison gas delay training. Instead they entered the building immediately so as to pick up their equipment. The concentration of methyl bromide was at this time 17g x m\(^{-3}\) instead of the advised 20mg x m\(^{-3}\), or almost one thousand times too high. Cartridge respirators which are not sufficiently protective because they are
saturable in a few minutes in this atmospheric concentration, were used instead of the obligatory autonomous air flow masks. Both workers stayed in the building for one hour and then drove the 100 miles back home. During their journey home, they had several symptoms including dizziness, fatigue, nausea and vomiting, chest pain and shortness of breath. They were both admitted to hospital.

The condition of the younger man improved rapidly and he was able to leave hospital three days later without almost no after-effects. His blood bromide level 48 hours after the acute intoxication was 46.6 mg x l¹. The older man became progressively confused and disoriented. Several hours later he developed twitching of the arms and became ataxic. He had generalized convulsions. There was no relevant past history of epilepsy, nor was he taking any medication. He was unable to communicate but was aware of his surroundings. Anti-convulsant therapy was started and positive pressure ventilation begun. The liver was undamaged but the respiratory system and the kidneys were affected. Anuria appeared rapidly. The electroencephalogram showed centro-encephalic spike discharges. It was consistent with the myoclonic jerks. Forty hours after the acute intoxication, his blood bromide level was 156mg x l¹. Daily monitoring after the withdrawal of the muscle relaxants was carried out. The convulsions had not completely ceased. The diuresis restarted at the same time. Five months later, he was still dysarthric, had myoclonic twitching, intention tremors, and could not stand up without help.

DISCUSSION

Methyl bromide, a colorless liquid but gaseous at room temperature, enters the body either by inhalation or through the skin or mucosa. A particular cause of danger is that the more serious effects due to central neurological lesions do not appear until after an interval of a few hours to several days.

In addition the toxicity is of an insidious nature. There is a diversity of symptoms. At first these symptoms range from discomfort, headaches, visual disturbances, nausea and vomiting followed by confusion, disorientation, delirium and convulsions. Later on periods of unconsciousness may occur.

In vitro and in vivo data suggest that methyl bromide is a methylyating agent. It causes the inhibition of enzymes and other proteins by alkylating sulfhydryl groups. It has been suggested that this is one of the possible mechanisms for the neurological effects. It is also a soluble liquid compound. It may cause direct disruption of lipid rich cell membranes, the myelin sheath of nerves. This may explain previously documented symptoms of paresthesias.

After inhaling a concentration of more than 20mg x 100ml¹, toxic effects are invariably noted. Acute intoxications with methyl bromide lead to pulmonary edema, convulsion, hyperthermia and coma. Longer standing exposures to lower concentrations of the gas cause neuropathies, ataxia, hepatotoxic and nephrotoxic signs, and behavioural disturbances.

Our second patient was poorly protected by his inefficient filter mask, and therefore exposed to high methyl bromide levels. These circumstances explain why most of his symptoms belong to a clinical picture which corresponds to acute intoxications. The long term sequelae of our patient include permanent neurological damage. This was been reported to occur in other non-fatal cases.

Once again this case demonstrates the marked toxicity of methyl bromide. Most accidents are a result of exposure in the field both during and after injection of the toxic agent into the soil. In our case, the sequence was different. But the seriousness of poisoning was comparable to earlier reported intoxications. Before this accident none of the employees of this firm had ever experienced any adverse health effects. One may wonder why the two men executing the same work did become poisoned. There are three complementary explanations. Firstly, a variation in susceptibility to methyl bromide poisoning after similar exposures had already been reported.

Secondly older age leads to a reduction of elimination of toxic substances and to less resistance. In this case however, the age difference was slight. Last but not least, the most seriously ill patient was the one who had been most exposed to methyl bromide. His cartridge mask became saturated quickly and he therefore inhaled toxic amounts. The concentration of bromide in the active charcoal of the cartridges showed a level of above 10mg of bromide per gram. The highest bromide level was found in the cartridge of the most injured worker.

The staff of the firm had received proper instruction and training in safe handling of the product as well as in the use of personal protection in gas detection equipment and first aid measures. There are several reasons for this operational accident: negative selection in the use of personal protection, reluctance to wear appropriate breathing apparatus, and the desire to save time when going back home.

BIBLIOGRAPHY