✕ "Lethane" Poisoning: Report of a Fatal Case ✕

Insecticides containing "lethane" are in common use for the treatment of pediculosis. Harrison (1947) reported a fatal case of poisoning due to this type of preparation, which he believed to be the first recorded example of its kind. A further non-fatal case was recorded by Guy (1951).

CASE REPORT

A male child aged 1 year and 11 months had recently been treated for miliary tuberculosis for about a year. While in his own home he drank a small quantity (the exact amount being unknown) of a liquid insecticide, believing it to be lemonade. He rapidly became unconscious, and was admitted to hospital in this condition.

On examination the child was deeply comatose. The limbs were flaccid; the colour was good, and the breathing regular. There was a smell of paraffin in the breath. The pupils were equal, were dilated, and did not react to light. There were no other clinical abnormalities. Gastric lavage was carried out and nikethamide given, both intramuscularly and intravenously. Later the breathing became stertorous and cyanosis was noted. Generalized clonic convulsions then developed, and these were controlled only by sodium phenobarbitone and lumbar puncture. Respiratory failure then set in, with prolonged periods of apnoea, and the child died two and a quarter hours after admission.

Necropsy showed miliary tuberculosis of lungs, liver, spleen, and kidneys, the miliary lesions being in varying stages of development and healing. The stomach was moderately dilated, but was otherwise normal. The lungs showed hypostatic congestion. There was no abnormality of the central nervous system. Analysis of the gastric contents showed the presence of a thiocyanate.

COMMENT

The composition of the preparation, as stated on the label, was as follows: lethane 384 special, 49%; ol. petrolei alb., 49%; and ol. citrionel., 2%. "Lethane 384 special" has the following composition: β -butoxy- β -thiocyanodiethyl ether, 12.5%; β -thiocyanoethyl laurate, 37.5%; and refined paraffin, 50%.

The clinical features of the present case were those of rapid coma, convulsions, and respiratory depression and failure. These findings are in agreement with those of Cameron *et al.* (1939), who investigated the toxic effects of several thiocyanates on rats, rabbits, and guinea-pigs. They concluded that β -butoxy- β -thiocyanodiethyl ether was highly toxic to these animals, and that death was due to respiratory failure, probably of medullary origin. Harrison, in his report of a fatal case of lethane poisoning, suggested that death was due to respiratory failure.

Guy (1951) reported the case of a child of 20 months who took 1 to 2 dr. (3.5 to 7 ml.) of a lethane hair-oil. Some of this was apparently inhaled, as a bronchopneumonia developed. The child remained ill for about two weeks and showed slight neck rigidity, drowsiness, and irritability. There was no evidence of hepatic or renal dysfunction. It is possible that the bronchopneumonia may have been due, in part at any rate, to the paraffin base. The neurological features probably represented a toxic effect from the thiocyanates: on the other hand it is possible that these findings could have been complications of the pneumonia *per se*.

Absorption of the poison seems to be rapid, and there appears to be no effective antidote. Glaister (1945) suggested a trial of methylene blue (50 ml. of 1% solution intravenously) or sodium thiosulphate (10–50 ml. of 20% solution intravenously). Apart from these measures, treatment must be symptomatic, and should include early and energetic gastric lavage.

The present case is reported in order to re-emphasize the dangerous nature of these preparations, which, as Harrison has pointed out, can be readily bought at any chemist's shop

and which are not labelled "poison." As the contained thiocyanates have a toxic action similar to that of hydrocyanic acid, it is suggested that the future sale of such preparations should be brought within the scope of the poisons regulations.

Our thanks are due to Dr. A. M. Tripathy for the necropsy report; to Mr. H. K. Lawton for the analysis of the gastric contents; and to Dr. R. W. M. Strain for permission to publish the details of this case.

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Death After Intravenous Iron

More or less severe reactions to the intravenous injection of saccharated iron oxide have occurred on several occasions during the last few years, but none of those previously reported have ended fatally. Nissim (1947) suggested gradually increasing dosage, and Nissim and Robson (1949) reported that "several patients received repeated doses of as much as 300 mg. of iron by intravenous injection without the slightest toxic reaction." In mice they said that "death is presumably due to multiple capillary embolism in internal organs." Davidson and Girdwood (1948) reported a case treated by repeated intravenous injections of saccharated iron oxide, in which sickness and rigors occurred with the last three injections of 100 mg. Severe reactions also occurred in several other cases. Slack and Wilkinson (1949) used 25 mg. on the first day, increasing to 50 mg. on the second, 100 mg. on the third, and 200 mg. subsequently. One of 60 patients developed a mild reaction at the 200-mg. level. In 60 other cases more than 800 mg. had been given intravenously. Mitchell (1949), Sinclair and Duthie (1949), and Govan and Scott (1949) have also described reactions, while Ramsey (1950) gave initial doses of 300 to 900 mg. of "ferrivenin" in 11 cases, in all but one of which there were severe reactions (weakness, sweating, sickness, and collapse for a few hours). Mooney (1950) reported one case treated with an initial dose of 100 mg., which three hours later developed a temperature of 103° F. (39.4° C.) with intense headache, shivering, and copious vomiting lasting some six hours. The manufacturers suggest the use of graded doses.

Below we record a case in which an initial intravenous dose of 100 mg. led to severe reactions and death.

CASE REPORT

A man aged 69 was admitted to hospital for treatment of anaemia. His blood count was haemoglobin, 61% (9 g. %); red cells, 3,890,000; C.I., 0.79; white cells, 6,500. Red cells showed marked anisocytosis, moderate poikilocytosis, anisochromia, and polychromasia. A few target cells were seen. Reticulocytes were less than 1%.

