

Case report

Near death due to inhalation of slurry tank gases

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Slurry tank storage facilities are playing an increasing role in mechanised farming. Accidental poisoning from gases produced in such tanks is a recognised hazard, and multiple fatalities have been reported.^{1,2} The principal constituents are methane, ammonia, carbon dioxide and hydrogen sulphide. The most dangerous of these is hydrogen sulphide which acts in a similar manner to cyanide, with reversible inhibition of the respiratory enzyme cytochrome oxidase. Concentrations above 200 parts per million (ppm) produce direct irritant effects on exposed surfaces, and pulmonary oedema on prolonged inhalation.³ Higher levels (above 500 ppm) depress the central nervous system, with paralysis of the respiratory centre, and almost instantaneous loss of consciousness. The familiar smell of 'rotten eggs' is not a reliable warning sign as paralysis of the olfactory nerve makes the gas odourless at lethal levels.

Most reported cases have been associated with agitation of slurry prior to, or during, emptying procedures, although toxic exposure to hydrogen sulphide can also occur in sewers, mines and the chemical industry. The problem was exacerbated in 1985 by the wet summer and autumn, which made fields unworkable. Large quantities of slurry therefore remained undisturbed for long periods and subsequently more gas than usual accumulated beneath a hard crust, to be released when the tanks were eventually emptied.

CASE HISTORY

A 31-year-old farm labourer, previously in good health, was preparing to empty a tank containing approximately 6,000 gallons of slurry. This had been undisturbed since the autumn one year previously. The surface crust had been broken and the contents agitated by a tractor-driven mechanical pump prior to removal to a tanker. His workmates noticed him slumped over the drainage slats above the slurry tank inside the cowshed. Help was summoned and he was dragged outside within three minutes. The patient was noticed to be blue, pulseless and unconscious, with very weak respiratory efforts only. No attempts at cardiopulmonary resuscitation were made. Two cows in the cowshed died immediately and a calf was later destroyed.

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On arrival of the ambulance about 20 minutes later, he was unconscious with no palpable carotid or femoral pulses, but there was still shallow respiration. The pupils were only moderately dilated. Oxygen was administered, and after several minutes he vomited and gradually regained consciousness. On admission to hospital the patient was orientated in person, time and place. He was not cyanosed. Blood pressure was 150/90mmHg, and his pulse irregularly irregular at 70 beats per minute. He had good bilateral air entry with normal breath sounds. Tone, power and reflexes were normal. The electrocardiogram confirmed atrial fibrillation, but was otherwise normal. The chest radiograph was normal, with no evidence of pulmonary oedema. Sinus rhythm returned within an hour of admission. Subsequent electrocardiograms and cardiac enzymes were normal. He made a rapid recovery and was discharged after two days. At follow-up one month later, he was in perfect health.

COMMENT

The reported case highlights the danger of working with slurry in a confined space. The toxic effects of slurry gases have been well documented³ and, although no qualitative or quantitative gas analysis was made at the scene, the clinical picture is consistent with inhalation of a high concentration of hydrogen sulphide. The sudden loss of consciousness was presumably due to paralysis of the respiratory centre with resulting hypoxia. Grand mal fits and long-term neurological sequelae have been reported,⁴ but despite collapse and unconsciousness our patient had no permanent ill effects. Supraventricular and sinus tachycardias have been associated with hydrogen sulphide intoxication, but we are not aware of atrial fibrillation being previously reported as a clinical complication. This arrhythmia was probably due to cardiac hypoxia although ventricular irritability would be a more expected finding. There was no indication in the patient's history, examination or investigations to suggest any pre-existing heart disease or paroxysmal tachyarrhythmia. There was no clinical or radiographic evidence of pulmonary oedema, and his initial cyanosis responded to administration of oxygen by the ambulance crew. The absence of pulmonary oedema can be explained by the sudden collapse and apnoea protecting the lungs from further toxic exposure.

Our patient must be considered lucky to have survived this episode. His rescuers were also at considerable risk when dragging him clear without breathing apparatus, but in so doing they undoubtedly saved his life. After removal from the toxic environment, further management of hydrogen sulphide is mainly supportive, with the need for oxygen administration, cardiopulmonary resuscitation and mechanical ventilation being determined by the severity of intoxication. Inhaled or intravenous nitrite has been suggested as a measure to protect and reactivate cytochrome oxidase,⁵ but the need for its early, and probably pre-hospital, administration limits its efficacy. The resulting formation of methaemoglobin is, moreover, not without its own hazards.

Including this case, there have been 14 slurry tank accidents reported to the Department of Agriculture in Northern Ireland in the 17 years prior to the end of 1985.⁶ Six incidents involved cattle only (with more than 35 deaths), and five involved a total of nine people, all of whom recovered, apart from one believed to have permanent brain damage. The three remaining incidents produced four human deaths. One of the victims entered the slurry tank attempting to rescue his colleague. The cause of death was in one case asphyxia by slurry gases, and in

three drowning in slurry probably preceded by asphyxia. Ten of the reported accidents occurred in 1985. This recent increase can be explained by the freak weather experienced in the Province that year. Hydrogen sulphide should be considered as lethal as cyanide, and clear advice has recently been given to the farming community to minimise the risks of accidental exposure when emptying slurry tanks.⁷

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CORRIGENDUM

C.A.T. scan pictures shown in Fig 1 of "Acquired Immune Deficiency Syndrome in Northern Ireland" (Vol 55, No 1, page 81) were of different levels of the brain. This was due to a mistaken submission with the final draft by R. D. Maw and W. W. Dinsmore. Comparable levels were, of course, taken and reported as showing cerebral involution.