A slow injection of 5 ml. (100 mg.) of saccharated iron oxide ("ferrivenin") was given intravenously, and three minutes later the patient became hot and flushed. After a further two minutes he became cold and drowsy. Ten minutes after the completion of the injection he became dyspnoeic and cyanosed, and then collapsed. He was pulseless, and no apex beat could be felt or heard. Gasping respirations continued. Intracardiac injections of nikethamide and adrenaline, as well as intravenous injections of nikethamide and lobeline, were given. Artificial respiration and oxygen were given from the time of the collapse. Weak heart beats were noted shortly after the intracardiac injections, but the patient died 30 minutes after the original injection.

At necropsy 18 hours later the only evidence of disease was in the coronary arteries, both of which were calcified, and the right artery was completely occluded. There was no evidence of multiple emboli.

The LD 50 of the batch of ferrivenin when originally tested was approximately 300 mg. of iron per kg. of body weight in mice, and a recent toxicity test on the same batch showed no significant departure from this original figure.

COMMENT

Trivial and severe reactions to the use of intravenous ferrivenin have previously been reported. This case in which sudden death followed the first injection emphasizes the necessity for care: the initial dose should not exceed 25 mg. and the possibility of severe reactions even in patients who have had repeated injections should be kept constantly in mind.

We are indebted to Dr. R. A. Hickling for permission to publish details of this case.

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Long-delayed Rupture of Spleen: Report of Two Cases

Delayed bleeding occurs in about 15% of all splenic injuries, the latent period varying between a few hours and many days. According to Zabinski and Harkins (1943) this latent period is less than seven days in 50% of the cases and less than 14 days in a further 25%. They could find only eight reported cases in which the delay was longer than 19 days, one of these being as long as two years. The following cases are of interest mainly on account of their long latent period—22 and 26 days respectively.

CASE REPORTS

Case 1.—A soldier aged 22 was admitted to a military hospital in 1942 with gunshot wounds of his feet and hands, sustained when he was blown up by a landmine. He had apparently no other injuries. These wounds healed, and on the 22nd day after injury he reported to the commanding officer's office for discharge from hospital. Here he collapsed with sudden severe abdominal pain and considerable shock. After resuscitation a laparotomy was performed and a ruptured spleen removed.

Case 2.—A married woman aged 29 was brought to hospital in November, 1950, having collapsed in the street with sudden severe upper abdominal pain, radiating to the tip of the left shoulder. She gave a history of a fall from a chair 26 days before, when she had "bruised" her left side. After this injury she had been confined to bed for five days with severe pain in her left side and shoulder. She had then returned to her work as a school teacher, which included "keep-fit exercises." She had experienced a dull ache in her side but no severe pain until her sudden collapse. On examination she was shocked and very pale. There was great tenderness in the upper abdomen, with rigidity in the left hypochondrium and loin. At operation, after resuscitation, the peritoneal cavity contained a fair amount of liquid blood. Examination of the spleen showed a small tear on the costal surface with a large amount of surrounding clot. Splenectomy was performed, and the patient made an uneventful recovery.

COMMENT

Numerous theories have been advanced to account for the latent period. The formation and later rupture of a subcapsular haematoma probably account for some cases, and McIndoe (1932) points out that the severity of the tear as found at operation may be much greater than the original injury suggests, and that it is possible that the haematoma ruptures with almost explosive violence, causing the very sudden collapse of the patient.

The theory of the perisplenic haematoma as advanced by McIndoe and later by Delannoy (1939) seems to fit in with the symptoms in many cases and with the findings at operation of large amounts of clotted blood in the region of the spleen. Other theories are: intrasplenic bleeding controlled by pressure of the haematoma on the pulp; temporary haemostasis due to shock; tamponade by omentum, colon, stomach, etc. McIndoe places these injuries in three categories: (1) minor capsular rupture or parenchymal injury; (2) subcapsular haematoma; and (3) capsular haemorrhage with perisplenic haematoma. Of these, Nos. 2 and 3 are the most important.

Delannoy associated these pathological lesions with the symptomatology, and suggested two clinical types. (1) Splenic rupture with delayed haemorrhage. This corresponds with McIndoe's subcapsular haematoma and usually with a symptom-free latent period. (2) Rupture with retarded symptoms, corresponding with the perisplenic haematoma. Here the latent period may be marked by pain of varying severity occurring in the left hypochondrium and shoulder or sometimes by generalized abdominal discomfort.

Examination at this stage will reveal persistent tenderness and rigidity in the left upper quadrant, and x-ray films may show a paralysed diaphragm on this side. Other symptoms are pallor, weakness, dizziness, and fainting attacks, though these latter are probably more common with a slow continuous leak than with true delayed rupture. A number of such cases have been recorded, and one reported by Coyle, Kleiman, and Smith (1950) presented with gradual abdominal distension and anaemia.

The cases reported here fit well into these clinical categories. Case 1 had no symptoms referable to the abdomen between the time of his injury and his collapse 22 days later. Case 2 experienced intermittent pain in the left hypochondrium, and at operation a large clot was found around the splenic region, with liquid blood in the rest of the peritoneal cavity.

The pain in these cases is usually sudden and severe, and most commonly felt in the splenic region. Zabinski and Harkins reported the frequency of associated fractures of the ribs—a fact previously recognized by McIndoe, who stressed the importance of keeping under observation for at least 14 days any patient with an injury to the left side, particularly if complicated by fractured ribs. It must, however, be remembered that delayed rupture of the spleen can occur after quite a trivial and sometimes forgotten injury, and it is probable that many, if not all, the cases reported as "spontaneous rupture of a normal spleen" fall into this category.

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